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POST-COVID-19 SURGE IN SUDDEN CARDIAC ARREST: A COMPREHENSIVE REVIEW OF METABOLIC SYNDROME, LIFESTYLE RISK FACTORS, AND EMERGING PATHOPHYSIOLOGICAL INSIGHTS

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ABSTRACT

Background: Sudden cardiac arrest (SCA) has notably increased in incidence following the COVID-19 pandemic. The intersection of SARS-CoV-2 infection with metabolic syndrome (MetS), lifestyle changes during the pandemic, and complex pathophysiological mechanisms contributes to this surge. **Objective:** This review aims to synthesize current evidence on the role of metabolic syndrome and lifestyle risk factors in post-COVID-19 SCA, explore emerging pathophysiological insights, and discuss prevention and management strategies. **Methods:** A comprehensive literature review was conducted, focusing on epidemiological data, mechanistic studies, and clinical reports related to COVID-19, metabolic syndrome, and SCA. **Results:** SARS-CoV-2 induces cardiovascular injury through inflammation, endothelial dysfunction, and autonomic imbalance. Metabolic syndrome components—hypertension, dyslipidemia, insulin resistance, and central obesity—exacerbate these effects. Pandemic-associated sedentary behavior, poor diet, and mental health stressors further elevate cardiovascular risk. Emerging research employing omics and AI techniques offers promise for improved risk stratification and targeted therapies. **Conclusion:** The post-COVID-19 rise in SCA necessitates integrated multidisciplinary approaches encompassing metabolic control, lifestyle modification, vigilant cardiovascular screening, and public health interventions. Continued research is essential to mitigate long-term cardiovascular sequelae.

KEYWORDS: Sudden cardiac arrest, COVID-19, Metabolic syndrome, Lifestyle risk factors, Cardiovascular complications, Inflammation, Post-acute sequelae, Prevention strategies, Artificial intelligence.

1. INTRODUCTION

Sudden cardiac arrest (SCA) is a critical public health concern characterized by an abrupt loss of heart function, often resulting from a cardiac arrhythmia, such as ventricular fibrillation or pulseless ventricular tachycardia. It is a leading cause of mortality worldwide,

with survival rates remaining dismally low despite advances in emergency response and cardiopulmonary resuscitation (CPR) techniques. Globally, SCA accounts for approximately 15–20% of all deaths and represents one of the most challenging emergencies in both community and hospital settings (Myerburg & Junttila, 2012).

The coronavirus disease 2019 (COVID-19) pandemic has had far-reaching effects on global health systems, economies, and disease patterns. Initially perceived as a primarily respiratory illness, COVID-19 has since been recognized for its widespread multisystem impact, particularly on the cardiovascular system. SARS-CoV-2, the virus responsible for COVID-19, exerts pathogenic effects through endothelial dysfunction, inflammation, and coagulopathy, all of which can contribute to adverse cardiac events including myocarditis, arrhythmias, and ultimately, SCA (Guo et al., 2020; Nishiga et al., 2020).

In the aftermath of acute COVID-19 infection, a growing body of epidemiological evidence has revealed a disturbing trend: an apparent surge in the incidence of sudden cardiac arrest. Observational studies and cardiac registry data suggest increased rates of out-of-hospital cardiac arrest (OHCA) during and after the pandemic period, particularly in populations with preexisting metabolic or cardiovascular risk factors (Marijon et al., 2020; Ball et al., 2021). Moreover, survivors of COVID-19 appear to exhibit a persistently elevated risk of cardiovascular complications months after recovery, implicating long-term pathophysiological changes in post-viral cardiovascular homeostasis (Xie et al., 2022).

Given the multifactorial underpinnings of SCA and the complex interplay between COVID-19, metabolic syndrome, and lifestyle changes exacerbated by the pandemic, a comprehensive understanding of these interconnections is urgently needed. This review aims to synthesize current knowledge on the post-COVID-19 surge in sudden cardiac arrest, with a focus on the roles of metabolic syndrome, behavioral and lifestyle risk factors, and emerging mechanistic insights. By integrating clinical data, epidemiological trends, and recent advances in cardiovascular science, this paper seeks to provide a foundation for improved risk stratification, prevention strategies, and future research directions.

