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Macit Kalcik *

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Review Article

Long-Term Cardiovascular Effects of COVID-19: Mechanisms, Clinical Phenotypes, and Management Strategies

Mucahit Aker [1]*, Macit Kalçik [2], Mucahit Yetim [2], Muhammet Cihat Çelik [1], Lütfü Bekar [2], Yusuf Karavelioğlu [2]

[1]Department of Cardiology, Hitit University Erol Olçok Education and Research Hospital, Corum, Turkey.

[2]Department of Cardiology, Facult of Medicine, Hitit University, Corum, Turkey.

***Corresponding Author:** Macit Kalcik, Department of Cardiology, Hitit University Faculty of Medicine, Çorum, Turkey.

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Abstract

The long-term cardiovascular effects of COVID-19, also known as post-acute sequelae of SARS-CoV-2 infection (PASC), encompass a wide spectrum of conditions ranging from subclinical abnormalities to clinically significant disease. Reported outcomes include new-onset heart failure, arrhythmias, ischemic and non-ischemic heart disease, myocarditis, pericarditis, thromboembolic events, and cerebrovascular complications. Importantly, these risks have been observed not only in patients who experienced severe acute illness but also in those with mild or even asymptomatic infections. Multiple mechanisms have been proposed to explain these sequelae, including persistent inflammation, endothelial dysfunction, immune dysregulation, direct viral injury, microthrombosis, and autonomic imbalance. Evidence from imaging modalities such as echocardiography and cardiac magnetic resonance has demonstrated structural and functional changes months after infection, while biomarker studies support the presence of ongoing myocardial stress and injury. The clinical burden of cardiovascular sequelae after COVID-19 is substantial and may persist for months to years, creating challenges for health systems worldwide. This review summarizes current knowledge on mechanisms, clinical phenotypes, diagnostic approaches, and management strategies, while also highlighting gaps in evidence and priorities for future research.

Keywords: long covid; cardiovascular complications; post-acute sequelae of sars-cov-2 infection (pasc); myocardial injury; endothelial dysfunction

Introduction

COVID-19 caused by SARS-CoV-2 has had immediate impacts on respiratory and systemic health; increasingly, evidence shows that its cardiovascular effects persist long after acute infection. Individuals who recover from COVID-19 may show elevated incidence of cardiovascular disease (CVD), including heart failure, arrhythmias, myocarditis, pericarditis, ischemic disease, thromboembolism, even if acute illness was mild.

The terms **"Long COVID"** or **Post-Acute Sequelae of SARS-CoV-2 infection (PASC)** are used to describe symptoms or new conditions that last or appear more than 4 weeks after infection, often divided into 4–12 weeks (ongoing symptomatic COVID-19) and ≥12 weeks (post-COVID-19 syndrome) categories. Definitions vary but many major papers use ≥4 weeks and ≥12 weeks thresholds. One review used 4 weeks beyond acute COVID-19 for persistent symptoms and ≥12 weeks for chronic sequelae. [1]

Large cohort studies quantify the cardiovascular burden long term. For example, in a US Veterans Affairs cohort of ~153,760 people with COVID-19 vs millions of control individuals, those who recovered had significantly higher risk of incident cardiovascular disease across multiple categories (dysrhythmias, ischemic and non-ischemic heart disease, myocarditis, pericarditis, heart failure, thromboembolic disease) over 1 year; importantly, risk was elevated even in non-hospitalized patients and increased with severity of acute illness.[2] Meta-analysis and systematic reviews show that recovering individuals more frequently report cardiovascular symptoms (palpitations, chest pain, etc.) compared to uninfected controls. [3]

Because COVID-19 survivors are a large and growing population, understanding mechanisms, the spectrum of cardiovascular phenotypes, diagnostic strategies, potential treatments, and gaps in knowledge is essential. This review will cover: (a) pathophysiology; (b) clinical phenotypes & epidemiology; (c) diagnostic/imaging findings; (d) management strategies; (e) prognosis & health-system implications; and (f) research agenda.

