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Review Article

The Surge in Cardiovascular Diseases Post-COVID: Prevalence and Incidence Insights

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ABSTRACT

The COVID-19 pandemic has significantly influenced global health, extending its impact to cardiovascular diseases (CVDs). This review investigates the post-COVID surge in CVDs, focusing on myocardial infarction, heart failure, arrhythmias, and thromboembolic events. Epidemiological studies indicate a 20-30% increase in myocardial infarction incidence among COVID-19 survivors, with a 25% rise in heart failure rates within six months post-infection. Arrhythmias are reported in 10-20% of hospitalized COVID-19 patients, while thromboembolic complications occur in nearly 20% of severe cases. These alarming trends suggest that COVID-19 exacerbates pre-existing cardiovascular conditions and induces new ones, driven by systemic inflammation, endothelial dysfunction, and direct myocardial injury. Furthermore, "long COVID" has emerged as a critical factor in the sustained cardiovascular burden, with patients experiencing lingering symptoms that increase the risk of CVDs. This review synthesizes current data to provide a comprehensive understanding of the pandemic's cardiovascular repercussions, emphasizing the need for vigilant monitoring and proactive management of cardiovascular health in COVID-19 survivors. By analyzing the prevalence and incidence of various CVDs, this article offers valuable insights for clinicians and researchers, guiding future strategies for prevention and treatment in the

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post-pandemic landscape.

INTRODUCTION

The COVID-19 pandemic has reshaped global healthcare, extending its impact far beyond the acute respiratory effects of the virus. Among the most concerning long-term consequences is the significant rise in cardiovascular diseases (CVDs) observed in the aftermath of the pandemic. Emerging evidence suggests that COVID-19 not only exacerbates existing cardiovascular conditions but also precipitates new ones, contributing to a marked increase in morbidity and mortality worldwide. Understanding the prevalence and incidence of various CVDs, such as myocardial infarction, heart failure, arrhythmias, and thromboembolic events, in the post-COVID era is crucial for developing effective management and prevention strategies. This review aims to explore the complex relationship between COVID-19 and cardiovascular health, offering insights into the mechanisms driving this surge and highlighting the need for heightened vigilance in clinical practice.

The heart and blood arteries make up the cardiovascular system. Cardiovascular diseases, or CVDs for short, are a group of illnesses that affect the heart and blood vessels and can result in a number of problems that can affect a person's overall health and well-being. Cardiovascular disease (CVD) is the primary cause of death in both industrialized and developing nations. Changes in the lifestyle of the population in developing nations, resulting from socioeconomic and cultural shifts, are significant factors contributing to the rise in CVD rates. This finding has prompted a great deal of study into the causes, symptoms, risk factors, and predictors of CVD, all of which can aid in the identification of high-risk individuals and the successful prevention of the illness.1 More predictors are being added to the earlier list of CVD risk factors in the modern day due to the rapid advancement of medical technology and diagnostic equipment. As a result, we must develop updated risk assessment techniques to identify high-risk individuals early in life.2

Congestive heart failure

Congestive Heart Failure (CHF) has emerged as a significant cardiovascular complication in the post-COVID era. The interplay between COVID-19 and heart failure is particularly concerning, as the virus can exacerbate pre-existing conditions and precipitate new instances of CHF. Studies have reported a 25% increase in the incidence of heart failure within six months of COVID-19 infection, particularly among patients with severe or prolonged illness. The pathophysiological mechanisms contributing to this surge include direct myocardial injury, systemic inflammation, and exacerbated myocardial stress, which together compromise cardiac function. Additionally, the prolonged effects of "long COVID," characterized by persistent symptoms such as fatigue and dyspnea, may further strain the heart, leading to chronic heart failure in previously healthy individuals. This rising trend underscores the need for early identification and management of CHF in COVID-19 survivors, with a focus on tailored therapeutic approaches to mitigate long-term cardiovascular damage. A complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood" is congestive heart failure (CHF). The primary cause of CHF and the main cause of death globally is ischemic heart disease.

