Frontiers in Biosciences

ISSN: 3106-5937 | 3106-5945
Volume 3, Issue 1, 2026

CARDIOVASCULAR MANIFESTATIONS AND COMPLICATIONS IN COVID-19: A REVIEW

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Keywords
COVID-19, SARS-COV-2, Cardiovascular disease, Muscle inflammation, Heart injury, Heart failure

Article History
Received: 02 February 2026
Accepted: 15 March 2026
Published: 31 March 2026

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Abstract

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus responsible for COVID-19, has infected over 670 million people globally and caused approximately 7 million deaths. Emerging research highlights a significant link between COVID-19 and cardiovascular disease (CVD). Conditions like diabetes mellitus and hypertension—already known CVD risk factors—appear to be further exacerbated by COVID-19, which may act as an independent risk modifier. Studies suggest that previous COVID-19 infection increases the risk of developing various CVDs to a degree comparable with traditional risk factors. Complications such as myocarditis, acute coronary syndrome, heart failure, thromboembolism, and arrhythmias have been observed, especially in individuals with preexisting heart conditions. COVID-19 may influence cardiovascular health both directly—through viral invasion of heart tissues—and indirectly—via systemic inflammation. This review summarizes current findings on the interplay between COVID-19 and CVD, explores underlying mechanisms, and discusses evolving treatment and prevention strategies in the context of the pandemic.

INTRODUCTION

The COVID-19 disease 2019 is triggered by the coronavirus that causes severe acute respiratory syndrome (SARS-CoV-2), which was first identified in Wuhan, China [1]. This pandemic spread from China to the entire world quickly [2]. The first reports of COVID-19 came from Wuhan, China, in December 2019 [3]. The most typical signs and symptoms that can cause COVID-19 are respiratory sickness and breathlessness; people can recover with or without any treatment [4]. COVID-19 has diseased over 670 million people internationally and been connected to about 7 million expiries [5]. It equally effect respiratory as well as cardiovascular system as shown in Figure 1.

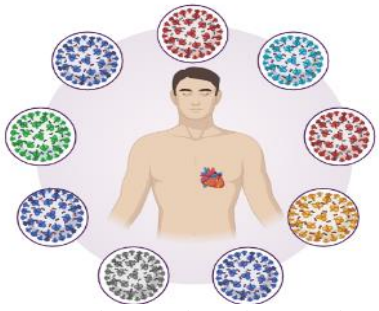

Figure 1 Covid-19 and cardiovascular system

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ISSN: 3106-5937 | 3106-5945
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Review of Literature

Results of Heart Scans after COVID-19 Illness

Heart-related symptoms can arise due to the SARS-CoV-2 virus. Using ultrasound of the heart to examine patients has revealed damage to the heart muscle, including issues with the left pumping chamber, notably impaired filling of the left ventricle and reduced pumping ability, as well as enlargement of the right pumping chamber, sometimes with reduced function [6-8]. Additionally, data suggests that right ventricle strain rises from pulmonary injury, and higher troponin measurements correlate with more severe right ventricle impairment [6, 7]. Moreover, lung blood flow speeds up, accompanied by declining right ventricle function, reduced tricuspid valve movement speed, and decreased tricuspid ring displacement, indicating a heightened right ventricle resistance [6]. The variety of observations implies that numerous pathways might elucidate the influence of COVID-19 on cardiac tissues. A comprehensive review highlighting the most prevalent heart anomalies detected via echocardiography and cardiac MRI demonstrated that many individuals with COVID-19 display raised pulmonary arterial systolic pressure [9]. Moreover, the analysis demonstrated that respiratory impairment resulting from the viral infection can contribute to a rise in pulmonary artery pressure, ultimately leading to right ventricular malfunction [9]. Despite the common reporting of left ventricular malfunction and left ventricular global longitudinal strain, right ventricular impairment demonstrates a greater prevalence and is considered the probable origin of the noted elevated troponin values [9, 10]. A comprehensive analysis of echocardiography results indicated that left ventricular systolic impairment was identified in less than 10% of patients exhibiting anomalous findings [6]. Analyzing right ventricular strain profiles could be valuable, alongside the measurement of cardiac biomarkers, in appraising disease progression. Right ventricular strain profiles demonstrate compromise in the initial phases, including in milder forms of the condition [7]. The endothelium may undergo injury via systemic mechanisms, particularly those involving reactive immune and inflammatory cytokine-driven reactions stemming from COVID-19 [11]. Furthermore, electrocardiographic recordings hold clinical significance for patient evaluation, as the identification of T-wave inversion can aid in the diagnosis of stress cardiomyopathy and myocardial inflammation [11]. In summary, echocardiography and cardiac magnetic resonance imaging accurately evaluated myocardial damage and cardiac sequelae in individuals affected by COVID-19. Nevertheless, these imaging techniques may not be universally accessible to patients, nor are they routinely employed or warranted in clinical settings. Under these circumstances, cardiac biomarkers, such as troponin and creatine kinase, along with sonographic imaging, represent readily available diagnostic instruments that can be utilized to detect cardiovascular complications [9].