2. COVID-19 and the Cardiovascular System

2.1 SARS-CoV-2 Mechanisms of Cardiovascular Injury

SARS-CoV-2, the virus responsible for COVID-19, affects the cardiovascular system through multiple interrelated mechanisms. These include direct viral invasion of myocardial tissue, systemic inflammation, hypoxia-induced myocardial stress, and activation of prothrombotic pathways. The virus enters host cells by binding to the angiotensin-converting enzyme 2 (ACE2) receptor, which is highly expressed not only in the lungs but also in vascular endothelial cells, cardiac myocytes, and pericytes (Nishiga et al., 2020). This facilitates viral tropism to cardiac tissue and contributes to direct cytopathic effects, leading to myocardial injury. Additionally, systemic inflammatory responses, characterized by cytokine storms, further aggravate cardiac dysfunction and can precipitate arrhythmias or heart failure (Guo et al., 2020).

2.2 Myocarditis, Arrhythmias, and Endothelial Dysfunction

Clinical and autopsy studies have reported cases of myocarditis in COVID-19 patients, often presenting with elevated cardiac biomarkers such as troponin and brain natriuretic peptide (BNP), alongside imaging findings suggestive of myocardial inflammation. Inflammation and viral infiltration of cardiac tissue disrupt the electrophysiological balance, increasing susceptibility to both atrial and ventricular arrhythmias (Siripanthong et al., 2020). Additionally, COVID-19 is associated with widespread endothelial dysfunction. SARS-CoV-2-induced endotheliitis, characterized by inflammation of the endothelium, promotes vasoconstriction, procoagulant states, and thrombogenesis, which are critical contributors to cardiovascular complications and sudden cardiac events (Varga et al., 2020).

2.3 Post-Acute Sequelae of SARS-CoV-2 Infection (PASC) or Long COVID

A subset of individuals recovering from COVID-19 continue to experience a range of symptoms and organ dysfunctions beyond the acute phase, collectively termed post-acute sequelae of SARS-CoV-2 infection (PASC) or Long COVID. Cardiovascular manifestations of PASC include persistent chest pain, palpitations, exercise intolerance, and elevated risk of myocarditis, arrhythmias, and thromboembolic events even months after recovery (Puntmann et al., 2020; Xie et al., 2022). These chronic sequelae underscore the prolonged burden of cardiovascular risk among COVID-19 survivors, with significant implications for the development of sudden cardiac arrest.

2.4 Role of ACE2 Receptors and Inflammatory Pathways

ACE2 plays a pivotal role in the renin-angiotensin-aldosterone system (RAAS) by degrading angiotensin II, a vasoconstrictor and pro-inflammatory peptide, into angiotensin-(1–7), which has vasodilatory and anti-inflammatory effects. SARS-CoV-2 binding to ACE2 not only facilitates viral entry but also leads to downregulation of ACE2 expression, tipping the balance toward unopposed angiotensin II activity (South et al., 2020). This imbalance exacerbates vasoconstriction, oxidative stress, inflammation, and fibrosis—key processes that can promote cardiovascular instability and arrhythmogenesis.

3. Metabolic Syndrome as a Precursor to Sudden Cardiac Arrest

3.1 Definition and Components of Metabolic Syndrome (MetS)

Metabolic syndrome (MetS) is a cluster of interrelated cardiometabolic abnormalities that significantly elevate the risk of atherosclerotic cardiovascular disease (ASCVD), type 2 diabetes mellitus (T2DM), and sudden cardiac arrest (SCA). The most commonly accepted definition is provided by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), which defines MetS as the presence of at least three of the five following components: hypertension, dyslipidemia, insulin resistance or type 2 diabetes, central obesity, and elevated fasting glucose (Grundy et al., 2005).

Table 1: Components of Metabolic Syndrome and Their Contribution to SCA Risk.

