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Theme: Is there any link between Heart Attacks and Covid 19

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Cardio Metabolic Risk Factor Associated With Covid -19

Abhijit Das
Executive Officer, Zydus Cadila
dasabhijit00@gmail.com

Abstract:
The cardiovascular system is significantly affected in corona virus disease-19 (COVID-19). Micro vascular injury, endothelial dysfunction, and thrombosis resulting from viral infection or indirectly related to the intense systemic inflammatory and immune responses are characteristic features of severe COVID-19. Pre-existing cardiovascular disease and viral load are linked to myocardial injury and worse outcomes. The vascular response to cytokine production and the interaction between severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) and angiotensin-converting enzyme 2 receptor may lead to a significant reduction in cardiac contractility and subsequent myocardial dysfunction. In addition, a considerable proportion of patients who have been infected with SARS-CoV-2 do not fully recover and continue to experience a large number of symptoms and post-acute complications in the absence of a detectable viral infection. These conditions often referred to as 'post-acute COVID-19 may have multiple causes. Viral reservoirs or lingering fragments of viral RNA or proteins contribute to the condition. Systemic inflammatory response to COVID-19 has the potential to increase myocardial fibrosis which in turn may impair cardiac remodeling. Here, we summarize the current knowledge of cardiovascular injury and post-acute squeal of COVID-19. As the pandemic continues and new variants emerge, we can advance our knowledge of the underlying mechanisms only by integrating our understanding of the pathophysiology with the corresponding clinical findings. Identification of new biomarkers of cardiovascular complications and development of effective treatments for COVID-19 infection are of crucial importance.

Keywords: SARS-CoV-2, RNA, Pathophysiology

Introduction

To date, the corona virus disease 2019 (COVID-19) pandemic has affected over 214 million of people and caused over 4.4 million deaths since December of 2019. Initially thought to be an acute respiratory distress syndrome (ARDS), it has since become clear that COVID-19 is in fact a multiple organ disease. The disease is characterized by cytokine storm, resulting in endothelial inflammation/dysfunction, micro and macro-vascular thrombosis, which may damage organs other than the lung. Human studies have offered an alarming view of the risks of severe complications in elderly patients and in those with underlying cardiovascular disease or who are at high cardiovascular risk due to one or more risk factors such as

hypertension, diabetes mellitus, hypercholesterolemia, or obesity. Moreover, recent studies revealed that some biological changes induced by COVID-19 throughout the organs are long-lasting. Consistent with this finding, a large number of patients who have been infected with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) continue to experience symptoms after the acute phase of the acute infection, which can evolve over time and persist for months. While still being defined, these effects are referred to as Post-Acute Squeal of SARS-CoV-2 infection or 'Long COVID. Therefore, the magnitude of the problem is still unknown. Post-acute COVID-19 is a matter of major concern for patients affected by cardiovascular disease, given that the presence of underlying cardiovascular co morbidities in patients with COVID-19 is associated with high mortality and COVID-19 can cause cardiovascular disorders, including myocardial injury, arrhythmias, acute coronary syndrome (ACS), and venous thromboembolism (VTE)[1-3]. Cardiovascular disease remains the leading cause of morbidity and mortality globally and is associated with 17.8 million deaths annually[4]. We cannot predict the impact of post-acute COVID-19 on future cardiovascular outcomes. Nevertheless, to meet the urgent need for effective treatment and preventative strategies, rigorous efforts should be made to investigate and integrate biological and clinical findings related to COVID-19 in cardiovascular disease. In this position paper, we assessed the evidence supporting the mechanisms of acute and post-acute cardiovascular injury among patients with COVID-19 and their clinical features to identify gaps that need to be addressed in future research[1, 2].

Underlying Cardio metabolic risk factors associated with worse outcome in COVID-19: Hypertension or age?