Impact of COVID-19 on cardiovascular system

Covid-19 adversely affects the anatomy and physiology of cardiovascular system as shown in Figure 2.

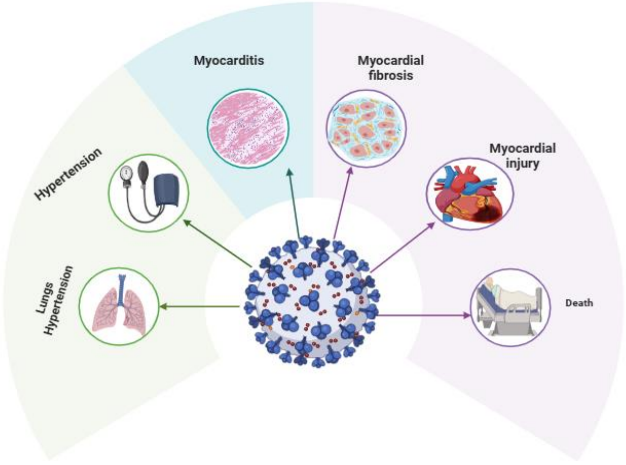

Figure 2 Mechanism of covid-19 impact on cardiac system

Frontiers in Biosciences
ISSN: 3106-5937 | 3106-5945
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Heart muscle inflammation (Myocarditis)

Myocarditis suspicion arises with elevated cardiac biomarkers, typical symptoms, and excluded ischemia, confirmed by cardiac MRI or endomyocardial biopsy (EMB) [12],[13],[14],[15],[16]. Primarily viral, including SARS-CoV-2, it often coincides with symptomatic COVID-19, featuring angina, dyspnea, or fatigue, with rare fulminant or delayed presentations [17],[18],[19]. Prevalence is challenging to ascertain due to limited routine screening and EMB use, especially in younger patients [20]. Authors found a prevalence of 2.4 per 1000 hospitalizations for definite/probable myocarditis, with a median age of 38 and frequent chest pain/dyspnea; males showed a higher risk post-COVID-19 [21, 22]. Outcomes are generally favorable, with most patients recovering, although severe cases with pneumonia can have higher mortality [23-25]. In athletes, symptom-based screening underestimates myocarditis prevalence compared to CMR, with varying resolution rates [26-28]. Possible mechanisms include viral entry via ACE2, endothelial cell infection, and cytokine-mediated damage [29-34]. Histologically, it's characterized by lymphocyte and macrophage infiltration, potentially driven by systemic inflammation and cytokine storms, notably IL-6, which can also promote thrombus formation [16, 35-38].