Component	Clinical Criteria (NCEP ATP III)	Contribution to SCA
Hypertension	Blood pressure ≥130/85 mmHg or on antihypertensive medication	Increases myocardial workload, LVH, and electrical instability (Carnethon et al., 2003)
Dyslipidemia	Triglycerides ≥150 mg/dL; HDL <40 mg/dL (men), <50 mg/dL (women)	Atherogenic profile promotes coronary plaque rupture and arrhythmia (Wilson et al., 2005)
Insulin Resistance / T2DM	Fasting glucose ≥100 mg/dL or on antidiabetic medication	Promotes inflammation, atherosclerosis, and autonomic dysfunction (Haffner et al., 1998)
Central Obesity	Waist circumference ≥102 cm (men), ≥88 cm (women)	Increases systemic inflammation and cardiac electrical remodeling (Lavie et al., 2009)
Elevated Fasting Glucose	Fasting glucose ≥100 mg/dL	Marker of insulin resistance and metabolic imbalance (Grundy et al., 2005)

3.2 Interaction Between COVID-19 and MetS

COVID-19 has exposed and amplified vulnerabilities in individuals with preexisting metabolic conditions. SARS-CoV-2 infection often leads to more severe outcomes in patients with MetS, due to impaired immune responses, chronic inflammation, and endothelial dysfunction (Bornstein et al., 2020). These factors also enhance the propensity for cardiovascular complications, including SCA. Moreover, individuals with MetS are more likely to experience exaggerated inflammatory responses and a higher viral load due to impaired cellular immunity (Hussain et al., 2020).

3.3 Synergistic Effects on Cardiovascular Risk and SCA

The overlap between COVID-19 and MetS produces a synergistic cardiovascular risk profile, elevating the chance of sudden cardiac arrest beyond the risk posed by either condition alone. MetS primes the cardiovascular system for instability through chronic endothelial damage and autonomic imbalance, while COVID-19 acts as a physiological stressor that can trigger acute events such as myocarditis, arrhythmias, and thromboembolism. Together, they form a “perfect storm” scenario for SCA (Petersen et al., 2020). Additionally, lifestyle disruptions during the pandemic (e.g., reduced physical activity, poor diet, stress) have worsened metabolic health, further amplifying risk.

4. Lifestyle Risk Factors and Behavioral Changes During the Pandemic

The COVID-19 pandemic led to profound shifts in daily behavior, many of which contributed to a heightened risk of metabolic deterioration and cardiovascular complications, including sudden cardiac arrest (SCA). Public health measures, including lockdowns and social distancing, disrupted physical activity, dietary patterns, and mental health—factors known to modulate cardiovascular risk.

4.1 Sedentary Behavior and Physical Inactivity

Stay-at-home orders and remote work dramatically reduced opportunities for physical activity. Studies have reported a significant decline in step counts and increased screen time, leading to prolonged sedentary behavior (Tison et al., 2020). Physical inactivity has long been associated with metabolic syndrome, obesity, insulin resistance, and cardiovascular dysregulation, all of which are precursors to SCA (Lavie et al., 2019).

4.2 Poor Dietary Habits and Weight Gain ("Quarantine Weight")

Dietary behaviors were adversely affected by pandemic-related disruptions, with many individuals reporting increased consumption of ultra-processed foods, sugar-sweetened beverages, and snacks (Di Renzo et al., 2020). Limited access to fresh produce and emotional eating due to stress also contributed to poor nutritional quality. This led to a phenomenon dubbed "quarantine weight gain," which exacerbated central obesity, dyslipidemia, and glucose intolerance—key components of metabolic syndrome and SCA risk.

4.3 Increased Alcohol and Substance Use

Alcohol consumption and substance use, including smoking and recreational drugs, rose during the pandemic, particularly among those facing job loss, isolation, and mental health challenges. Alcohol is

known to impair cardiac electrical activity and may trigger arrhythmias such as atrial fibrillation and ventricular tachycardia, both risk factors for SCA (Pollard et al., 2020). Increases in stimulant use, such as cocaine, are particularly concerning due to their direct pro-arrhythmic effects.

4.4 Mental Health Issues: Stress, Anxiety, and Depression

The psychological toll of the pandemic has been immense, with widespread reports of increased stress, anxiety, depression, and sleep disturbances (Vindegaard & Benros, 2020). These mental health conditions can independently elevate cardiovascular risk by inducing sympathetic overactivity, increasing cortisol levels, and promoting inflammation—all of which are linked to arrhythmogenesis and myocardial vulnerability (Lichtman et al., 2014).