Globally, an estimated 1.13 billion individuals worldwide have hypertension, and the greatest burden is in individuals aged 60 years and older. Early small case series offered an alarming view suggesting that people living with hypertension were at higher risk of severe COVID-19 and mortality. Preliminary data showed that the incidence of hypertension ranged from 32.6% to 34% among confirmed patients with COVID-19. Among patients with myocardial injury and elevated cardiac troponin T levels, 63.5% had hypertension. Similar findings were observed concerning mortality from COVID-19. A meta-analysis incorporating early data of patients with COVID-19, demonstrated that the presence of hypertension was associated with nearly 2.5-fold higher risk of severe disease, intensive care unit (ICU) hospitalization, and mortality. Altogether, these findings indicate that hypertensive patients have a higher risk of developing severe COVID-19. However, the mechanisms that link pre-existing hypertension and COVID-19 are yet to be fully elucidated as hypertension coexists with many other risk factors. One approach to disentangle the independent relationship between COVID-19 outcomes and exposure to hypertension is to study patients with hypertension while excluding those with other known risk factors of adverse outcomes. Recent evidence from the UK population-based study Open SAFELY involving over 17 million patients was based on this approach. Open SAFELY quantified a wide range of clinical risk factors for death from COVID-19, some of which were not previously well characterized. There was no association between hypertension (defined as a recorded diagnosis, or blood pressure ≥140/90 mmHg at the last measurement) and COVID-19 mortality (hazard ratio: 0.95, 95% confidence interval: 0.89-1.01). In contrast, age, cardiovascular disease, diabetes, obesity, respiratory diseases, history of malignancy kidney, liver, neurological, and autoimmune diseases were all associated with increased risk of death. The strongest predictor of mortality was age[3,4].

Other recent studies reinforced these observations reporting that age >60 years, overweight/obesity and, diabetes but neither hypertension nor anti-hypertensive treatments were associated with adverse prognosis. Poor blood pressure control is associated with target end-organ damage, and means blood pressure increases with age. Additionally, age-related low-grade chronic inflammation with enhanced pro-inflammatory cytokines and chemokines, underlie several cardiovascular diseases including hypertension, which in turn is associated with senescence of $CD8^{+}$T cells, a mainstay of antiviral immunity. A small study showed those macrophages and neutrophils of hypertensive patients with COVID-19, exhibit higher expression of pro-inflammatory chemokines such as ligands for chemokines with two

adjacent cytokines (CCL3, CCL4) and the chemokines receptor CCR1. A recent study showed an age-related increase of ACE2 expression in human kidney and lung tissues and lack of association between hypertension, RAS blockers, and renal expression of ACE2. These findings are in agreement with previous reports suggesting that RAS blockers use were not associated with higher ACE2 and TMPRSS2 expression in lung tissues, or with increased circulating plasma concentrations of ACE2. Taken together, these observations may explain the reported associations between age, hypertension, and severity of COVID-19 infection. In sum, hypertension is very strongly associated with age and although many studies adjusted for this, disentangling the effects of each other is difficult. Age appears to be the strongest predictor for severe disease and mortality in COVID-19, which may be due to immune senescence, inflammation, exaggerated AT_1 pro-inflammatory, pro-thrombotic and pro-fibrotic signaling[4].

Obesity

Obesity and particularly metabolically unhealthy obesity are major contributors to cardiovascular disease, and mortality. Achieving a metabolically healthy weight is a risk modifier associated with improved cardiac and vascular function.

Epidemiological data show a J-shaped relationship between body mass index (BMI), COVID-19 severity and mortality, with lower risks at BMI thresholds near normal weights. Interestingly, this relationship was more pronounced among younger patients (<65 years old). In COVID-19 patients from New York City, those aged under 60 years with a BMI ranging from 30 to 34 kg/m^2 had a 2-fold increase in the probability of ICU admission compared to patients with a BMI <30 kg/m^2. This likelihood increased to 3.6-fold in patients with a BMI ≥35 kg/m^2. Likewise, a BMI >35 kg/m^2 increased the risk of invasive mechanical ventilation 7-fold and was associated with lower survival rates. In the Open Safely study, adjusted mortality rates increased with increasing BMI ranging from 1.05 for BMI <34.9 kg/m^2 to 1.92 for BMI ≥40 kg/m^2 when compared with non-obese patients. Thus, the relationship between obesity and severe COVID-19 and whether obesity could shift this increased risk into younger age groups is still a matter of concern given the high burden of obesity. Some studies addressed the question of why COVID-19 is deadlier in people with obesity, even if they are young. These studies noted that fat distribution and an impaired adipose tissue function, rather than total fat mass and BMI are related with COVID-19 complications at the individual level especially in younger patients[2,3].

