



RHYTHM AND CONDUCTION DISORDERS IN COVID-19: FROM MYOCARDIAL INJURY TO CLINICAL ARRHYTHMIAS

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ABSTRACT:

COVID-19 is associated with a broad spectrum of rhythm and conduction disorders, particularly among hospitalized and critically ill patients. Arrhythmogenesis is multifactorial and reflects systemic inflammation, hypoxaemia, myocardial ischaemia or injury, myocarditis, autonomic imbalance, thromboembolic disease, electrolyte disturbances and drug-related proarrhythmia. Atrial fibrillation is the most frequently reported sustained arrhythmia, while ventricular tachyarrhythmias, bradyarrhythmias and atrio-ventricular conduction disturbances occur predominantly in patients with severe disease or myocardial injury. Contemporary population-based evidence also indicates that arrhythmic risk may persist beyond the acute infection, although the absolute risk and the contribution of direct viral myocardial injury remain incompletely defined. This review summarizes the principal mechanisms, clinical phenotypes and management considerations of COVID-19-related arrhythmias and highlights emerging evidence concerning post-acute autonomic dysfunction and long-term rhythm complications.

Keywords: arrhythmias, atrial fibrillation, COVID-19, myocardial injury, conduction disorders,

INTRODUCTION:

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causative pathogen of coronavirus disease 2019 (COVID-19), emerged in late 2019 and rapidly caused a global pandemic. SARS-CoV-2 belongs to the Coronaviridae family. Among the human coronaviruses, SARS-CoV, MERS-CoV and SARS-CoV-2 can cause severe respiratory disease, whereas HKU1, NL63, OC43 and 229E are generally associated with milder respiratory syndromes. The term “coronavirus” derives from the crown-like appearance created by the surface spike proteins. SARS-CoV-2 infection ranges from asymptomatic disease to severe pneumonia, acute respiratory distress syndrome and multiorgan failure [1]. Acute respiratory infections can precipitate cardiovascular events, while pre-existing cardiovascular disease can worsen the clinical course of infection [2]. Severe COVID-19 is associated with intense inflammation, endothelial dysfunction, thrombosis and multiorgan injury, with the cardiovascular system among the commonly affected organ systems [3]. Viral entry is mediated mainly through angiotensin-converting enzyme 2 (ACE2), which is expressed in vascular endothelial cells, arterial smooth-muscle cells and cardiomyocytes [4].