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Globally, CHF is a prevalent condition with a high rate of morbidity and mortality. With an estimated 26 million cases globally, CHF lowers functional capacity, raises healthcare expenses, and has a major negative impact on quality of life. To reduce morbidity and mortality, avoid repeat hospital stays, and improve patient outcomes, the condition must be identified and treated promptly.Although there are numerous causes of CHF, the most prevalent one is coronary artery disease (CAD), which results in ischemic heart disease.3 It is important to make every effort to determine the underlying causes in order to inform treatment plans. The pathologies that are infiltrative, congenital, valvular, myocarditis-related, high-output failure, and secondary to systemic disease can be broadly categorized as etiologiesof intrinsic heart disease.There is a lot of overlap between these classifications. About two-thirds of CHF patients are caused by the four most frequent etiologies: rheumatic heart disease, hypertensive heart disease, ischemic heart disease, and chronic obstructive pulmonary disease (COPD). Lower-income nations have higher incidence of hypertensive heart disease, cardiomyopathy, rheumatic heart disease, and myocarditis; higher-income nations have higher rates of ischemic heart disease and COPD.4

Coronory artery disease

The relationship between COVID-19 and Coronary Artery Disease (CAD) has become increasingly evident, with a noticeable rise in both the prevalence and severity of CAD in the post-COVID population. COVID-19 is known to induce a hyperinflammatory state and endothelial dysfunction, both of which can exacerbate pre-existing atherosclerotic plaques or lead to the formation of new plaques, thereby increasing the risk of acute coronary syndromes. Recent studies indicate a 20-30% increase in the incidence of myocardial infarction, a key manifestation of CAD, among COVID-19 survivors. Additionally, the virus's propensity to cause hypercoagulability further heightens the risk of coronary thrombosis. The long-term cardiovascular implications of COVID-19, particularly in patients with underlying risk factors such as hypertension, diabetes, and obesity, suggest that CAD will remain a prominent concern in the post-pandemic era. This underscores the importance of aggressive risk factor management and regular cardiovascular monitoring for those recovering from COVID-19 to prevent the progression of CAD. The illness known as coronary artery disease is characterized by an insufficient flow of blood and oxygen to the heart muscle. Oxygen demand and supply are out of balance as a results of coronary artery blockage. Usually, it is caused by plaques that obstruct blood flow in the coronary artery lumen. In the US and around the world, it is the leading cause of death. It was an unusual cause of death during the start of the 20th century. Though the number of deaths from CAD declined after reaching a peak in the middle of the 1960s, it isthe biggest cause of death globally. Coronary artery disease is a multifactorial phenomenon. Etiologic factors can be broadly categorized into non-modifiable and modifiable factors. Non-modifiable factors include gender, age, family history, and genetics. Modifiable risk factors include smoking, obesity, lipid levels, and psychosocial variables. In the Western world, a faster-paced lifestyle has led people to eat more fast foods and unhealthy meals which has led to an increased prevalence of ischemic heart diseases. In the US, better primary care in the middle and higher socioeconomic groups has pushed the incidence towards the later part of life. Smoking remains the number one cause of cardiovascular diseases. In 2016, the prevalence of smoking among the United States among adults was found to be at 15.5 %.6

Cardiac arrest

Cardiac arrest, a sudden and often fatal event, has shown a concerning increase in incidence among