In one small study of patients with COVID-19, 10 cm^2 of increase in visceral adipose tissue area, measured by computed tomography (CT), was associated with a 1.36-fold increase in risk for ICU hospitalization. In contrast, BMI and total adipose tissue area showed weak association with COVID-19 severity.

Smoking

Smoking remains a leading risk for early death and disability with 6.4 million deaths per year attributable to smoking worldwide. Smoking is an independent risk factor for atherosclerotic cardiovascular disease and a factor positively associated with respiratory diseases, impaired immune system and consequently increased incidence of infectious diseases. Smoking has been shown to up-regulate ACE2 expression especially in the lower respiratory tract which might make current smokers vulnerable to infection by COVID 19 compared with former/never smokers.

However, data on this issue are controversial. Several early observational studies and subsequent meta-analyses based on these reports found an inverse relationship between smoking and severe COVID-19 leading to the misconception that current smoking is of benefit during COVID 19 infection. In contrast, other reports linked current smoking with severe clinical course of COVID-19 and need of ICU care. Among of 8910 hospitalized patients with COVID-19 current smokers accounted for 5.5% of the study population. A 1.79-fold increase in hospital mortality was observed in current smokers as compared with former/never smokers. The same may also be the case for water pipe, electronic cigarettes or 'heat-not-burn' IQOS users. Of note, the prevalence of smokers was higher amongst those patients with myocardial injury as assessed by increased cardiac troponin T levels compared

with non-smokers (13.5% vs. 8.1%). In sum, further studies are needed to clarify the reasons behind the reported low prevalence of current smokers among hospitalized patients with COVID-19. The effect of current smoking on SARS-CoV-2 infection is a delicate and complex topic that should be addressed rigorously before delivering messages that could be misinterpreted[5].

What causes elderly patients, men and those with cardio metabolic risk factors to be at higher risk of severe COVID-19?

Observations suggest the importance of a 'metabolic disease exposure, including dietary lifestyle, glycogenic disorders, obesity, and sedentariness, among other potential disease severity modifiers such as systemic hypertension and aging, leading to chronic low-grade inflammation, which may aggravate COVID-19-induced acute organ failure. Therefore, it is essential to precisely identify the factors underlying the severity and the clinical presentation of the disease, especially when considering the risk of COVID-19 epidemics. Aging has also a significant effect on the response to pharmacological interventions. It may be necessary to design trials that focus exclusively on elderly. Finally, ageing is associated to oxidative stress and immune-senescence impairing therefore the answer of the immune system against the viral insult. The study population of an RCT should ideally reflect the population that is at the highest risk of the disease and that is most difficult to treat in clinical practice.

How can we better address failure of the microcirculation in COVID-19 and personalize potential therapeutic approaches?

Persistence of SARS-CoV-2 and viral RNA may act as potential viral reservoirs. They tend to induce an inflammatory response, stimulating endothelial dysfunction, accelerated atherosclerosis, hypercoagulability, and micro vascular thrombosis. Thus, novel and prognostic biomarkers, combined with genetic differences and functional testing are required. To tackle the challenge, a large international interdisciplinary network of clinical and non-clinical scientists is needed. Initial examples of such cooperation are the EU-Cardiogram COST Action CA17129 which would try to identify RNA biomarkers combined with artificial intelligence.