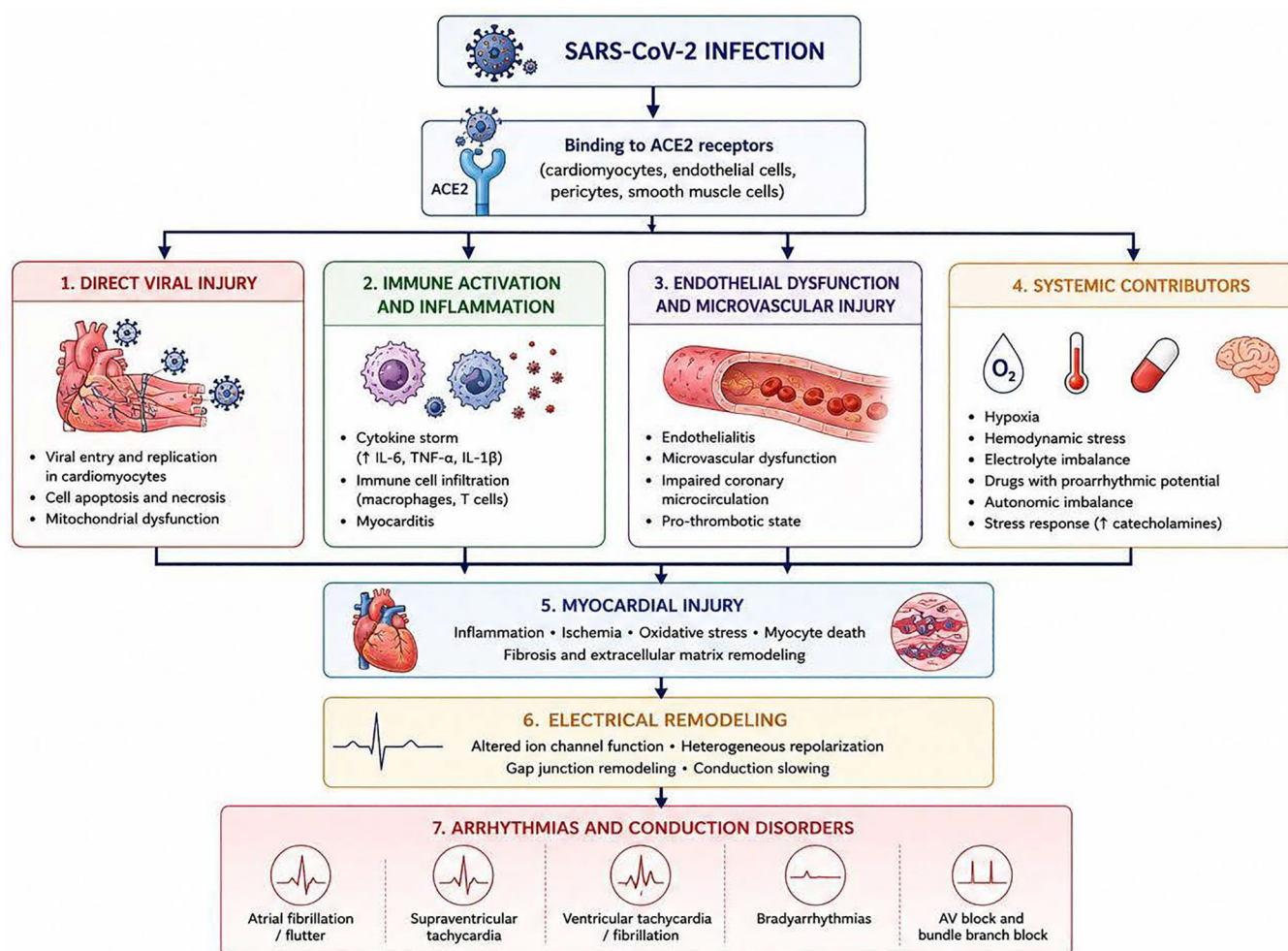
The interaction between SARS-CoV-2 and ACE2 is one component of COVID-19-related vascular and myocardial injury. Viral binding and internalization can reduce membrane ACE2 activity and shift the renin–angiotensin system toward angiotensin II-mediated vasoconstriction, inflammation, oxidative stress and adverse myocardial remodeling [5, 6]. These effects, together with systemic inflammation, hypoxaemia, thrombosis and autonomic activation, can promote the onset or exacerbation of arrhythmias. The reported spectrum includes atrial fibrillation (AF), atrial flutter, supraventricular tachycardia, monomorphic and polymorphic ventricular tachycardia, ventricular fibrillation and different forms of conduction disease.

REVIEW RESULTS:

Mechanisms of arrhythmogenesis. Early clinical studies demonstrated an increased frequency of cardiac arrhythmias in patients with COVID-19. The underlying pathophysiology is heterogeneous. Myocardial injury can result from inflammatory myocyte damage, oxygen supply–demand imbalance, acute coronary syndromes, microvascular thrombosis, stress cardiomyopathy or myocarditis. Increased right-ventricular pressure due to severe lung disease, pulmonary hypertension or pulmonary embolism can further destabilize the myocardium. Im-

mune-mediated injury can involve cytotoxic T-lymphocyte infiltration and an amplified cytokine response, producing a self-perpetuating cycle of inflammation and myocardial damage (Fig. 1). A reported case of COVID-19-associated acute myocarditis was confirmed by cardiac magnetic resonance (CMR) and endomyocardial biopsy, with diffuse T-lymphocyte infiltration but no detectable SARS-CoV-2 genome in myocardial tissue. The imaging phenotype resembled reverse Takotsubo syndrome, with predominant basal and mid-ventricular dysfunction [7].

Fig. 1. Mechanisms of COVID-19-related myocardial injury leading to electrical remodeling, arrhythmias and conduction disorders.



Additional arrhythmogenic factors include hypoxaemia, fever, endogenous catecholaminergic activation, electrolyte disturbances and drug-related proarrhythmia. Several agents used during the early pandemic, particularly hydroxychloroquine, chloroquine and azithromycin, prolong ventricular repolarization and can increase the risk of marked QTc prolongation and torsades de pointes, especially when combined or admin-

istered to patients with renal dysfunction, bradycardia or electrolyte abnormalities [8]. Patients with underlying cardiovascular disease more often had elevated cardiac troponin T and adverse outcomes. Ventricular tachycardia or ventricular fibrillation occurred more frequently in patients with elevated troponin than in those without myocardial injury (11.5% vs. 5.2%) [9]. These observations support myocardial injury as a marker of

disease severity and arrhythmic vulnerability rather than proof of direct viral myocarditis [10].