4.5 Healthcare Access Disruption and Delayed Diagnosis

Many individuals delayed or avoided seeking medical care during the pandemic due to fear of infection or overwhelmed healthcare systems. This resulted in missed diagnoses of hypertension, diabetes, and cardiac arrhythmias, which might have otherwise been managed proactively. Elective procedures and preventive visits were postponed, potentially allowing cardiovascular risk factors to progress unchecked (Czeisler et al., 2020). Such delays are especially dangerous for at-risk populations already predisposed to sudden cardiac events.

5. Pathophysiological Mechanisms Underlying Post-COVID Sudden Cardiac Arrest

Emerging evidence indicates that SARS-CoV-2 infection leads to long-term cardiovascular complications that may increase the risk of sudden cardiac arrest (SCA), even after recovery from acute illness. Several intertwined pathophysiological mechanisms contribute to this elevated risk, including inflammation, autonomic dysregulation, and structural myocardial changes.

Table 2: Overview of Mechanisms.

Mechanism	Description	Contribution to SCA
Inflammation & Cytokine Storm	Exaggerated immune response characterized by IL-6, TNF-α, and IL-1β elevation	Triggers myocardial injury, electrical instability, and arrhythmias (Guo et al., 2020)
Hypercoagulability	Elevated D-dimer, fibrinogen; endothelial dysfunction	Leads to microthrombi, coronary thrombosis, and ischemic arrhythmias (Connors & Levy, 2020)
Autonomic Dysfunction	Postural orthostatic tachycardia, vagal imbalance	Disrupts heart rate variability, increases arrhythmic risk (Shouman et al., 2021)
Myocardial Fibrosis & Scarring	Post-viral myocardial inflammation leads to interstitial fibrosis and remodeling	Fibrotic tissue promotes re-entrant arrhythmias (Puntmann et al., 2020)
Mitochondrial Dysfunction	Viral effects on cellular energetics and oxidative stress pathways	Affects ATP-dependent ion channels critical for cardiac conduction (Gordon et al., 2020)
Genetic & Epigenetic Alterations	Long-term gene expression changes and viral impacts on host genome	May predispose to arrhythmogenic syndromes (Zhao et al., 2021)

5.1 Inflammation and Cytokine Storm

A hallmark of severe COVID-19 is the cytokine storm, involving a hyperinflammatory response with elevated levels of IL-6, TNF-α, and CRP. These cytokines cause direct myocardial damage, promote electrical instability, and contribute to ventricular arrhythmias, increasing SCA risk (Guo et al., 2020). Even in mild cases, persistent inflammation has been detected months after recovery, suggesting ongoing myocardial stress.

5.2 Hypercoagulability and Thrombotic Complications

COVID-19 is characterized by a prothrombotic state, driven by endothelial dysfunction and systemic inflammation. Increased D-dimer levels, platelet activation, and microvascular thrombosis have been implicated in myocardial infarction, pulmonary embolism, and stroke, all of which may precipitate SCA (Connors & Levy, 2020).

5.3 Autonomic Dysfunction and Cardiac Arrhythmias

Dysregulation of the autonomic nervous system is observed in Long COVID patients, often manifesting as postural orthostatic tachycardia syndrome (POTS) or baroreceptor dysfunction. This imbalance enhances sympathetic tone, which shortens ventricular repolarization and increases susceptibility to ventricular tachyarrhythmias (Shouman et al., 2021).

5.4 Myocardial Fibrosis and Scarring

Cardiac MRI studies have shown that even asymptomatic or mildly symptomatic COVID-19 patients can develop myocardial fibrosis, which may persist long after viral clearance (Puntmann et al., 2020). Fibrotic scars serve as

substrates for reentrant circuits, a primary mechanism of ventricular arrhythmias leading to SCA.

5.5 Mitochondrial Dysfunction and Metabolic Derangements

SARS-CoV-2 impairs mitochondrial function in host cells, disrupting ATP production and promoting oxidative stress. These changes impair cardiac energetics, disrupt ion channel function, and contribute to electrophysiological instability (Gordon et al., 2020), predisposing the heart to fatal arrhythmias.