Future directions

We have only known about the SARS-CoV-2 for only one and a half year, it is actually quite remarkable how much we have learned about its epidemiology and pathophysiology. As we increase our understanding of the disease there is growing consensus that COVID-19 is a macro- and micro-vascular disease and as such the cardiovascular system is largely affected. While, both clinical and basic research has been very responsive to tackle the challenge, most questions, What makes some people more vulnerable than others? What are the biological mechanisms underlying this? however, remain unsolved, as our understanding of the pathophysiology of the disease is still under evolving and needs to be addressed and expanded in future research efforts. Approximately 25% of individuals who had COVID-19 still have physical symptoms one month after they became ill, and about 10% have symptoms that persist after 12 weeks. COVID-19 is a 'new disease' that pushes the research community and the world more generally into 'uncharted territories'. We should commit to set up a network of scientists and laboratories around Europe taking a multidisciplinary approach.

Conclusion

The presence of cardiovascular diseases such as arterial hypertension and type 2 diabetes mellitus constitute a risk factor that increases the possibility of a serious outcome in acute COVID-19. The systematic follow-up of COVID-19 patients with a deep and complete clinical and biological phenotype will enable the identification of individuals at risk in order to provide personalized care and with the aim of preventing further vulnerability for-and exposure to long-term sequel.

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Article: 2

Post Covid Effect on Heart Patients

Shradhanjali Jena
Utkal University, Bhubaneswar, Odisha - 751004
shradhanjali444jena@gmail.com

Abstract
The cardiovascular complications of acute Coronavirus disease 2019 (COVID-19) are well-documented, but its long-term cardiovascular manifestations remain insufficiently characterized. Using national healthcare databases from the US Department of Veterans Affairs, we analyzed a cohort of 153,760 individuals with COVID-19 and two control groups: contemporary controls (5,637,647 individuals) and historical controls (5,859,411 individuals). This study estimates the risks and 12-month burdens of pre-specified cardiovascular outcomes. Beyond the acute phase, COVID-19 survivors displayed elevated risks for cerebrovascular disorders, dysrhythmias, ischemic and non-ischemic heart disease, pericarditis, myocarditis, heart failure, and thromboembolic disease. These risks were observed even among non-hospitalized individuals and escalated with increased severity of the acute infection. Our findings highlight the substantial cardiovascular burden in COVID-19 survivors, necessitating a focus on cardiovascular health in post-acute care.
Keywords: Coronavirus, COVID-19, Heart disease

Introduction
Post-acute sequelae of severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2), the causative agent of COVID-19, affect multiple organ systems, including the cardiovascular system[1]. While prior studies have examined cardiovascular outcomes post-COVID-19, their focus has been limited to hospitalized individuals and short follow-up durations. Comprehensive assessments of long-term cardiovascular outcomes across all care settings non-hospitalized, hospitalized, and intensive care unit (ICU)-admitted patients are lacking. Addressing this knowledge gap is critical for optimizing post-acute COVID-19 care strategies[2].
This study leverages the US Department of Veterans Affairs national healthcare databases to assess the long-term cardiovascular risks in COVID-19 survivors. The study population includes 153,760 individuals who survived the first 30 days of COVID-19, alongside contemporary and historical control groups, followed for up to 12 months to evaluate the incidence of cardiovascular outcomes.

Methods
Cohort design: The COVID-19 cohort consisted of 153,760 US veterans diagnosed with COVID-19. Control groups included 5,637,647 contemporary users of the Veterans Health Administration (VHA) system without evidence of SARS-CoV-2 infection and 5,859,411 historical controls from 2017[1].
Follow-up period: Median follow-up times were 347 days (COVID-19 cohort), 348 days (contemporary controls), and 347 days (historical controls)[1].

Outcomes: Pre-specified incident cardiovascular outcomes included cerebrovascular disorders, dysrhythmias, ischemic and non-ischemic heart disease, pericarditis, myocarditis, heart failure, and thromboembolic disease.

Data analysis: Risks and 12-month burdens of cardiovascular outcomes were estimated using a combination of hazard ratios (HRs) and absolute risk differences, adjusted for demographic and clinical variables.

Results

Cohort characteristics

COVID-19 cohort: 153,760 participants.

Contemporary controls: 5,637,647 participants.

Historical controls: 5,859,411 participants.

