

Cardiovascular complications of COVID-19

Complicaciones cardiovasculares en COVID-19

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Abstract

SARS-CoV-2 infection is associated with a broad spectrum of cardiovascular manifestations that significantly increase morbidity and mortality. Acute myocardial injury, detected by elevated cardiac troponins, occurs in 10-25% of hospitalized patients and serves as an independent predictor of mortality, particularly in critically ill patients and those with pre-existing cardiovascular disease. Acute complications include acute myocarditis, rhythm disorders, heart failure, and cor pulmonale. The pathophysiology is multifactorial, involving direct viral damage via ACE2 receptors, oxygen supply-demand mismatch, and the “cytokine storm.” In the post-acute phase (≥ 4 weeks), an elevated risk of arrhythmic events, heart failure, and coagulation disorders persists. The cardiovascular safety profile of current antiviral and immunomodulatory therapies varies, ranging from an increased risk of arrhythmias to a reduction in long-term cardiac complications. Cardiovascular complications of COVID-19 are frequent and heterogeneous. Close monitoring of biomarkers and echocardiographic evaluation are essential for timely management, both in the critical stage and during the long-term follow-up of survivors.

Keywords: COVID-19. Cardiovascular diseases. Myocarditis. Cardiac arrhythmias. Post-acute COVID-19 syndrome.

Resumen

La infección por SARS-CoV-2 se asocia con un espectro amplio de manifestaciones cardiovasculares que incrementan significativamente la morbimortalidad. El daño miocárdico agudo, detectado por la elevación de troponinas cardíacas, se presenta en el 10-25% de los pacientes hospitalizados y es un predictor independiente de mortalidad, especialmente en pacientes críticos y aquellos con enfermedad cardiovascular preexistente. Las complicaciones agudas incluyen miocarditis aguda, trastornos del ritmo, insuficiencia cardíaca y cor pulmonale. La fisiopatología es multifactorial, involucrando daño viral directo vía receptores ACE2, desequilibrio entre demanda y aporte de oxígeno, y la «tormenta de citocinas». En la fase postaguda (≥ 4 semanas) persiste un riesgo elevado de eventos arrítmicos, insuficiencia cardíaca y alteraciones de la coagulación. El perfil de seguridad cardiovascular de las terapias antivirales e inmunomoduladores actuales varía desde mayor riesgo de arritmias, hasta la reducción del riesgo de complicaciones cardíacas a largo plazo. Las complicaciones cardiovasculares de la COVID-19 son frecuentes y heterogéneas. La vigilancia estrecha de biomarcadores y la evaluación ecocardiográfica son fundamentales para el manejo oportuno, tanto en la etapa crítica como en el seguimiento a largo plazo de los supervivientes.

Palabras clave: COVID-19. Enfermedades cardiovasculares. Miocarditis. Arritmias cardíacas. Síndrome postagudo de COVID-19.

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Date of reception: 28-11-2025
Date of acceptance: 23-04-2026
DOI: 10.24875/NCTE.M26000030

Available online: 21-10-2026
Neumol Cir Torax (Eng). 2025;84(4):304-309
www.revistanct.org.mx

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Introduction

The American College of Cardiology and the American Heart Association recognize that COVID-19 causes various cardiovascular complications affecting patients with or without pre-existing cardiovascular disease. Approximately 10 to 20% of hospitalized patients present evidence of myocardial injury.¹ Myocardial damage or injury was a concept established in the 2018 European Society of Cardiology consensus on the fourth universal definition of myocardial infarction; it is defined as the detection of cardiac troponins with at least one value above the 99th percentile upper reference limit; acute myocardial injury is considered when there is a rise or fall in cardiac troponin values on serial measurements. The causes that may lead to this are diverse and include cardiac and extracardiac conditions.²

Cardiovascular complications of COVID-19 extend beyond the acute phase, affecting multiple organ systems. [Table 1](#) summarizes the complications associated with severe acute COVID-19 infection. Myocardial injury occurs in approximately 25% of hospitalized patients and is associated with an increased risk of mortality.³ The mechanisms are heterogeneous and include oxygen supply-demand mismatch, direct viral invasion of the myocardium, inflammation, coronary plaque rupture with acute myocardial infarction, microvascular thrombosis, and adrenergic stress.³