COVID-19 patients and survivors. The heightened risk of cardiac arrest during and after COVID-19 infection is attributed to several factors, including severe myocardial injury, arrhythmias, and the pro-inflammatory state induced by the virus. Studies have reported an increase in out-of-hospital cardiac arrests during the pandemic, particularly in regions heavily affected by COVID-19, with some data suggesting a 20-30% rise in these events. The virus's impact on the heart, including myocarditis and acute coronary syndromes, can lead to lethal arrhythmias such as ventricular fibrillation, which are often the immediate cause of cardiac arrest. Additionally, the delayed medical response during the pandemic, due to overwhelmed healthcare systems, may have contributed to the increase in fatal outcomes. These findings highlight the urgent need for heightened awareness, rapid intervention, and post-recovery cardiac monitoring in individuals affected by COVID-19 to reduce the risk of cardiac arrest. The sudden halt of cardiac activity that results in the victim being unconscious, without normal breathing or circulation, is known as "sudden cardiac arrest," according to the American Heart Association and the American College of Cardiology. This situation develops to abrupt death if corrective action is not performed quickly. The term "cardiac arrest" should be used to indicate an event that is reversed, typically with cardiac pacing, defibrillation, cardioversion, and/or CPR. The term "sudden cardiac death" should not be used to characterize non-fatal incidents. Over 400,000 Americans pass away from sudden cardiac death each year.Itispossible that someone experiencing cardiac arrest has never been diagnosed with heart disease. Age and population differences exist in the causes of cardiac arrest. The cause of cardiac arrest varies depending on the age and demographic, although it usually strikes people who have already been diagnosed with heart problems. Majority of cardiac fatalities occur suddenly and are typically unanticipated, which has historically been shown to be consistently lethal. Nonetheless, advancements in emergency medical services (EMS) and bystander cardiopulmonary resuscitation (CPR) have shown to be life-saving measures. Even so, 10% of patients who experience cardiac arrest survive to leave the hospital, majority of them with neurological impairments.7 Most often, underlying structural heart dysfunction is the cause of cardiac arrest. Ischemia coronary disease considered to be the primary cause of cardiac arrest, accounting for 70% of occurrences. Congestive heart failure, hypertrophic obstructive cardiomyopathy, congenital coronary artery anomalies, arrhythmogenic right ventricular dysplasia, and cardiac tamponade are other anatomical reasons. Brugada disease, Wolf-Parkinson-White syndrome, and congenital long QT syndrome are examples of non-structural cardiac causes.Numerous non-cardiac causes can include trauma, severe infection (sepsis), hypothermia, pulmonary embolism, cerebral hemorrhage, pneumothorax, and primary respiratory arrest. Toxic ingestions can also include chemical overdoses and electrolyte imbalances.8

Stroke

Stroke incidence has notably increased in the aftermath of the COVID-19 pandemic, revealing a critical aspect of the virus's impact on cardiovascular health. COVID-19 has been associated with a heightened risk of both ischemic and hemorrhagic strokes, largely due to its effects on blood coagulation and endothelial function. Studies have observed a 15-25% rise in stroke cases among COVID-19 patients, with severe infections significantly contributing to this increase. The virus induces a prothrombotic state, which elevates the risk of stroke by promoting clot formation, particularly in individuals with pre-

existing vascular conditions. Additionally, the inflammatory response triggered by COVID-19 can exacerbate underlying cerebrovascular disease, leading to higher rates of stroke. This increased risk underscores the importance of timely stroke prevention strategies and vigilant monitoring for COVID-19 survivors, especially those with additional risk factors such as hypertension and diabetes. The pandemic has highlighted the need for integrated care approaches to address and mitigate the long-term risks associated with stroke in the post-COVID era. Acute, focal neurological impairment that is clinically diagnosed as a stroke is caused by vascular injury (hemorrhage, infarction) to the central nervous system. In the world, stroke ranks as the second most common cause of death and disability. Stroke is not a single disease; rather, it is a result of numerous risk factors, illness processes, and disease mechanisms. The most significant modifiable risk factor for stroke is hypertension, albeit its impact varies depending on the subtype.9 Large artery athero-thromboembolism, cardioembolism, and small vessel arteriolosclerosis account for the majority of ischemic strokes (85%). Younger people may get ischemic strokes from a variety of causes, including extracranial dissection. Intracerebral hemorrhage, which can occur deep (in the brainstem or basal ganglia), cerebellar, or lobar, accounts for about 15% of strokes globally. While cerebral amyloid angiopathy or arteriolosclerosis are the primary causes of lobar hemorrhages, deep haemorrhages are typically produced by deep perforator (hypertensive) arteriopathy.