Incident cardiovascular outcomes

Overall findings: COVID-19 survivors had significantly higher risks of developing cardiovascular disorders within 12 months post-infection compared to controls.

Cerebrovascular disorders: Increased risk of stroke and transient ischemic attacks.

Dysrhythmias: Elevated incidence of atrial fibrillation and ventricular arrhythmias.

Heart disease: Higher rates of ischemic and non-ischemic heart disease, pericarditis, and myocarditis.

Heart failure: Substantially increased risk of heart failure.

Thromboembolic disease: Higher incidence of pulmonary embolism and deep vein thrombosis.

Severity of acute infection and cardiovascular risk

Non-hospitalized individuals exhibited elevated risks, though less pronounced than hospitalized and ICU-admitted patients.

Risks increased in a graded fashion according to care setting severity[1-3].

Discussion

The findings underscore the significant long-term cardiovascular burden of COVID-19, even among individuals with mild acute disease. This necessitates routine cardiovascular evaluation in post-COVID-19 care pathways, irrespective of initial disease severity. Increased surveillance and early intervention strategies may mitigate long-term cardiovascular complications[1].

Heart problems after COVID do happen, sometimes even in people who have mild cases. After you recover, you may be at a higher risk of a heart attack or heart failure. This is often because of inflammation from your body's response to the COVID-19 infection. SARS-CoV-2, the virus that causes COVID-19, most commonly affects the lungs but it can also lead to serious heart problems. Lung damage caused by the virus prevents oxygen from reaching the heart muscle, which in turn damages the heart tissue and prevents it from getting oxygen to other tissues.

Conclusion

COVID-19 survivors face a substantial risk of developing cardiovascular diseases within one-year post-infection. COVID-19 can increase the risk of heart attack and stroke for years. It can also cause other heart problems, such as irregular heart rhythms, heart failure, cardiac arrest, and strokes. These risks persist across all levels of acute disease severity and highlight the need for comprehensive cardiovascular monitoring and care in post-acute COVID-19 management.

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Article: 3

Exploring the Connection between Heart Attacks and COVID-19

Aakash Gupta
Department of Pharmaceutical Technology, JIS University, Kolkata, West Bengal, 700109
guptaaakash63387@gmail.com

Abstract
COVID-19, caused by the SARS-CoV-2 virus, has affected millions of people worldwide and brought many health complications to light. Among these, its impact on the heart is gaining attention. Recent studies suggest a connection between COVID-19 and an increased risk of heart attacks. The virus can create a storm of inflammation, damage blood vessels, and cause blood clots, all of which can lead to heart issues. This article explores how COVID-19 may trigger heart attacks and what can be done to manage this risk.
Keywords: COVID-19, Heart attacks, Inflammation, Blood clots, Cardiovascular health

Introduction

When COVID-19 emerged, it was seen primarily as a respiratory illness. However, as we learned more, it became clear that the virus can also harm other parts of the body, including the heart. Heart attacks, which occur when blood flow to the heart is blocked, have been reported in patients with COVID-19. This link is particularly concerning for those with pre-existing heart conditions. SARS-CoV-2, the virus that causes COVID-19, most commonly affects the lungs but it can also lead to serious heart problems. Lung damage caused by the virus prevents oxygen from reaching the heart muscle, which in turn damages the heart tissue and prevents it from getting oxygen to other tissues.

Table 1: The link between heart attacks and COVID-19

Aspects	Heart attack	Covid-19	Ref
Definition	A heart attack, also known as a myocardial infarction, occurs when blood flow to a part of the heart is blocked. This blockage is typically caused by a buildup of fat, cholesterol, and other substances (plaque) in the arteries, which supply blood to the heart. When the blood supply is cut off, the affected heart muscle can be damaged or die.	COVID-19 is a contagious respiratory illness caused by the SARS-CoV-2 virus. It primarily affects the lungs but can impact multiple organ systems, including the heart. COVID-19 spread rapidly worldwide, leading to a global pandemic.	
Symptoms	1. Chest pain or discomfort (pressure, squeezing, or fullness) 2. Shortness of breath 3. Pain in the arms, neck, jaw, back, or stomach 4. Cold sweats, nausea, or dizziness	1. Fever, cough, and fatigue 2. Loss of taste or smell 3. Shortness of breath or difficulty breathing 4. Muscle aches, sore throat, and congestion 5. Severe cases may involve pneumonia or organ failure	1
Risk Factors	1. High blood pressure 2. High cholesterol 3. Smoking 4. Obesity 5. Diabetes 6. Family history of heart disease	1.Advanced age 2.Chronic illnesses (e.g., diabetes, heart disease, lung disease) 3.Compromised immune system 4.Obesity	