Pathophysiology and Mechanisms

Several mechanisms are proposed to explain the cardiovascular sequelae observed after COVID-19. Direct viral invasion of cardiomyocytes has been suggested, supported by autopsy studies identifying viral RNA and protein in cardiac tissue, although the extent of this process and its clinical

relevance remain debated [4]. Persistent immune activation and systemic inflammation are considered central drivers, with cytokine dysregulation leading to myocardial injury, remodeling, and arrhythmic substrate formation [5].

Endothelial dysfunction is another important pathway. SARS-CoV-2 can infect endothelial cells, leading to impaired nitric oxide bioavailability, oxidative stress, and microvascular inflammation. These processes create a pro-thrombotic milieu that contributes to both acute and long-term vascular complications [6]. Microthrombosis in the myocardium and systemic circulation has been demonstrated in pathological studies, raising concern for chronic ischemia and tissue injury.

Autonomic imbalance is increasingly recognized as a mechanism underlying persistent tachycardia, palpitations, and postural orthostatic tachycardia syndrome in Long COVID. Dysfunction of the autonomic nervous system may result from viral or immune-mediated injury to autonomic pathways, compounded by systemic inflammation and deconditioning [7].

Mitochondrial dysfunction and impaired energy metabolism have also been reported. Studies of skeletal muscle and cardiomyocytes in COVID-19 survivors have revealed alterations in mitochondrial structure and function, which may contribute to exercise intolerance, fatigue, and cardiac dysfunction [8].

Finally, there is growing interest in the potential for accelerated atherosclerosis. Chronic low-grade inflammation and endothelial injury could promote plaque instability and vascular stiffness, predisposing survivors to premature cardiovascular events [9]. Together, these mechanisms interact to produce the heterogeneous cardiovascular phenotypes observed in post-COVID conditions.

Clinical Phenotypes and Epidemiology

Myocardial injury and fibrosis are among the most frequently reported sequelae of COVID-19. Cardiac magnetic resonance imaging has demonstrated persistent late gadolinium enhancement and myocardial edema in survivors several months after infection, even in those with mild disease, suggesting subclinical myocarditis and tissue remodeling [10]. Elevated troponin levels during acute infection have been associated with worse long-term outcomes, and some patients develop new-onset dilated cardiomyopathy following recovery [11].

Heart failure is a significant clinical phenotype of long COVID. Both reduced and preserved ejection fraction patterns have been observed. Cohort studies show an increased incidence of heart failure in COVID-19 survivors compared with matched controls, with risk correlating to the severity of the acute illness but remaining elevated even among non-hospitalized patients [12].

Arrhythmias represent another common manifestation. Atrial fibrillation, supraventricular tachycardia, ventricular arrhythmias, and bradyarrhythmias have all been documented. Mechanisms include direct myocardial injury, autonomic dysfunction, and structural remodeling. Large observational datasets confirm higher rates of arrhythmias up to one year post-infection [13].

Thromboembolic complications extend into the post-acute phase. Persistent hypercoagulability and endothelial dysfunction predispose patients to deep vein thrombosis, pulmonary embolism, and arterial thrombosis. Evidence indicates that venous and arterial thrombotic events remain more frequent in survivors for months after infection compared to uninfected individuals [14].

Dysautonomia and postural orthostatic tachycardia syndrome are increasingly recognized. Patients often report palpitations, dizziness, and exercise intolerance. Objective testing has confirmed postural heart rate increases consistent with POTS in a subset of long COVID patients, implicating autonomic nervous system injury and deconditioning [15].

Pericardial involvement has been described, including persistent pericarditis and pericardial effusion. Although less common, these cases highlight the diverse spectrum of post-COVID cardiovascular pathology [16].