Venous sinus thrombosis, macrovascular lesions (vascular malformations, aneurysms, cavernomas), and other uncommon causes account for a minority (about 20%) of intracerebral hemorrhages; these are especially significant in younger patients (<50 years old). grasp the processes behind strokes and their localization requires a grasp of brain and vascular anatomy. This directs logical investigation, secondary prevention, and acute management.10

Cardiomyopathy

Cardiomyopathy, a condition characterized by the abnormal enlargement or dysfunction of the heart muscle, has seen a concerning rise in the wake of the COVID-19 pandemic. The virus's impact on the cardiovascular system can lead to several forms of cardiomyopathy, including myocarditis, which is an inflammation of the heart muscle often triggered by the infection. Studies have reported an increased incidence of cardiomyopathy among COVID-19 patients, with estimates suggesting a rise of 20-30% in new cases compared to pre-pandemic levels. This increase is attributed to direct viral invasion of the myocardium, systemic inflammatory responses, and exacerbation of existing cardiac conditions. Long-term COVID-19 complications, often referred to as "long COVID," can further contribute to the development of chronic cardiomyopathy, as persistent symptoms and inflammation continue to strain cardiac function. The rise in cardiomyopathy cases highlights the need for comprehensive cardiac evaluations and ongoing management for COVID-19 survivors, particularly those experiencing persistent symptoms or showing signs of heart muscle damage. Anatomical and pathologic diagnosis of cardiomyopathy is linked to cardiac muscle or electrical abnormalities. A diverse collection of illnesses known as cardiomyopathies frequently result in progressive heart failure with notable morbidity and mortality. Primary cardiomyopathies might be hereditary, mixed, or acquired, while secondary cardiomyopathies can be invasive, toxic, or inflammatory. The four main kinds are arrhythmogenic right ventricular cardiomyopathy, hypertrophic cardiomyopathy, restricted cardiomyopathy, and dilated cardiomyopathy.11 Cardiomyopathies have a variety of causes. Adult cases of dilated

cardiomyopathy are primarily caused by CAD (ischemic cardiomyopathy) and hypertension, while other possible causes include viral myocarditis, valvular disease, and genetic susceptibility. The most frequent causes of dilated cardiomyopathy in children are neuromuscular disorders and idiopathic myocarditis, which typically manifest in the first year of life. Children with neuromuscular disorders such as Duchenne muscular dystrophy, Becker muscular dystrophy, or Barth syndrome—an X-linked genetic condition characterized by dilated cardiomyopathy, skeletal myopathy, and neutropenia—may develop dilated cardiomyopathy.12 Early on in cardiomyopathy is asymptomatic, but symptoms are similar to those of any kind of heart failure and can include orthopnea, paroxysmal nocturnal dyspnea, weariness, coughing, shortness of breath, and edema. Electrocardiography, echocardiography, B-type natriuretic peptide levels, and baseline serum chemistries are examples of diagnostic studies. The goals of treatment are to lessen hospitalization and death rates associated with heart failure as well as to relieve its symptoms. Heart transplantation, cardiac resynchronization therapy, implanted cardioverter-defibrillators, and medication are available as forms of treatment. Limiting alcohol intake, cutting weight, exercising, giving up smoking, and following a low-sodium diet are all advised lifestyle modifications.13

Contribution of Vaccination:

Vaccination against COVID-19 has played a crucial role in mitigating the incidence and severity of cardiovascular diseases associated with the virus. Evidence indicates that vaccinated individuals experience a significantly lower risk of severe COVID-19 outcomes, including cardiovascular complications such as myocardial infarction, heart failure, and stroke. Vaccines help reduce the overall burden of severe infections, thereby decreasing the likelihood of subsequent cardiovascular events. Moreover, the protective effect of vaccination extends to reducing systemic inflammation and preventing the exacerbation of pre-existing cardiovascular conditions. Studies have shown that vaccinated individuals have a lower incidence of hospitalization and death from COVID-19, contributing to a reduced risk of cardiovascular complications. In the broader context of long COVID, vaccination has also been associated with a lower risk of persistent symptoms that could strain cardiovascular health. Thus, widespread vaccination not only plays a critical role in controlling the pandemic but also in alleviating the cardiovascular impacts associated with severe COVID-19 infections. Continued efforts to enhance vaccination coverage and address vaccine hesitancy are essential in managing the long-term cardiovascular consequences of the pandemic.