Myocardial injury also occurred in patients without a previous history of cardiovascular disease, although less frequently than in those with established cardiovascular disease [29]. In the reported cohort, cardiac troponin T was elevated in 13.2% of patients without known cardiovascular disease and in 54.5% of those with cardiovascular disease [9]. Interpretation requires caution because elevated troponin in severe COVID-19 can reflect type 2 myocardial infarction, acute coronary syndromes, right-ventricular strain, systemic inflammation, renal dysfunction or critical illness, rather than myocarditis alone.

In a worldwide survey of 4,526 hospitalized patients with COVID-19, 827 developed a clinically significant arrhythmia. Atrial fibrillation was the predominant rhythm disturbance; ventricular arrhythmias and bradyarrhythmias were also reported. Arrhythmias were associated with greater illness severity, a higher need for mechanical ventilation and lower survival to hospital discharge [11]. An early Wuhan case series of 138 hospitalized patients reported arrhythmias in 16.7% overall and in 44.4% of patients requiring intensive care, compared with 6.9% of patients treated outside the intensive care unit [12]. These early reports did not always provide rhythm-specific adjudication, and their estimates should not be extrapolated directly to vaccinated populations, later viral variants or predominantly outpatient disease.

In a cohort of 700 patients hospitalized with COVID-19, nine experienced cardiac arrest, including six pulseless electrical activity events, one torsades de pointes event and two asystolic arrests. Twenty-five patients developed AF, ten had non-sustained ventricular tachycardia, and nine had clinically significant bradyarrhythmias. Cardiac arrest was usually associated with severe systemic illness rather than a primary ventricular tachyarrhythmia [13]. These findings reinforce the multifactorial nature of arrhythmogenesis in COVID-19 and the importance of disease severity, metabolic disturbance and respiratory failure.

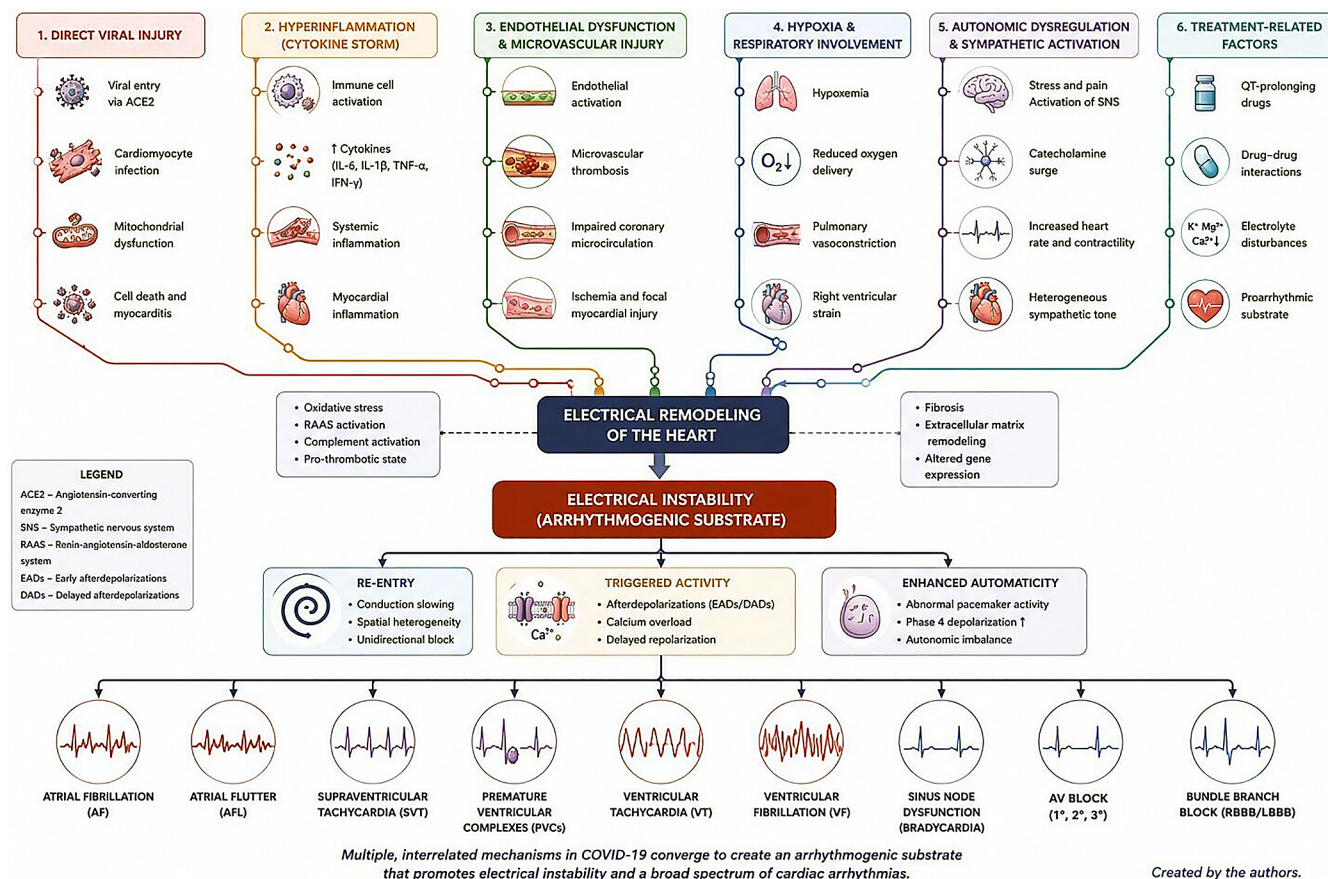
A global survey conducted through the Heart Rhythm Society documented a broad spectrum of atrial, ventricular and bradyarrhythmic events in hospitalized

