This article can be cited as: Parizad R. The hidden cardiovascular crisis of long COVID. Cardiovasc Biomed J. 2025; 5(2): 3-4.

Letter to Editor

The hidden cardiovascular crisis of long COVID

Razieh Parizad [1,*]

[1] Cardiovascular Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

*** Corresponding Author:**
Address: University Street - Tabriz University of Medical Sciences - Shahid Madani Hospital, Tabriz, Iran. **Postal code:** 5166615573; **Tel:** +98 09143134453; **Email:** r_parizad2003@yahoo.com

Article Information:
Received: 12 Oct 2025; Revised: 13 Nov 2025; Accepted: 22 Dec 2025 DOI: 10.18502/cbj.v5i2.21131

Dear Editor

The coronavirus disease 2019 (COVID-19) pandemic, initially characterized by acute respiratory distress syndrome (ARDS) and thrombotic complications, has progressively evolved into a persistent global health challenge owing to its long-term sequelae, particularly cardiovascular complications. These chronic manifestations, commonly referred to as post-acute sequelae of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (PASC) or Long COVID, place a considerable burden on healthcare systems worldwide, including in individuals who experienced only mild or asymptomatic acute infection. This letter explores the diverse cardiovascular manifestations of Long COVID, elucidates their complex pathophysiological mechanisms, and underscores the urgent need for integrated clinical and research strategies to address this growing concern. Long COVID is associated with a broad spectrum of cardiovascular manifestations, including persistent palpitations, exertional dyspnea, chest pain (CP), and severe fatigue (2). Objective evidence supports these symptoms; for instance, cardiac magnetic resonance imaging (CMR) has demonstrated persistent myocardial inflammation and fibrosis several months after viral clearance (3). Electrocardiographic (ECG) studies have further identified new-onset arrhythmias, such as atrial fibrillation (AF) and ventricular tachycardia (VT), indicating an elevated risk for such events (4). Subclinical myocarditis, initially observed as an acute complication, may persist beyond the acute phase and contribute to ongoing symptoms (2). Moreover, postural orthostatic tachycardia syndrome (POTS), defined by an exaggerated heart rate (HR) response upon standing, has been reported, compounding morbidity with manifestations such as dizziness and cognitive impairment (4). These data underscore the cardiovascular system as a sustained target of both SARS-CoV-2 infection and the host inflammatory response (1). The cardiovascular effects of Long COVID arise from a complex interplay of various mechanisms. Direct viral invasion of cardiomyocytes via the angiotensin-converting enzyme 2 (ACE2) receptor may precipitate acute injury, especially in myocarditis (5). However, chronic symptoms likely arise from broader processes, including persistent low-grade systemic inflammation and immune dysregulation, which impair microvascular function and cause endothelial dysfunction, contributing to CP and fatigue (2). Furthermore, dysregulation of the autonomic nervous system (ANS), notably in POTS, leads to exercise intolerance and orthostatic symptoms, potentially due to immune-mediated neural injury or brainstem inflammation (4). Additionally, a prolonged prothrombotic state elevates the risk of microvascular thrombosis, exacerbating cardiovascular strain (5). To fully understand the mechanisms, this complex cascade of direct viral effects, chronic inflammation, autonomic dysfunction, and thrombogenicity necessitates further research. The cardiovascular burden of Long COVID represents a substantial public health challenge. With millions of individuals affected worldwide, even a modest proportion of patients developing chronic sequelae places considerable strain on healthcare systems (1). The heterogeneity of clinical presentations further complicates management, as

standardized diagnostic and therapeutic protocols remain lacking (2). Managing these complex manifestations requires a multidisciplinary approach that integrates expertise from cardiology, neurology, pulmonology, and rehabilitation medicine. For instance, POTS may be managed with beta-blockers and structured rehabilitation programs, whereas persistent myocardial inflammation could necessitate anti-inflammatory interventions (4). To guide clinical practice, large-scale prospective studies are urgently needed to establish standardized diagnostic criteria, identify reliable biomarkers, and develop targeted therapies, including anti-inflammatory agents and autonomic modulators, aimed at reducing the long-term cardiovascular consequences of SARS-CoV-2 infection (1). The cardiovascular sequelae of Long COVID persist well beyond the acute phase of COVID-19, posing an escalating public health challenge. Mitigating this crisis requires a coordinated effort from scientific and clinical communities to enhance understanding, develop standardized care pathways, and provide patient-centered care. Sustained dedication to research and clinical innovation is essential to alleviating the long-term burden of this silent pandemic.

References

1. Raman B, Bluemke DA, Lüscher TF, et al. Long COVID: post-acute sequelae of COVID-19 with a cardiovascular focus. *Eur Heart J*. 2022;43(11):1157-1172.
2. Xie Y, Xu E, Bowe B, et al. Long-term cardiovascular outcomes of COVID-19. *Nat Med*. 2022;28(3):583-590.
3. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). *JAMA cardiol*. 2020;5(11):1265-1273.
4. Jamal SM, Landers DB, Hollenberg SM, et al. Prospective evaluation of autonomic dysfunction in post-acute sequela of COVID-19. *J Am Coll Cardiol*. 2022;79(23):2325-2330.
5. Tsampasian V, Bäck M, Bernardi M, et al. Cardiovascular disease as part of Long COVID: A systematic review. *Eur J Prev Cardiol*. 2025;32(6):485-498.