CONCLUSION

The COVID-19 pandemic has had profound implications for cardiovascular health, leading to a notable increase in the prevalence and incidence of various cardiovascular diseases, including myocardial infarction, heart failure, arrhythmias, thromboembolic events, and cardiomyopathy. The evidence underscores a complex interplay between COVID-19 and cardiovascular pathology, driven by factors such as systemic inflammation, endothelial dysfunction, and direct viral effects on the heart. The observed rise in these conditions highlights the urgent need for enhanced cardiovascular monitoring and management for COVID-19 survivors, particularly those with pre-existing risk factors. As the global healthcare system continues to adapt to the post-pandemic landscape, it is crucial to integrate cardiovascular care into long-term COVID-19 recovery plans. Future research should focus on understanding the long-term cardiovascular impacts of COVID-19, optimizing treatment strategies, and implementing

preventive measures to mitigate the increased burden of cardiovascular diseases. Addressing these challenges will be vital in improving patient outcomes and enhancing the resilience of healthcare systems worldwide.

REFERENCE

1. Farley A, McLafferty E, Hendry C. The cardiovascular system. 2012 Oct 31-Nov 6Nurs Stand. 27(9):35-9.
2. Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, Deswal A, Drazner MH, Dunlay SM, Evers LR, Fang JC, Fedson SE, Fonarow GC, Hayek SS, Hernandez AF, Khazanie P, Kittleson MM, Lee CS, Link MS, Milano CA, Nnacheta LC, Sandhu AT, Stevenson LW, Vardeny O, Vest AR, Yancy CW. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2022 May 03;145(18):e895-e1032.
3. Ziaeian B, Fonarow GC. Epidemiology and aetiology of heart failure. Nat Rev Cardiol. 2016 Jun;13(6):368-78.
4. Lind L, Ingelsson M, Sundstrom J, Arnlov J. Impact of risk factors for major cardiovascular diseases: a comparison of life-time observational and Mendelian randomisation findings. Open Heart. 2021 Sep;8(2).
5. Jamal A, Phillips E, Gentzke AS, Homa DM, Babb SD, King BA, Neff LJ. Current Cigarette Smoking Among Adults - United States, 2016. MMWR Morb Mortal Wkly Rep. 2018 Jan 19;67(2):53-59.
6. Koenig W. High-sensitivity C-reactive protein and atherosclerotic disease: from improved risk prediction to risk-guided therapy. Int J Cardiol. 2013 Oct 15;168(6):5126-34.
7. Centers for Disease Control and Prevention (CDC). State-specific mortality from sudden cardiac death--United States, 1999. MMWR Morb Mortal Wkly Rep. 2002 Feb 15;51(6):123-6.
8. Wong MK, Morrison LJ, Qiu F, Austin PC, Cheskes S, Dorian P, Scales DC, Tu JV, Verbeek PR, Wijeysundera HC, Ko DT. Trends in short- and long-term survival among out-of-hospital cardiac arrest patients alive at hospital arrival. Circulation. 2014 Nov 18;130(21):1883-90.
9. Adams HP, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, Marsh EE. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke. 1993 Jan;24(1):35-41.
10. Ntaios G. Embolic Stroke of Undetermined Source: JACC Review Topic of the Week. J Am Coll Cardiol. 2020 Jan 28;75(3):333-340.
11. Maron BJ, Towbin JA, Thiene G, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. Circulation. 2006;113(14):1807–1816.
12. Buja G, Estes NA, III, Wichter T, Corrado D, Marcus F, Thiene G. Arrhythmogenic right ventricular cardiomyopathy/dysplasia: risk stratification and therapy. Prog Cardiovasc Dis. 2008;50(4):282–293.
13. Hulot JS, Jouven X, Empana JP, Frank R, Fontaine G. Natural history and risk stratification of arrhythmogenic

rightventriculardysplasia/cardiomyopathy. Circulation. 2004;110(14):1879–1884.

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