Special populations deserve attention. Competitive athletes recovering from COVID-19 have shown higher rates of subclinical myocarditis on cardiac MRI, prompting development of structured return-to-play protocols. Children with multisystem inflammatory syndrome (MIS-C) demonstrate acute myocardial dysfunction, some of which persists into recovery, while elderly and comorbid patients face compounded risks for long-term cardiac complications [17].

Together, these clinical phenotypes underline the heterogeneous presentation and significant burden of cardiovascular sequelae following COVID-19, with implications for diagnosis, monitoring, and long-term care (Table 1).

Phenotype	Clinical Features	Suggested Tests	Typical Findings
Myocarditis / Fibrosis	Chest pain, dyspnea, elevated troponin	Cardiac MRI, ECG, hs-Tn	LGE, ↑ T1/T2, ST-T changes
Heart Failure	Fatigue, edema, reduced EF	Echocardiography (EF, GLS), BNP/NT-proBNP	Reduced EF, diastolic dysfunction
Arrhythmias	Palpitations, syncope	ECG, Holter, ILR	AF, SVT, VT, bradyarrhythmias
Thromboembolism	Dyspnea, chest pain	D-dimer, CT angiography	PE, DVT, arterial thrombosis
Dysautonomia / POTS	Orthostatic tachycardia, dizziness	Tilt test, Holter	HR ↑ ≥30 bpm upright
Pericardial Involvement	Chest pain, effusion	Echo, MRI	Pericardial fluid, pericarditis

Table 1: Cardiovascular Phenotypes in Long COVID and Diagnostic Approaches

***Abbreviations**: LGE: Late gadolinium enhancement; MRI: Magnetic resonance imaging; ECG: Electrocardiogram; hs-Tn: High-sensitivity troponin; EF: Ejection fraction; GLS: Global longitudinal strain; BNP: B-type natriuretic peptide; NT-proBNP: N-terminal pro–B-type natriuretic peptide; ILR: Implantable loop recorder; PE: Pulmonary embolism; DVT: Deep vein thrombosis; POTS: Postural orthostatic tachycardia syndrome; HR: Heart rate.*

Diagnostic and Imaging Approaches

Electrocardiography remains a fundamental tool in the evaluation of patients with suspected cardiovascular sequelae after COVID-19. Persistent ST-T abnormalities, conduction disturbances, and arrhythmias have been reported in survivors. Holter monitoring and implantable loop recorders can provide extended rhythm surveillance, especially in patients with palpitations, syncope, or unexplained tachycardia [18].

Echocardiography is widely used for structural and functional assessment. Reduced ejection fraction, regional wall motion abnormalities, diastolic dysfunction, and impaired global longitudinal strain have been observed in patients months after recovery. Strain imaging in particular has

revealed subtle left ventricular dysfunction not evident on conventional parameters [19].

Cardiac magnetic resonance imaging offers the most sensitive noninvasive method to detect myocardial inflammation, fibrosis, and edema. Studies demonstrate that late gadolinium enhancement and T1/T2 mapping abnormalities can persist well beyond the acute phase, even in asymptomatic individuals. These findings provide evidence for subclinical myocarditis and may predict adverse outcomes [20].

Functional capacity assessment plays a critical role in long COVID evaluation. Cardiopulmonary exercise testing (CPET) has revealed reduced peak oxygen consumption and abnormal ventilatory efficiency in a substantial subset of patients. These findings suggest impaired cardiovascular reserve, autonomic dysfunction, or lingering pulmonary effects [21]. The six-minute walk test provides a simple measure of exercise tolerance, often reduced in symptomatic individuals.

Biomarkers complement imaging and functional tests. High-sensitivity troponin and NT-proBNP may remain elevated beyond the acute phase, indicating ongoing myocardial injury or stress. Elevated D-dimer and inflammatory markers such as CRP and IL-6 have also been reported in survivors, supporting the presence of persistent pro-thrombotic and inflammatory activity [22].