Factors increasing the risk of heart attacks due to COVID-19

Inflammation: The virus triggers a strong immune response, leading to inflammation that may destabilize plaques in arteries.

Blood clots: COVID-19 can cause a hypercoagulable state, increasing the risk of clots that block blood flow.

Oxygen deprivation: Severe respiratory issues can reduce oxygen levels, straining the heart.

Heart muscle damage: COVID-19 may directly infect heart cells or cause myocarditis (inflammation of the heart muscle).

Table 2: How COVID-19 influences cardiovascular health

Condition	Mechanism	Effect on health	Ref
Inflammation	Cytokine storm destabilizes plaques in arteries.	Increases the risk of Acute coronary syndrome (ACS).	2
Endothelial damage	Virus infects the blood vessel lining, causing dysfunction.	Promotes clot formation and reduces blood flow to the heart.	
Hypercoagulable state	Elevated clotting factors lead to blood clots in arteries.	Increases heart attack risk, especially in severe COVID-19 cases.	
Oxygen deprivation	Severe respiratory symptoms reduce oxygen supply to the heart.	Causes stress on the heart, leading to type 2 myocardial infarction.	
Pre-existing conditions	Hypertension, diabetes, or cardiovascular diseases worsen outcomes.	Higher likelihood of severe disease and complications.	
Post-Covid syndromes	Persistent inflammation or myocarditis seen in recovered patients.	May increase the long- term risk of heart attacks or other cardiovascular events.	

Table 3: Diagnosis and treatment of heart attack and Covid-19

Condition	Diagnosis	Treatment	Ref
Heart attack	•**Electrocardiogram (ECG):** Detects abnormal heart rhythms. •**Blood tests (Troponin, CK-MB):** Measures heart muscle damage. •**Echocardiogram:** Assesses heart function and damage.	•**Aspirin:** Reduces blood clot formation. •**Nitroglycerin:** Relieves chest pain by dilating blood vessels. •**Thrombolytic** (Clot-busting drugs): Dissolves clots.	2
Heart attack + Covid-19	•Blood Tests (D-dimmer, CRP): Monitors inflammation and clotting. •Simultaneous ECG and RT-PCR: To diagnose both conditions. •Chest CT scan and Echocardiogram: Assess lung and heart damage.	•**Anticoagulants**: Prevents blood clotting, reducing the risk of stroke or heart issues. •**Blood thinners**: Manage clotting risks in both conditions. •**Integrated care approach**: Coordination between cardiologists and infectious disease specialists.	

Why It Matters

The COVID-19-specific mortality rate has been reported to be from 2% to 20%, depending on the availability of medical resources and economic status. One of the most important issues in managing COVID-19 is the accurate and early identification of high-risk patients. Early risk stratification can help medical decision making and resource allocation, for example, high-risk patients can be transferred to the intensive care unit for close monitoring

and organ support. Although several studies have investigated the risk factors for mortality in COVID-19, there has been no systematic effort to develop a prediction tool for risk stratification at an early stage[3]. Recognizing this link is important for saving lives. Doctors need to closely monitor COVID- 19 patients for heart-related symptoms like chest pain, shortness of breath, or dizziness. Preventative treatments, like blood thinners or anti-inflammatory drugs, can help reduce risks.

Conclusion

COVID-19 is not just a lung disease; it can affect the heart as well. By understanding how it contributes to heart attacks, we can take steps to protect at-risk individuals. Early detection, careful monitoring, and lifestyle changes are key to reducing this risk. The fight against COVID-19 is not just about managing the virus but also preventing its complications.