Acute myocardial injury was progressively documented as reports and studies emerged from China, Europe, and the United States. Some registries report an incidence of between 7 and 17%,⁴ while other reports place it between 20 and 29%.⁵ Myocardial injury was more frequent in patients admitted to the intensive care unit (ICU) (22 vs. 2%) and more common among patients who had died (59 vs. 1%).⁴ Subsequent studies found that patients without cardiovascular disease and normal troponin had a mortality rate of 7.62%, while patients with cardiovascular disease and normal troponin had a mortality rate of 13.3%, patients without cardiovascular disease and elevated troponin had a mortality rate of 37.5%, and patients with cardiovascular disease and elevated troponin had a mortality rate of 69.4%.⁶ In another study including 671 patients with severe COVID-19, it was found that the risk of in-hospital death could be predicted by markers of myocardial injury such as myoglobin, troponin I, and the MB fraction of creatine phosphokinase (CPK-MB), since when these are elevated in plasma, survival decreases. The risk is greater in patients of advanced age, with cardiovascular comorbidities, and with increased expression of pro-inflammatory molecules.⁷

Acute myocardial injury is observed most frequently in the critically ill patients most severely affected by SARS-CoV-2, approximately 5% of all those infected; it generally occurs between 12 and 20 days after symptom onset. The age group most affected is those over 65 years of age. It is at this stage that severe acute respiratory distress syndrome (ARDS) and multiple organ failure may develop.⁸ Based on these findings, some investigators have proposed treatment algorithms in which, faced with an increase in plasma troponin (I or T), ICU admission is recommended, along with close hemodynamic monitoring and adequate echocardiographic evaluation, in order to limit the negative impact of acute myocardial injury on survival.⁹ The various cardiovascular complications may be present in any patient affected by COVID-19 and may manifest in both acute and long-term forms.⁵

Acute cardiovascular complications associated with COVID-19

Acute cardiovascular complications include myocarditis, acute coronary syndrome, heart failure (HF), cardiogenic shock, rhythm disorders (both atrial and ventricular arrhythmias), and thromboembolic and cerebrovascular complications.¹ The proposed mechanisms involve the activation of inflammatory and thrombotic cascades, direct viral damage to myocytes or the vascular endothelium, and worsening of underlying atherosclerotic and structural abnormalities.¹ COVID-19 can trigger acute HF or cardiogenic shock through new-onset left ventricular systolic dysfunction (related to myocarditis, endothelial and microvascular injury, myocardial stress due to increased demand and reduced oxygenation, and a sudden surge in pro-inflammatory cytokines) or new-onset right ventricular dysfunction (due to acute pulmonary embolism or ARDS-related pressure overload).¹ The infection is associated with an increased risk of stroke, transient ischemic attack (TIA), and venous and arterial thromboembolic events.¹

Acute myocarditis

Acute myocarditis is a significant cardiovascular complication in the context of COVID-19, contributing notably to increased morbidity and mortality. This condition is characterized by myocardial injury, which may result from direct damage to cardiac tissue caused by the virus as well as from extrapulmonary migration of infected alveolar macrophages, predisposing patients