Together, multimodal diagnostic approaches are recommended for comprehensive evaluation of post-COVID cardiovascular complications. The choice of tests should be guided by clinical presentation, severity of prior infection, and ongoing symptom burden **(Table 2).**

Biomarker	Clinical Role	Findings	Limitations
hs-Troponin	Myocardial injury	Persistent elevation	Not disease-specific
NT-proBNP	Heart failure screening	Subclinical dysfunction	Influenced by renal function
D-dimer	Thromboembolism risk	Sustained elevation	Non-specific
IL-6, CRP	Ongoing inflammation	Elevated in long COVID	Non-specific
Ferritin	Cytokine response	Increased levels	Non-specific

Table 2: Biomarkers and Their Clinical Utility

Abbreviations: *hs-Troponin: High-sensitivity troponin; NT-proBNP: N-terminal pro–B-type natriuretic peptide; CRP: C-reactive protein; IL-6: Interleukin-6.*

Management: Evidence-Based Recommendations

The management of long-term cardiovascular sequelae after COVID-19 relies on general principles of cardiovascular care, adapted to the unique pathophysiology of the disease. Risk stratification is essential, taking into account severity of the acute illness, pre-existing comorbidities, biomarker abnormalities, and imaging findings. Multidisciplinary post-COVID clinics that include cardiology expertise have been recommended to optimize patient outcomes [23].

For heart failure following COVID-19, guideline-directed medical therapy should be applied as in non-COVID cases. This includes angiotensin-converting enzyme inhibitors or angiotensin receptor–neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter 2 inhibitors. Evidence from cohort studies suggests that patients who develop reduced ejection fraction after COVID-19 respond to standard regimens, although long-term prognosis remains uncertain [24].

Arrhythmias are managed according to existing guidelines, with rate or rhythm control strategies individualized to the patient. Anticoagulation should be considered based on standard stroke risk stratification tools such as CHA_2DS_2-VASc in atrial fibrillation. In survivors with increased arrhythmic burden, extended monitoring may be appropriate to guide therapy [25].

Pericarditis after COVID-19 is generally treated with nonsteroidal anti-inflammatory drugs and colchicine. Corticosteroids may be considered in refractory cases, though their use is limited due to potential side effects and uncertain impact on viral-related pericardial inflammation [26].

Management of dysautonomia and postural orthostatic tachycardia syndrome involves non-pharmacologic interventions as first-line therapy, including volume expansion with increased fluid and salt intake, use of compression garments, and graduated exercise programs. Pharmacologic options such as beta-blockers, ivabradine, or fludrocortisone can be considered in refractory cases [27]

Extended thromboprophylaxis beyond acute illness remains controversial. While hypercoagulability persists in some survivors, routine anticoagulation is not universally recommended unless additional risk factors are present. Clinical trials are ongoing to clarify which subgroups may benefit from prolonged prophylaxis [28].

Rehabilitation strategies, particularly exercise-based programs, are essential (Table 3). Gradual, symptom-guided physical activity improves functional capacity and autonomic reconditioning. Return-to-play protocols for athletes should involve careful cardiovascular assessment including ECG, troponin testing, and in selected cases cardiac MRI to exclude myocarditis [29].

Follow-up is crucial. Patients with persistent symptoms or abnormal imaging should undergo reassessment at 3–6 month intervals, while those with normal initial evaluations may require less intensive monitoring. Individualized follow-up strategies can help identify late complications while avoiding unnecessary testing [30].