Arrhythmias

COVID-19 promotes cardiac arrhythmias, with sinus tachycardia and atrial fibrillation (AF) being prevalent in hospitalized patients [39]. AF rates range from 10-18%, which may elevate mortality risk [40-42] [42, 43],[44, 45] [43]. A registry study showed 5.4% new-onset AF, initially linked to higher in-hospital death, though this association weakened after adjustments [44]. Another study reported 12.5% new-onset AF with increased mortality [40]. Ventricular arrhythmias, particularly non-sustained ventricular tachycardia (nsVT), were observed in 17.7% of hospitalized patients, with higher rates in those with elevated troponin [39]. Cardiac arrests, bradyarrhythmias, and nsVT were also reported in another study [46]. ECG analysis revealed potential impairment of cardiac conduction, with increased ventricular repolarization markers [47]. While direct correlation between COVID-19 and ventricular arrhythmias is still being established, potential triggers include myocardial injury, hypoxemia, inflammation, and severe illness.

Physiological disturbance

SARS-CoV-2 can inflict harm upon the heart through multiple pathways, encompassing cardiac muscle cell attachment and endothelial cell injury. Cardiac myocytes exhibit a substantial quantity of ACE2 receptors, surpassed only by pulmonary tissue cells [48]. Viral-induced cellular damage causes disruption and impairment, which can precipitate myocardial injury and subsequent inflammatory responses [49]. Vascular endothelial cells, located within the heart and the entire vascular network, are susceptible to cardiac and systemic complications [50]. Several research endeavors have indicated that ACE2 receptors, localized within the lining of blood vessels, enhance viral intrusion in a manner directly comparable to that seen in myocytes [34, 51-56]. Subsequent investigations have likewise posited that pericytes, the cells surrounding the endothelium, could be the actual facilitators of the disease process, given their possession of ACE2 receptors [57]. The specific direct mechanism(s) by which infection affects endothelial cells and pericytes remains to be fully elucidated; however, data supports indirect mechanisms of COVID-19-related endothelial cell damage and dysfunction, resulting in myocardial tissue harm. Endothelial cells can sustain damage through systemic processes, notably reactive immune and inflammatory cytokine-mediated responses elicited by COVID-19 [58-60].

Heart injury

The immune and cardiovascular systems depend on the membrane-bound amino peptidase angio tension-converting enzyme 2 (ACE2) [61]. The development of diabetes mellitus, hypertension, and heart function are all influenced by ACE2. Moreover, ACE2 has been identified as a corona virus receptor [61].SARS-CoV-2's spike protein causes infection via binding to ACE2, which is highly expressed in the heart and lungs [61].In individuals with CVD, SARS-CoV-2 induces respiratory symptoms that worsen as a result of elevated ACE2 secretion. The safety and possible side effects of antihypertensive medication with ACE inhibitors or blockers should be assessed, and further study is required to decide if patients should switch [62].

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ISSN: 3106-5937 | 3106-5945
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Heart Failure

COVID-19 significantly impacts heart failure (HF) management, with observed reductions in HF hospitalizations potentially contributing to increased mortality [63-65]. During the pandemic, hospitalized HF patients presented with more severe symptoms, and intra-hospital mortality rose, with 2020 hospitalization being an independent mortality predictor [64, 65]. SARS-CoV-2 infection exacerbates pre-existing HF, with HF prevalence among COVID-19-positive individuals ranging from 3% to 21% [63, 66, 67]. Acute HF complicated 7% to 63% of COVID-19 hospitalizations, significantly elevating death risk (OR 9.36) [68]. Elevated troponin levels were common, and HF history correlated with increased hospitalization and adverse outcomes [68]. HF was a strong predictor of hospital admission and critical illness in a cohort study [69]. Retrospective analyses highlighted higher mechanical ventilation and mortality rates in HF patients with COVID-19, with HF independently predicting mortality and other complications [70, 71]. Notably, a 1.72 hazard ratio for developing HF was observed 12 months post-COVID-19, establishing a causal link [72]. Mechanisms for these adverse impacts include increased adrenergic stimulation, myocardial damage from viral infection, and cytokine storm effects [73].

Cardiac cases due to covid-19 in world

In China, a two-month-old infant developed pneumonia, liver impairment, and heart damage [74]. Of those without underlying cardiovascular disease, 11.8% had significant cardiac damage [75]. Given that four out of five patients with COVID-19 developed myocardial injury and were hospitalized to the intensive care unit (ICU), this condition is significant. Myocardial damage associated with SARS-CoV-2 was found in five of the 41 patients in Wuhan initially diagnosed with COVID-19 [1]. Of the 187 COVID-19 patients, 77.7% were released, while 23.3% died. Most of the 66 patients had underlying cardiovascular disease (CVD), with coronary heart disease, cardiomyopathy, and hypertension.