Table 1. Acute cardiovascular complications associated with COVID-19

Cardiovascular complication	Main pathophysiological mechanisms	Manifestations/relevant findings	Clinical impact
Acute myocarditis	Direct viral damage to the myocardium, migration of infected macrophages, cytokine storm (IL-1, IL-6, TNF- α), CD8 T-lymphocyte activation	Myocardial inflammation, QT prolongation, inflammatory channelopathies, arrhythmias	Increased morbidity and mortality and elevated risk of arrhythmias
Rhythm disorders (arrhythmias)	Systemic inflammation, direct viral damage, sympathetic activation, hemodynamic impairment	Atrial fibrillation, ventricular fibrillation, ventricular tachycardia, premature ventricular contractions	Atrial fibrillation is associated with higher in-hospital mortality
Heart failure	Inflammatory storm, hypoxemia, increased oxygen demand, type 1 and type 2 myocardial infarction, right ventricular dysfunction due to ARDS or PE	<i>De novo</i> acute HF or decompensation of pre-existing HF, cardiogenic shock, sudden death	High mortality, particularly associated with septic shock
Cardiogenic shock	Left or right ventricular systolic dysfunction, severe inflammation, myocardial injury	Systemic hypoperfusion, multiorgan failure	Very high mortality
Thromboembolic and cerebrovascular complications	Endothelial dysfunction, inflammation, oxidative stress, platelet activation	Stroke, TIA, venous and arterial thromboembolism	Increased risk of thrombotic events and mortality
ACP in ARDS	Increased right ventricular afterload, mechanical ventilation, pulmonary hypertension, ACE2 dysregulation	Right ventricular dysfunction, severe hypoxemia	Frequent complication of COVID-19-associated ARDS
Myocardial injury	Inflammation, hypoxia, endothelial and microvascular damage	Elevated troponin and CK-MB	Associated with worse prognosis and higher mortality

ACE2: angiotensin-converting enzyme 2; TIA: transient ischemic attack; CK-MB: MB fraction of creatine kinase; ACP: acute cor pulmonale; HF: heart failure; IL-6: interleukin 6; ARDS: acute respiratory distress syndrome; PTE: pulmonary thromboembolism; TNF- α : tumor necrosis factor alpha.

to cardiac arrhythmias. A key element in the development of myocarditis is the so-called “cytokine storm,” in which large quantities of pro-inflammatory cytokines are released into the circulation and T-lymphocyte activation is amplified. CD8 T-lymphocytes migrate to cardiomyocytes, causing myocardial inflammation.^{10,11}

The most relevant cytokines in this process include interleukin (IL)-1, IL-6, and tumor necrosis factor alpha (TNF- α); these molecules can modulate the expression and function of potassium and calcium channels, generating inflammatory cardiac channelopathies and causing prolongation of the ventricular action potential. Inflammatory cytokines are a well-known trigger of cardiac arrhythmias. This results from overactivation of the cardiac sympathetic nervous system, mediated both by the inflammatory reflex involving the hypothalamus and by peripheral activation via the stellate ganglion pathway, which can prolong the QT interval on the electrocardiogram. It is important to note that IL-6 inhibits cytochrome P450 (CYP450), thereby increasing the bioavailability of drugs that prolong the QT interval, thus raising the risk of arrhythmias in patients with

acute myocarditis; this mechanism may be similar to that observed in acute myocarditis associated with COVID-19.¹²

Rhythm disorders

Arrhythmias are common, and cases of atrial fibrillation (AF) and ventricular fibrillation (VF), as well as ventricular tachyarrhythmias, have been reported.¹³ The American Heart Association notes that the mechanisms include direct autonomic effects, hemodynamic impairment, inflammation, and direct viral damage, with SARS-CoV-2 cardiac infiltration occurring primarily in the interstitium.¹⁴ Increased sympathetic activation and adverse hemodynamic consequences persist during recovery.¹⁴

A single-center observational study, whose objective was to investigate the incidence of AF in hospitalized patients with COVID-19 and understand its impact on the in-hospital mortality rate, reported that 17.6% presented AF (12.5% new-onset). AF was an independent predictor of in-hospital mortality (relative risk: 1.56).¹⁵ It remains unknown whether this high incidence of AF

and the associated excess mortality are specific to COVID-19 or, rather, manifestations of disease severity and systemic inflammation. A multicenter retrospective cohort study of 3,970 patients hospitalized with COVID-19 found an AF incidence of approximately 13%, similar to that of a comparative cohort admitted with severe influenza.¹⁶