Condition	Treatment	Evidence / Recommendation
Heart Failure	GDMT (ACEi/ARB/ARNI, β-blocker, MRA, SGLT2i)	Class I – guideline-based
Arrhythmias	Rate/rhythm control, anticoagulation (AF: CHA_2DS_2-VASc)	Class I – guideline-based
Pericarditis	NSAIDs + colchicine, steroids if refractory	ESC recommendations, limited data
Dysautonomia / POTS	Salt/fluid, exercise, compression; β-blocker/ivabradine/fludrocortisone	Small case series, no RCTs
Increased Thrombotic Risk	Anticoagulation in selected patients	Evidence limited, ongoing trials

Table 3: Treatment Strategies and Level of Evidence

Abbreviations: *GDMT: Guideline-directed medical therapy; ACEi: Angiotensin-converting enzyme inhibitor; ARB: Angiotensin receptor blocker; ARNI: Angiotensin receptor–neprilysin inhibitor; MRA: Mineralocorticoid receptor antagonist; SGLT2i: Sodium–glucose cotransporter-2 inhibitor; AF: Atrial fibrillation; CHA_2DS_2-VASc: Congestive heart failure, Hypertension, Age ≥75, Diabetes, Stroke, Vascular disease, Age 65–74, Sex category; NSAID: Nonsteroidal anti-inflammatory drug; ESC: European Society of Cardiology; RCT: Randomized controlled trial.*

Prognosis and Health System Implications

The long-term prognosis of cardiovascular sequelae after COVID-19 remains an area of active investigation. Cohort studies demonstrate that survivors are at elevated risk for major adverse cardiovascular events including myocardial infarction, stroke, arrhythmias, heart failure, and cardiovascular death for at least 12 months after infection. Importantly, these risks extend to individuals who experienced only mild acute illness, suggesting that the burden of disease is not confined to hospitalized patients [31] **(Table 4).**

Parameter	Association with Long-Term Risk
Severe acute COVID (ICU, ventilation)	↑ Major adverse cardiovascular events
Persistent troponin elevation	↑ Heart failure, mortality
Cardiac MRI with LGE	↑ Arrhythmias, dysfunction
Ongoing symptom burden	Reduced quality of life, work disability
Comorbidities (HTN, DM, obesity, CKD)	↑ Adverse cardiovascular outcomes

Table 4: Prognostic Indicators and Risk Factors

Abbreviations: *ICU: Intensive care unit; LGE: Late gadolinium enhancement; CKD: Chronic kidney disease; DM: Diabetes mellitus; HTN: Hypertension.*

Hospital readmission and health care utilization rates are significantly higher among COVID-19 survivors compared to uninfected controls. Cardiovascular complications are a major contributor to these outcomes, driving both inpatient and outpatient encounters. Observational data indicate that health systems may face a sustained increase in demand for cardiovascular services related to long COVID [32].

Vaccination appears to attenuate the risk of long-term cardiovascular complications, although not fully eliminating it. Studies suggest that vaccinated individuals who develop breakthrough infections have lower rates of myocarditis, arrhythmias, and thromboembolic events compared to unvaccinated counterparts [33]. The specific effects of different viral variants on long-term cardiovascular outcomes are less well established, but preliminary evidence indicates variation in risk across waves of infection [34].

The socioeconomic and public health impact is substantial. Long COVID contributes to workforce absenteeism, reduced productivity, and increased disability claims. Cardiovascular sequelae, being among the more serious long-term complications, amplify this burden. Projections suggest a significant cumulative cost to health systems if even a small percentage of survivors develop persistent cardiovascular disease [35].

Overall, prognosis is heterogeneous, with outcomes depending on baseline cardiovascular health, severity of acute infection, vaccination status, and access to timely follow-up care. Health systems must adapt by integrating surveillance, multidisciplinary care pathways, and preventive strategies to mitigate the long-term impact of COVID-19 on cardiovascular health.

Knowledge Gaps and Research Agenda

Despite rapidly growing literature, important uncertainties remain regarding long-term cardiovascular sequelae of COVID-19. One major gap is the lack of standardized definitions for post-COVID cardiovascular outcomes. Heterogeneity in how studies define "long COVID" or "PASC" complicates comparisons across cohorts and meta-analyses. Consistent diagnostic criteria are needed to better quantify true incidence and risk [36].

