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Research Article

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Cardiovascular Sequelae of COVID-19: A Narrative Review on Myocarditis, Microthrombosis, and Beyond

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Abstract

Background

SARS-CoV-2 infection has been linked to both acute and long-term cardiovascular (CV) complications.

Objective

Provide a narrative synthesis of evidence on cardiac remodeling, myocarditis, arrhythmia, microthrombosis, and heart failure following COVID-19.

Methods

We reviewed PubMed-indexed clinical and epidemiologic studies (2020–2025) addressing post-acute CV complications.

Results

COVID-19 survivors face elevated risks of myocarditis (~6×), arrhythmias (~1.6×), and heart failure (1.7–2×) even after mild disease [1-3]. Endothelial dysfunction, microthrombi, direct myocardial injury, and inflammation underpin these sequelae [3-5]. CMR studies in athletes show low prevalence of clinically relevant myocarditis (~1–3%), with subclinical fibrosis in a minority [6-8].

Conclusions

Persistent CV sequelae after COVID-19 are prevalent and clinically relevant; surveillance with imaging, biomarkers, and guideline-based therapy is imperative.

Keywords: COVID-19, Myocarditis, Microthrombosis, Cardiac Remodeling, Arrhythmia

1. Introduction

SARS-CoV-2 targets the CV system via ACE2 receptors, provoking systemic inflammation, endotheliitis, and thrombotic microangiopathy [1, 2]. Although acute cardiac injury during hospitalization is well-characterized, long-COVID cardiovascular sequelae—including myocarditis, arrhythmias, and heart failure—persist in survivors [1, 3-9].

2. Pathophysiological Mechanisms

- Endothelial dysfunction & microthrombosis: Microthrombi are frequently observed in cardiac microvasculature, distinct from typical coronary thrombi, contributing to ischemia and remodeling [3, 5, 10].
- Direct myocardial injury: Viral infiltration and cytokine-mediated damage trigger myocarditis, fibrosis, and dysautonomia [2, 7, 11].
- Chronic inflammation: Persistent immune activation may potentiate arrhythmia and autonomic dysfunction [1, 4, 7].

3. Clinical Sequelae

3.1. Myocarditis & Remodeling

Post-COVID myocarditis risk increases six-fold relative to controls [1]. CMR studies in athletes found ~1–3% with myocarditis; a small subset showed subclinical fibrosis up to 6 months post-infection [6-8, 12]

3.2. Arrhythmias

Arrhythmia incidence is ~1.6 times higher [1, 3]. Potential mechanisms include inflammation, fibrotic remodeling, and autonomic dysregulation [4, 7].

3.3. Heart Failure

Risk of new-onset heart failure increases by ~1.7–2 times [1, 3]. Studies using large health databases report sustained elevated risk for up to 12 months [1, 9].

4. Epidemiology & Risk Stratification

Cohort	Risk Increase Post-COVID	Notes
General survivors (any severity)	Myocarditis ~6×; arrhythmia ~1.6×; heart failure ~1.7–2× [1,3,9]	Even mild outpatient cases are at risk
Hospitalized patients	Highest incidence; microthrombi common [3,10]	Worse prognosis
Athletes (ORCCA/NCAA data)	CMR myocarditis 1–3%; fibrosis <5% [6,8,12]	Low clinical adverse events
Elite athletes	Rare sustained cardiac impairment [7,8]	Subclinical findings resolve

5. Clinical Sequelae

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6. Microthrombosis & Ischemic Injury

Autopsy and imaging studies confirm microthrombi as a major driver of myocardial necrosis [3, 10]. These vascular insults aggravate long-term cardiac dysfunction.

7. Diagnostic & Monitoring Recommendations

- CMR: Gold standard; essential for diagnosing myocarditis and fibrosis, particularly in athlete [6-8].
- Biomarkers/ECG/Echo: Troponin, NT-proBNP, D-dimer, ECG, echo to monitor those with symptoms or high risk [3, 9].
- Longitudinal Surveillance: Healthcare systems should incorporate CV follow-up into post-COVID care pathways [1].

8. Management Strategies

- Medical Therapy: ACE-I/ARB, beta-blockers, diuretics per myocarditis/heart failure guidelines. [1, 4]
- Anticoagulation: Consider in high-risk microthrombotic cases (persistent D-dimer elevation). [3, 11]
- Return-to-Play (RTP): For athletes with myocarditis, restrict activity 3–6 months; RTP only after negative CMR and normalized biomarkers.[12, 13]

9. Future Research Directions

- Longitudinal registries to track CV outcomes and arrhythmic events. [6, 12]
- Clinical trials targeting endothelial repair and antithrombotic therapy. [3, 10]
- Research into dysautonomia and microvascular dysfunction in persistent symptoms. [4, 7]

Population Group	Myocarditis Risk	Arrhythmia Risk	Heart Failure Risk	Notes
General Survivors	↑6× [1,3]	↑1.6× [1,3]	↑1.7–2× [1,3]	Includes mild cases
Hospitalized Patients	Highest absolute risk	Significant	Highest risk	Microthrombi common [3]

Athletes (ORCCA/NCAA)	1–3% (CMR-detected) [6,8]	Subclinical only	Rare; functional	No sustained impairment

Population Group	Myocarditis Risk	Arrhythmia Risk	Heart Failure Risk	Notes
Elite Athletes	Low; <5% fibrosis [7]	No adverse events	N/A	Functional resolution [7]

Table 1: Summary of Post-COVID Cardiovascular Risk by Population

Patient Profile	Initial Evaluation	Follow-Up / Action Plan
Symptomatic survivors	ECG, troponin, NT-proBNP, D-dimer, echo [3,9]	If abnormal → CMR → myocarditis protocol (restrict activity)
CMR-confirmed myocarditis	Repeat CMR + biomarkers at 3–6 months [12]	Resume activity when resolved
All COVID survivors	Symptoms monitoring; consider biomarker screening	Annual follow-up, esp. in athletes, hospitalized

Table 2: Diagnostic & Management Algorithm for Post-COVID Cardiovascular Sequelae

10. Conclusion

COVID-19 poses a lasting threat to cardiovascular health, manifested in myocarditis, arrhythmias, heart failure, and microvascular damage. Evidence supports structured surveillance, targeted imaging, and adherence to management protocols. Integration of cardiopulmonary care into long-COVID pathways is critical to mitigating morbidity in survivors, particularly among high-risk groups [14].

Author Disclosures

Author Contributions

I, Brendan Jones, conceptualized the review, conducted the literature search, synthesized the findings, and wrote the manuscript.

Conflict of Interest

The author declares no conflicts of interest. Funding: No external funding was received for this work.

Use of AI Tools

This manuscript was prepared with the assistance of AI language models to enhance clarity, grammar, and structure. All intellectual content, critical analysis, and interpretation of data remain the sole responsibility of the author.

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