A total case death rate (CFR) of 1.67% was recorded in Pakistan. The middle age of the 75 patients, 75 percent of whom were men, was 64.5 years old. Of all recorded deaths, 71 (71%) had one or more recognized conditions at the time of diagnosis. The most often reported co-morbidities were hypertension (67%) and ischemic cardiovascular disease (27%), followed by diabetes mellitus (945%). Fever (79%) and dyspnea (87%) were the most common presenting symptoms, and the first recorded death occurred on March 18, 2020 [77]. There were approximately 19,752 fatalities and 882,928 impacted individuals in Pakistan as of May 17, 2021 [78].

In India, 20% to 30% of hospitalized patients appear to have heart damage, and 40% of COVID-19-related deaths are caused by cardiac issues [76,79]. 94 youngsters from 20 Indian heart centers were afflicted by COVID-19. Nearly half of them were men, and 88% of them were young and had cardiac issues. The majority were children, with 42% and 33%, respectively, suffering cyanotic heart disease. For their heart problems, over 80 percent had not had surgery. Thirty patients showed signs of infection, and thirteen people died [80].

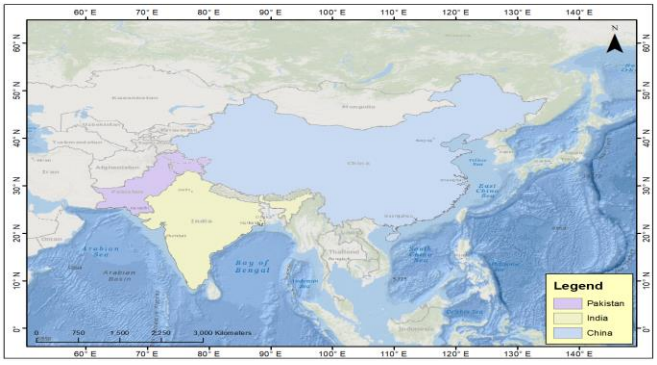

Figure 3. Cardiac cases due to Covid-19 in Pakistan, India and China

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ISSN: 3106-5937 | 3106-5945
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Treatment

The COVID-19 pandemic has upset the balance of healthcare systems around the world, which may significantly impact the prognosis of individuals with acute cardiovascular disease [81]. In the current pandemic scenario, alternative techniques are required for the best cardiovascular patient monitoring and care [82]. Digital health care may be a new and innovative way to protect cardiovascular patients from corona virus disease by avoiding frequent journeys to hospitals already packed with COVID-19 patients [82]. COVID-19 may be slowed down by treatments and immunizations, but they may also have negative effects on the heart, which could compromise cardiovascular health [83]. Managements during covid-19 are shown in Figure 4.