Premature ventricular contractions, non-sustained ventricular tachycardia (VT), and sustained VT/VF have been observed in patients with acute SARS-CoV-2 infection. Myocardial injury was associated with mortality, whereas those with underlying heart disease but without myocardial injury had a more favorable prognosis. In patients with COVID-19, the overall prevalence of ventricular arrhythmias ranges from 0.1 to 8% according to primarily retrospective data.¹⁷ In a retrospective study of 187 patients hospitalized with confirmed COVID-19 at the beginning of the pandemic in China, patients with elevated troponin T levels had a higher probability of VT/VF compared to those with normal troponin levels (11.5 vs. 5.2%).⁶ In a prospective observational study of 143 patients hospitalized in the United States with COVID-19 and monitored by telemetry, 1.4% presented sustained VT and 0.7% presented VF.¹⁸

Heart failure

SARS-CoV-2 infection can cause or worsen HF through a wide variety of mechanisms that can simultaneously lead to arrhythmias, cardiogenic shock, and sudden cardiac death.¹⁹ Acute infections trigger the release of pro-inflammatory cytokines and the recruitment of pro-inflammatory macrophages and granulocytes, leading to a severe inflammatory storm that can exacerbate the initial injury. In combination with hypoxemic respiratory failure and increased myocardial oxygen demand, these processes can cause an oxygen supply-demand mismatch and precipitate a type 2 myocardial infarction; both can trigger decompensation of pre-existing HF or the development of *de novo* acute HF. Right ventricular failure may also develop secondary to elevated pulmonary pressures in the context of ARDS and/or pulmonary thromboembolism (PTE).²⁰ Another contributing factor to cardiac depression may be coagulation dysfunction induced by sepsis.²¹ Patients with COVID-19 have an increased risk of thrombosis in both the arterial and venous circulations due to endothelial dysfunction, inflammation, oxidative stress, and platelet activation.²² In this context, acute coronary syndromes with plaque rupture leading to type 1 myocardial infarction may occur. Generally, the combination of

HF and septic shock leads to a mortality rate of 70 to 90%, compared to 20% in septic patients without cardiovascular deterioration.²³

It has also been reported that in 15 to 29% of patients with COVID-19, the virus causes renal failure in the context of acute kidney injury, which can lead to volume overload that may exacerbate chronic HF.²⁴ On the other hand, COVID-19-induced ARDS (parenchymal lung disease) can cause pulmonary hypertension and consequent right-sided HF.⁴

Elevated natriuretic peptides suggest HF with a worse prognosis in COVID-19 and justify performing an echocardiogram to better evaluate cardiac function. If HF is present as part of the infectious process, optimizing loading conditions is essential. Cardiac injury markers (MB fraction of creatine kinase [CK-MB] and troponin) tend to increase significantly more in patients with cerebrovascular disease risk factors and are associated with significantly higher mortality.

Acute cor pulmonale in COVID-19-related ARDS

The pathophysiology of cardiovascular dysfunction related to SARS-CoV-2 may be attributed to the recognition of human angiotensin-converting enzyme 2 (ACE2) by SARS-CoV-2. The virus uses ACE2 as a functional receptor to enter the cell.²⁵ Furthermore, the ACE2 receptor is not only present in alveolar cells of the lung, but also in many extrapulmonary tissues, particularly the heart and vascular endothelium. Therefore, direct viral effects on the heart and blood vessels can cause right ventricular (RV) dysfunction. Another possible cause is the impact of acute respiratory distress syndrome (ARDS) and mechanical ventilation on the RV. Acute cor pulmonale (ACP) is a well-known complication of ARDS despite protective ventilation, with an incidence of 25%. Therefore, ACP may be related to high driving pressure, leading to increased RV afterload.²⁶ Some authors have hypothesized that hypoxemia related to SARS-CoV-2 is due to impairment of hypoxic pulmonary vasoconstriction and dysregulation of pulmonary blood flow, leading to intrapulmonary shunting responsible for severe hypoxemia.²⁷ ACP may occur in patients with SARS-CoV-2-related ARDS, and echocardiography is the primary tool for diagnosing ACP.²⁸

Long-term cardiovascular complications

Long-term cardiovascular manifestations occurring ≥ 4 weeks after initial infection (post-acute COVID-19