patients with COVID-19. AF was the most commonly reported sustained arrhythmia, followed by atrial flutter, atrial tachycardia and paroxysmal supraventricular tachycardia. Reported ventricular events included monomorphic and polymorphic ventricular tachycardia, ventricular fibrillation and cardiac arrest. Conduction abnormalities included sinus bradycardia, atrioventricular block, bundle-branch block and intraventricular conduction delay [14]. Because the survey was based on physician-reported cases rather than systematic monitoring of a defined cohort, its percentages should be interpreted descriptively.

Remdesivir inhibited SARS-CoV-2 in vitro and was assessed in a randomized, double-blind, placebo-controlled trial in adults with severe COVID-19; the trial did not show a statistically significant difference in time to clinical improvement in its primary analysis [15]. Chloroquine, hydroxychloroquine and azithromycin are not recommended as routine treatment for hospitalized COVID-19 because clinical benefit was not established, and their combined use can substantially prolong the QTc interval [28]. When a QT-prolonging regimen is unavoidable, baseline and follow-up ECG assessment, correction of potassium and magnesium abnormalities, review of interacting medications and renal-dose adjustment are required [8,19].

In a study of 113 consecutive patients with PCR-confirmed SARS-CoV-2 infection and radiographically documented pulmonary involvement who required intensive care, 50 sustained atrial tachyarrhythmias, five sustained ventricular arrhythmias and 30 bradycardic events were recorded during 2,321 telemetry-days. New-onset atrial arrhythmias were associated with haemodynamic deterioration in 13 cases (35.1%) and occurred in older patients with higher troponin T and NT-proBNP concentrations and more severe disease. The five ventricular arrhythmias comprised two ventricular tachycardias, two ventricular fibrillation episodes and one torsades de pointes; all were terminated by immediate cardioversion or defibrillation. Precipitating factors were identified in 19 of 30 bradycardic events, and no patient required permanent pacing [16]. Arrhythmias in critical COVID-19 therefore often reflect the interaction between acute systemic illness and a susceptible myocardial substrate (Fig. 2).

Fig. 2. Parallel mechanisms leading to electrical instability and arrhythmias in COVID-19.



Role of myocardial injury. Myocardial injury, conventionally defined by cardiac troponin concentrations above the 99th percentile upper reference limit, has been reported in a substantial proportion of hospitalized patients with COVID-19 and is consistently associated with greater disease severity and mortality [17]. Two broad mechanisms are considered: virus-related cellular injury and immune-mediated myocardial damage. SARS-CoV-2 uses its spike protein to bind ACE2 and facilitate cellular entry; downregulation of ACE2 can favour angiotensin II signalling through the type 1 angiotensin receptor and promote inflammation, oxidative stress and adverse remodeling [5,6,18]. In parallel, activated immune cells can infiltrate the myocardium and amplify tissue injury through cytokine-mediated pathways. These processes can alter intercellular coupling, calcium handling and ion-channel expression, thereby promoting triggered activity, abnormal automaticity and re-entry. If inflammation resolves with residual scar or fibrosis, an arrhythmogenic substrate can persist beyond the acute phase [18].