5.6 Genetic and Epigenetic Alterations

Emerging studies suggest that COVID-19 may induce epigenetic changes in host cells, including DNA methylation and histone modification, altering expression of genes linked to cardiac electrophysiology and inflammation (Zhao et al., 2021). These long-term changes may explain persistent cardiovascular symptoms and increased SCA risk in post-acute patients.

6. Clinical Evidence and Epidemiological Data

Growing clinical and epidemiological evidence suggests a significant increase in sudden cardiac arrest (SCA) incidence following SARS-CoV-2 infection. This section synthesizes the current literature—including observational studies, case series, and autopsy reports—to highlight key patterns and demographic disparities, and to examine the influence of COVID-19 variants on cardiovascular outcomes.

6.1 Studies Reporting Increased SCA Incidents Post-COVID

Several retrospective and prospective studies have documented a notable rise in out-of-hospital cardiac arrests (OHCAs) during and after COVID-19 surges. A French nationwide registry found a doubling of OHCA incidence during the pandemic peak compared to the same period in previous years (Marijon et al., 2020). Similarly, U.S. cities reported a 20–60% increase in OHCA rates, with higher mortality and lower rates of return of spontaneous circulation (Baldi et al., 2020; Lai et al., 2021).

Table 3: Sudden Cardiac Arrest Trends Before and After COVID-19 Onset.

Study	Location	Time Period	SCA Incidence (per 100,000)	Change
Marijon et al. (2020)	France	March–April 2020	26.6 vs. 13.4 (2019)	+99%
Baldi et al. (2020)	Italy	March 2020	21.5 vs. 13.1 (2019)	+64%
Lai et al. (2021)	U.S.	Jan–Dec 2020 vs. 2019	89.0 vs. 68.5	+30%

6.2 Case Reports, Cohort Studies, and Autopsy Findings

Case reports have identified SCA in younger individuals with no prior cardiovascular history following mild or moderate COVID-19. Autopsy studies frequently reveal myocarditis, microvascular thrombosis, and fibrosis in the hearts of COVID-19 victims, consistent with arrhythmogenic substrates (Basso et al., 2020). Large cohort studies also confirm increased cardiovascular morbidity, with one U.S. Veterans Affairs study showing a 63% increased risk of arrhythmias post-COVID, even in non-hospitalized patients (Xie et al., 2022).

6.3 Demographic Disparities (Age, Sex, Race/Ethnicity)

The **disproportionate impact** of COVID-19 and post-COVID cardiovascular outcomes has been well-documented. Older adults, males, and racial/ethnic minorities (particularly Black and Hispanic populations) experienced higher rates of both COVID-19 infection and cardiac arrest (Chan et al., 2021). Socioeconomic factors, healthcare access, and pre-existing comorbidities contribute to these disparities.

6.4 COVID-19 Variants and Their Cardiovascular Profiles

Emerging data suggest that different **SARS-CoV-2 variants** may differ in their cardiovascular pathogenicity. For instance.

- **Delta variant** was linked with more severe acute disease and higher myocardial injury rates (Wang et al., 2022).
- **Omicron variant**, while associated with milder respiratory symptoms, has still been linked to long-term cardiovascular sequelae (Bhatt et al., 2022).
- The **Alpha variant** has been implicated in elevated hypercoagulability and endothelial dysfunction.

These differences may influence arrhythmia burden and post-COVID SCA risk profiles.

7. Prevention, Management, and Public Health Strategies

Addressing the surge in sudden cardiac arrest (SCA) post-COVID-19 requires comprehensive strategies spanning individual patient care, public health initiatives, and systemic interventions. This section highlights key prevention and management approaches focusing on metabolic syndrome, lifestyle modification, cardiovascular screening, and community-based measures.

7.1 Addressing Metabolic Syndrome and Lifestyle Risk Factors

Metabolic syndrome (MetS) components—hypertension, insulin resistance, dyslipidemia, and central obesity—are critical modifiable risk factors for SCA. Lifestyle interventions such as increased physical activity, dietary modifications, and weight control are essential. The COVID-19 pandemic's impact on sedentary behaviors and unhealthy diets underscores the urgency of integrating these strategies into post-pandemic recovery plans (Sattar et al., 2020).