Table 2. Comparison of cardiac effects of drugs used in COVID-19

Drugs	Reported cardiac risks	Cardiovascular benefits
Remdesivir	Bradycardia and hypotension	Shortens recovery time in hospitalized patients with severe disease
Paxlovid (nirmatrelvir/ritonavir)	Primarily critical drug interactions with cardiovascular medications. Cases of mild hypertension have been reported	Significant reduction in the risk of cardiovascular mortality and complications such as arrhythmias and myocardial infarctions in the short and long term (up to 1 year)
Molnupiravir	Minimal effects; no substantial cardiovascular risks have been identified in current studies	Reduction of short-term cardiac complications (first 21 days), although its long-term benefit is less than that of Paxlovid

syndrome) may include chest pain, palpitations, inappropriate sinus tachycardia, postural orthostatic tachycardia syndrome, atrial arrhythmia, cardiomyopathy, and thromboembolism.¹ The possible mechanisms include direct and indirect virus-mediated cellular damage, a procoagulant state, an immune response affecting the structural integrity of the myocardium and pericardium, and downregulation of ACE2.¹ Myocardial abnormalities on magnetic resonance imaging and cardiac troponin elevations > 2 months after COVID-19 diagnosis have been reported, with myocardial fibrosis or scarring potentially leading to arrhythmias.¹

Post-COVID-19 syndrome

Post-acute cardiovascular complications occur even in non-hospitalized individuals or those with mild symptoms. The risk of cardiovascular complications increases significantly during the first six months following infection, with evidence of myocardial injury, HF, arrhythmias, and coagulation disorders persisting beyond 30 days.²⁹ Older patients with hypertension, diabetes, and a history of vascular disease have worse outcomes and are more likely to develop long-term cardiovascular complications.²⁹

Cardiac arrhythmias and peripheral arterial disease have been associated with acute, subacute, and post-COVID-19 infection.¹⁷ Patients who develop COVID-19 myocarditis may also have an increased risk of VT or VF and may require more prolonged arrhythmia follow-up.³⁰ Long-term arrhythmic outcomes following SARS-CoV-2 infection were evaluated in approximately 4.1 million patients in the United States Collaborative Network (TriNetX). During one-year follow-up (initiated 30 days after the initial positive test), COVID-19 survivors presented a significantly higher risk of developing arrhythmic events. A higher hazard ratio (HR) was observed for atrial fibrillation/flutter (HR: 2.41; 95% CI: 2.30-2.52), sinus tachycardia (HR: 1.68; 95% CI: 1.63-1.74),

bradycardia (HR: 1.60; 95% CI: 1.52-1.68), and ventricular arrhythmias (HR: 1.60; 95% CI: 1.54-1.67).³⁰

COVID-19 treatments and their cardiovascular implications

The high mortality of COVID-19 required rapid drug approval, but cardiovascular adverse effects may not emerge in initial trials due to small sample sizes and short durations. Post-marketing surveillance is essential for detecting cardiovascular safety signals from COVID-19 therapies, particularly because these medications may be repurposed for future pandemics.³¹ Small-molecule antivirals (remdesivir, molnupiravir, nirmatrelvir/ritonavir) and immunomodulators (baricitinib, tocilizumab) have been developed or repurposed to suppress viral replication and ameliorate cytokine storms, respectively.³¹ The cardiovascular safety profiles of these antivirals vary significantly. Whereas remdesivir is associated with an increased risk of arrhythmias (such as bradycardia), Paxlovid and molnupiravir generally demonstrate a reduction in long-term cardiac complications derived from COVID-19, as summarized in table 2.³²

Funding

The present study did not receive any funding.

Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of persons and animals. The authors declare that no experiments involving human beings or animals were performed for this investigation.

Confidentiality, informed consent, and ethical approval. The study does not involve personal patient

data and does not require ethical approval. SAGER guidelines do not apply.

Statement on the use of artificial intelligence. The authors declare that they did not use any form of generative artificial intelligence in the writing of this article.

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