Supraventricular tachycardia (SVT). Management should begin with correction of precipitating factors, including hypoxaemia, fever, acid-base and electrolyte disturbances, myocardial ischaemia and interacting medications. In a regular narrow-complex tachycardia, vagal manoeuvres and intravenous adenosine remain appropriate when no contraindication is present. Synchron-

ized electrical cardioversion is indicated for haemodynamic instability. Beta-blockers or non-dihydropyridine calcium-channel blockers can be selected for ongoing rate control according to ventricular function, blood pressure, bronchospasm risk and drug interactions. Excessive bradycardia should be avoided, particularly in patients receiving QT-prolonging drugs [14,19].

Atrial fibrillation and atrial flutter. The therapeutic objectives are ventricular-rate control, prevention of thromboembolism, symptom relief and rhythm control when clinically indicated. In haemodynamically stable patients, rate control is often appropriate during acute systemic illness, while urgent synchronized cardioversion is indicated when AF or atrial flutter causes haemodynamic instability. Amiodarone can be considered in selected critically ill patients or in the presence of heart failure, but QT prolongation, bradycardia and drug interactions require close monitoring [19,20]. Class Ic agents should be restricted to patients without significant structural or ischaemic heart disease and should not be selected solely because of COVID-19 [28]. Stroke prevention should follow contemporary AF guidance. Current ESC guidance favours the CHA₂DS₂-VA score for thromboembolic risk assessment; oral anticoagulation is based on the patient's underlying stroke-risk profile and bleeding risk, not on SARS-CoV-2 infection alone [21]. The long-term rhythm-control strategy should be reassessed after recovery from the acute illness.

Ventricular tachyarrhythmias and COVID-19.

Management requires rapid identification and correction of reversible triggers, including hypoxaemia, ischaemia, metabolic disturbance, electrolyte abnormalities and proarrhythmic medication. Sustained ventricular tachycardia, ventricular fibrillation and cardiac arrest should be treated according to established advanced life-support and ventricular-arrhythmia guidelines [22]. Fever deserves particular attention in patients with Brugada syndrome because it can unmask or accentuate a type 1 Brugada ECG pattern and increase arrhythmic risk. Prompt antipyretic treatment, ECG reassessment and monitored hospital care are appropriate in patients with a spontaneous or fever-induced type 1 pattern accompanied by syncope, documented ventricular arrhythmia or other high-risk features [22]. A pathogenic SCN5A variant alone does not define clinical risk and should be interpreted together with the phenotype and family history.

Long-term arrhythmic risk after COVID-19. Population-based evidence published after the early pandemic phase indicates that arrhythmic risk is not restricted to acute hospitalization. In a nationwide Swedish study including more than one million individuals with laboratory-confirmed infection and more than four million matched controls, the risk of first atrial tachyarrhythmia, paroxysmal supraventricular tachycardia and bradyarrhythmia was increased after COVID-19, with the magnitude and duration of risk strongly related to age and disease severity. Increased risk persisted for up to 60 days for atrial tachyarrhythmias and up to 180 days for paroxysmal supraventricular tachycardia; excess ventricular-arrhythmia risk was not demonstrated beyond the day of diagnosis [23]. A large UK primary-care study also identified a temporal clustering of post-COVID cardiovascular events and provided individualized risk estimates based on age and comorbidity [24]. These data support symptom-directed surveillance in patients with palpitations, syncope, unexplained dyspnoea or exercise intolerance rather than indiscriminate rhythm screening of all survivors.

Post-COVID atrial fibrillation. A 2024 systematic review and meta-analysis found a higher subsequent incidence of AF among individuals who had recovered from COVID-19 than among non-infected comparison groups. The analysis included approximately 20 million participants, but heterogeneity in population characteristics, follow-up duration and methods of AF ascertainment remained substantial [25]. Newly detected AF after COVID-19 should therefore be evaluated according to standard principles, including assessment of reversible triggers, structural heart disease, symptom burden and thromboembolic risk. Anticoagulation and rhythm-control decisions should follow established AF guidelines rather than being driven by prior infection alone [21].