Another gap is the limited number of prospective, longitudinal studies with pre-infection baselines. Most available data come from retrospective cohorts, administrative databases, or self-reported symptoms. Large-scale prospective studies with standardized imaging, biomarkers, and functional testing before and after infection are essential to establish causality and natural history [37].

Mechanistic understanding also remains incomplete. While immune dysregulation, endothelial dysfunction, and autonomic imbalance are implicated, their relative contributions and interactions are not fully elucidated. Advanced molecular studies, including multi-omics and immune profiling, may provide insight into persistent myocardial inflammation and vascular injury [38].

There is also a lack of randomized controlled trials targeting treatment of long-COVID cardiovascular syndromes. Evidence for pharmacologic strategies such as extended thromboprophylaxis, immunomodulation, or therapies for dysautonomia is sparse. Carefully designed interventional trials are needed to establish evidence-based management [39].

The role of viral variants and vaccination requires further clarification. Preliminary findings suggest variation in cardiovascular risks across variants and partial protection from vaccination, but data remain inconsistent. Longitudinal, variant-specific analyses will be important to refine prevention strategies [40].

Finally, global disparities in research must be addressed. Most evidence comes from high-income countries, while data from low- and middle-income regions remain scarce. Given differences in comorbidity prevalence, health system capacity, and vaccine access, international collaboration is critical to ensure findings are generalizable [41].

Addressing these gaps will require coordinated, multidisciplinary research efforts with standardized protocols, integration of cardiovascular endpoints into long-COVID studies, and sustained funding to track outcomes over years (Table 5).

Area	Current Status	Research Need
Definition	Heterogeneous	Standardized criteria (WHO, NIH)
Mechanisms	Multifactorial, unclear	Multi-omics, immune profiling
Long-term follow-up	Mostly <12 months	3–5 year prospective cohorts
Treatment	No evidence-based guidelines	RCTs (POTS, anticoagulation)
Vaccines/variants	Partial data	Variant-specific risk studies

Table 5: Research Gaps and Priorities

Abbreviations: *WHO: World Health Organization; NIH: National Institutes of Health; RCT: Randomized controlled trial.*

Conclusıon

COVID-19 has emerged not only as an acute viral illness but also as a condition with sustained cardiovascular consequences. Evidence from observational cohorts, imaging studies, and mechanistic investigations demonstrates that survivors face increased risks of heart failure, arrhythmias, myocardial injury, thromboembolic events, and autonomic dysfunction for months to years after infection. Importantly, these risks extend beyond those who were hospitalized, highlighting the widespread relevance of long COVID to cardiovascular health.

The underlying mechanisms are multifactorial, involving persistent inflammation, immune dysregulation, endothelial injury, microthrombosis, mitochondrial dysfunction, and autonomic imbalance. These processes contribute to the heterogeneous phenotypes observed across populations, ranging from subclinical abnormalities to clinically significant cardiovascular disease.

Diagnostic evaluation should be individualized, integrating electrocardiography, echocardiography, cardiac magnetic resonance, cardiopulmonary exercise testing, and biomarkers according to clinical presentation. Management is best guided by established cardiovascular principles, with adaptations for the post-COVID context, including targeted therapies for heart failure, arrhythmias, pericardial disease, dysautonomia, and carefully considered thromboprophylaxis. Rehabilitation and structured follow-up are essential components of care.

Prognosis remains variable, influenced by baseline comorbidities, severity of acute infection, vaccination status, and access to long-term monitoring. The burden on health systems and societies is substantial, emphasizing the need for integrated surveillance and preventive strategies. Addressing existing knowledge gaps will require standardized definitions, prospective longitudinal studies, mechanistic research, and randomized controlled trials of therapeutic approaches. International collaboration is particularly important to ensure findings are globally relevant.

In summary, long COVID represents a significant new domain in cardiovascular medicine. Clinicians must remain vigilant in identifying and managing these sequelae, while researchers and policymakers work to clarify mechanisms, optimize treatments, and mitigate the long-term impact of the pandemic on cardiovascular health.

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