7.2 Post-COVID Cardiovascular Screening Recommendations

Given the documented cardiovascular sequelae of COVID-19, targeted screening of high-risk groups—including those with MetS, severe acute COVID-19, or persistent symptoms—is recommended. Cardiac biomarkers (troponins, BNP), ECG, echocardiography, and cardiac MRI may be utilized to detect myocarditis, arrhythmias, or fibrosis early (Nakamura et al., 2021). Early identification of at-risk individuals facilitates timely intervention to prevent SCA.

7.3 Role of Vaccination and Antiviral Therapies

COVID-19 vaccination reduces not only acute infection severity but also mitigates long-term cardiovascular complications, lowering post-infection SCA risk (Xie et al., 2022). Antiviral therapies, when administered early, may decrease viral replication and subsequent cardiac injury. Continued efforts to increase vaccine coverage and antiviral accessibility remain vital components of SCA prevention.

7.4 Rehabilitation and Monitoring of Recovered COVID Patients

Post-acute care for COVID-19 survivors should include cardiovascular rehabilitation tailored to individual risk profiles. Exercise programs, autonomic function monitoring, and metabolic control are pivotal in reducing arrhythmogenic risk (Bhatia et al., 2021). Multidisciplinary clinics specializing in Long COVID can provide comprehensive follow-up and patient education.

7.5 Public Awareness and CPR Education

Public health campaigns emphasizing recognition of cardiac arrest signs, widespread CPR training, and access to automated external defibrillators (AEDs) are critical to improving survival rates. Pandemic-related delays in emergency response highlight the need for community empowerment and emergency preparedness (Beaney et al., 2020).

8. Emerging Research and Future Directions

As the COVID-19 pandemic evolves, understanding the long-term cardiovascular consequences—including sudden cardiac arrest (SCA)—remains a critical research priority. Cutting-edge technologies and multidisciplinary approaches offer promising avenues to deepen insight and improve patient outcomes.

8.1 Longitudinal Studies on COVID-19 and Cardiac Health

Ongoing prospective cohort studies tracking COVID-19 survivors over months and years are vital to delineate the natural history of post-infection cardiac complications (Xie et al., 2022). These studies will clarify incidence rates, risk factors, and mechanisms underlying late-onset SCA, guiding clinical monitoring and intervention strategies.

8.2 Omics Approaches (Genomics, Metabolomics, Proteomics)

Multi-omics technologies enable comprehensive profiling of molecular changes induced by SARS-CoV-2 infection. Genomic analyses can identify susceptibility variants linked to arrhythmias and myocardial injury (Nieman et al., 2021). Metabolomics and proteomics further reveal dysregulated metabolic pathways and inflammatory markers, advancing biomarker discovery and personalized risk assessment.

8.3 AI and Machine Learning for Risk Prediction

Artificial intelligence (AI) and machine learning algorithms are increasingly applied to integrate clinical, imaging, and molecular data for early identification of patients at high risk for SCA post-COVID (Dey et al., 2022). These tools promise improved predictive accuracy and individualized care planning.

8.4 Development of Targeted Therapies

Future therapeutic development focuses on targeted modulation of pathogenic pathways such as inflammation, fibrosis, and autonomic dysfunction implicated in post-COVID cardiac injury. Novel anti-inflammatory agents, anti-fibrotics, and gene therapies are under investigation to prevent arrhythmogenesis and improve cardiac recovery (Guzik et al., 2021).

9. CONCLUSION

This comprehensive review highlights the significant surge in sudden cardiac arrest (SCA) following COVID-19, underscoring the complex interplay between metabolic syndrome, lifestyle risk factors, and emerging pathophysiological mechanisms. The cardiovascular impact of SARS-CoV-2 extends beyond the acute infection phase, with inflammation, autonomic dysfunction, and myocardial injury contributing to increased arrhythmogenic risk.

Implications for clinical practice include the urgent need for vigilant cardiovascular screening and management of high-risk populations, particularly those with metabolic syndrome or post-COVID symptoms. From a public health perspective, strategies addressing lifestyle modification, vaccination, and community CPR education are essential to mitigate the growing burden of SCA.

Ultimately, tackling this multifaceted challenge requires integrated, multidisciplinary approaches that combine clinical care, epidemiological surveillance, and cutting-edge research. Collaborative efforts will be critical to improving long-term cardiovascular outcomes in the post-pandemic era.

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