Autonomic dysfunction, inappropriate sinus tachycardia and POTS. Persistent palpitations and tachycardia after the acute infection do not always represent a primary cardiac arrhythmia. Post-COVID cardiovascular autonomic dysfunction can present as inappropriate sinus

tachycardia, orthostatic intolerance, reflex syncope or postural orthostatic tachycardia syndrome (POTS). Proposed mechanisms include autonomic imbalance, hypovolaemia, deconditioning, endothelial and microvascular dysfunction, immune dysregulation and autoantibody-mediated interference with adrenergic or muscarinic signalling [26]. Diagnosis requires correlation of symptoms with heart-rate and blood-pressure responses to active standing or tilt testing and exclusion of anaemia, thyroid disease, active inflammation, pulmonary disease and structural heart disease. Management should be phenotype directed, beginning with adequate fluid and salt intake when appropriate, physical countermeasures, compression garments and individually adapted recumbent exercise; pharmacological treatment is reserved for selected patients [19,26].

Myocardial inflammation, CMR abnormalities and arrhythmogenic substrate. Persistent myocardial oedema, scar or diffuse interstitial abnormalities on CMR can provide an anatomical substrate for ventricular ectopy or re-entry, but imaging findings must be interpreted in the clinical context. Late gadolinium enhancement alone does not establish active viral myocarditis, and the relationship between post-COVID CMR abnormalities and future malignant ventricular arrhythmias remains incompletely defined. Selective CMR is appropriate in patients with persistent chest pain, elevated cardiac biomarkers, ventricular arrhythmias, new ventricular dysfunction or other findings suggesting myocardial involvement; routine imaging of asymptomatic individuals is not supported. Longitudinal studies combining CMR, ambulatory rhythm monitoring, biomarkers and clinical outcomes are required to determine which imaging phenotypes have prognostic significance [18,19,27].

Contemporary clinical perspective. Current evidence supports a distinction between arrhythmias occurring during severe acute systemic illness and rhythm symptoms developing during recovery. During acute COVID-19, treatment should focus on correction of hypoxaemia, inflammation, ischaemia, electrolyte imbalance and drug-related QT prolongation, together with guideline-directed treatment of the documented arrhythmia. During the post-acute phase, evaluation should be driven by symptoms and objective findings and can include a 12-lead ECG, ambulatory rhythm monitoring for intermittent episodes, echocardiography when structural disease is suspected, and autonomic testing when the clinical pattern is orthostatic. The available evidence does not support attributing every post-COVID palpitation to myocarditis or a malignant rhythm disorder [19].

CONCLUSION:

Available evidence supports a multifactorial model of rhythm and conduction disturbances in COVID-19. The relative contributions of myocardial injury, immune-mediated inflammation, hypoxaemia, ischaemia, haemodynamic stress, autonomic imbalance, electrolyte abnormalities and drug-related effects differ substantially among patients and across disease phases. This heterogeneity

limits direct comparison between cohorts and complicates pooled estimates, particularly when studies combine patients with mild outpatient disease and those with critical multiorgan failure.

Future studies should clarify how disease severity, hypoxaemia, pre-existing cardiovascular disease and myocardial injury influence the type, burden and prognosis of arrhythmias. Standardized definitions, systematic rhythm monitoring and clear separation of acute infection from post-acute sequelae are required. Clinically, arrhythmias arising during acute COVID-19 should be interpreted within the context of the underlying systemic illness, while persistent palpitations or tachycardia after recovery warrant evaluation for recurrent arrhythmia, inappropriate sinus tachycardia, POTS and structural myocardial disease. Future investigations should account for vaccination status and viral variants and should combine

extended rhythm monitoring with biomarkers, echocardiography and CMR. Prospective studies are needed to determine which imaging or biomarker abnormalities predict clinically relevant arrhythmias, whether risk diminishes over time and which patients benefit from prolonged surveillance. Such evidence is required to move from descriptive associations toward individualized risk stratification and targeted prevention.

Abbreviations:

ACE2 – angiotensin-converting enzyme 2; AF – atrial fibrillation; CMR – cardiac magnetic resonance; ECG – electrocardiogram; NT-proBNP – N-terminal pro-B-type natriuretic peptide; POTS – postural orthostatic tachycardia syndrome; SARS-CoV-2 – severe acute respiratory syndrome coronavirus 2; SVT – supraventricular tachycardia.

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