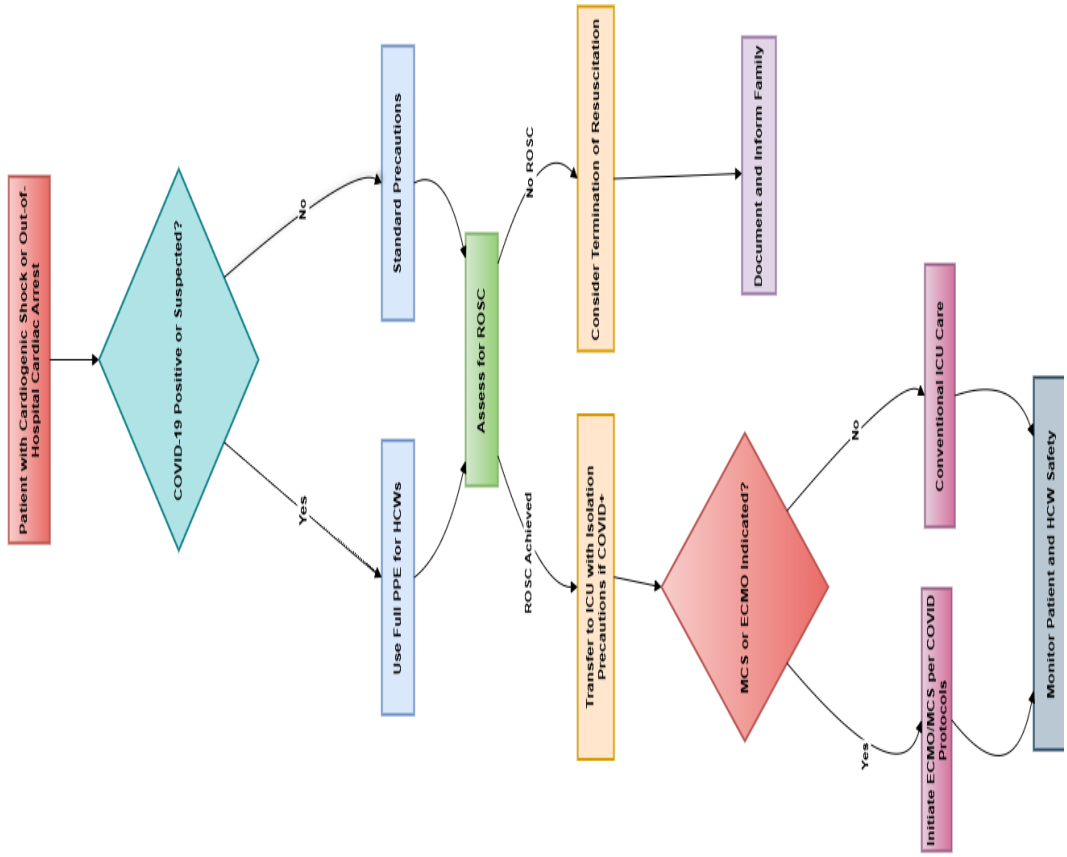

Figure.4 Managements of patients with cardiogenic shock during pandemic

To avoid Covid-19, take precautions as illustrated in Figure 5.Four well-known mRNA vaccines are AZD1222, BNT162b2, mRNA-1273, and Ad26.COV2.S. These lipid nano-particles contain nucleoside-modified mRNA containing the SARS-CoV-2 spike protein sequence, which triggers an immune response [84, 85].

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Figure 5 Preventive measures

Conclusions

It is crucial to keep assessing how the unique SARS-CoV-2 virus spreads and how its harmful health effects appear, especially in light of the unusual initial manifestation and subsequent pandemic it produced. Despite being primarily thought of as a respiratory virus, COVID-19's systemic impacts are likely to cause long-term health issues that affect and are not related to the respiratory system. Gaining a better understanding of this virus and other viral infections requires an understanding of its systemic pathophysiology, which includes its impact on the cardiovascular system.

This paper emphasizes how COVID-19 can significantly damage the cardiovascular system, causing myocardial tissue to dramatically malfunction. This damage is caused by a number of processes, such as the virus attaching to endothelial cells and cardiac myocytes, causing a cytokine storm, and causing tissue hypoxia. Pre-existing comorbidities, including as diabetes mellitus, hypertension, and cardiovascular disease, may make the virus's detrimental effects on the cardiovascular system worse. The various ways that cardiac problems manifest in COVID-19 patients lend credence to the microbe's complex pathogenicity in the cardiovascular system. Therefore, in order to speed up their identification and enable prompt treatment, any cardiovascular consequences from this illness should be evaluated in infected patients.

Antiviral treatments and antithrombolytic medications are required for the treatment and management of severe infections. One of the most important ways to counteract the virulence of COVID-19, which persists worldwide despite declining infection rates, is to prevent the disease by immunization. The hazards of vaccination, including myocarditis, are still far lower than those of COVID-19 infection, despite the fact that myocarditis is an uncommon adverse event linked to mRNA vaccines. The scientific community must thus keep informing the public about the safety and efficacy of vaccines.

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