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Article: 4

Complication in Cardiac Patients with Covid 19

Dr. Susmita Dutta
Dental Surgeon, Assam
drsusmita2011@gmail.com

Abstract:

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has significant systemic effects beyond the respiratory system. Among the emerging concerns is its impact on cardiovascular health, including an increased risk of myocardial infarction (heart attack). This review aims to explore the pathophysiological mechanisms, clinical observations, and long-term implications of COVID-19 on cardiovascular health, with a focus on heart attacks.

Keywords: SARS-CoV-2, pathophysiology, COVID-19.

Introduction:

COVID-19, primarily known as a respiratory illness[1], has demonstrated widespread effects on multiple organ systems, including the cardiovascular system. Studies suggest a significant increase in the incidence of acute coronary syndromes (ACS) in COVID-19 patients, highlighting the virus's potential role in triggering heart attacks.[2] Cardiovascular complications are a common manifestation of acute and post-acute COVID-19 infection. Complications include cardiomyopathy, myocardial infarction, arrhythmias, heart failure, and deep venous thrombosis. Pathophysiology remains poorly defined and complex.

Pathophysiological mechanisms

COVID-19 influences cardiovascular health through several pathways:

Inflammatory response: The cytokine storm associated with severe COVID-19 leads to systemic inflammation, endothelial dysfunction, and plaque rupture.

Hypercoagulability: COVID-19 is linked with increased clot formation due to elevated D-dimmer levels, platelet activation, and fibrinogen, heightening the risk of thrombotic events.

Direct myocardial injury: SARS-CoV-2 may directly infect cardiac tissue through ACE2 receptors, causing myocarditis and stress-induced cardiomyopathy.

Hypoxia and demand ischemia: Severe respiratory failure leads to hypoxia, which can exacerbate myocardial oxygen demand-supply mismatch, precipitating ischemic events.

Complications in COVID-19 Patients with myocardial infarction

COVID-19 patients experiencing myocardial infarction are at higher risk of complications

such as, carcinogenic shock, arrhythmias, heart failure and higher in hospital mortality

Therapeutic implications:

Antithrombotic therapy: Given the hypercoagulable state in COVID-19, anticoagulation therapy is often recommended to prevent thrombotic complications. Low-molecular-weight heparin (LMWH) and direct oral anticoagulants (DOACs) have shown benefits.

Anti-inflammatory treatment: Medications targeting the inflammatory cascade, such as corticosteroids (e.g., dexamethasone) and interleukin-6 inhibitors (e.g., tocilizumab), may reduce the risk of myocardial injury and subsequent infarction.

Early detection and management: Early identification of cardiovascular involvement in COVID-19 patients is critical. Biomarkers such as troponin, NT-pro BNP, and D-dimmer, along with imaging studies, can help in risk stratification and timely intervention[3].

Future directions

The COVID-19 pandemic has fundamentally reshaped our understanding of infectious diseases and their interplay with cardiovascular health, particularly with respect to acute coronary syndromes (ACS) and heart attacks. While the immediate link between SARS-CoV-2 infection and cardiovascular complications has been established, the long-term implications of COVID-19 on cardiac health remain an evolving field of study[4].

Conclusion

The relationship between COVID-19 and myocardial infarction highlights the systemic nature of SARS-CoV-2 infection and its impact on cardiovascular health. Understanding the mechanisms underlying this association is crucial for developing effective prevention and treatment strategies. Long-term studies are needed to elucidate the chronic cardiovascular consequences of COVID-19 and to guide clinical management in the post-pandemic era.

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Article: 5

Cardiac Manifestation of Corona Virus

Souvik Pal

Senior Officer, Macleod Pharmaceutical ltd.

souvikpal6038@gmail.com

Abstract:

Corona viruses are a large family of single positive-sense single-stranded, enveloped RNA viruses that can infect many animal species and humans. Human corona viruses can be divided based on their pathogen city. Severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2) infections were first identified in December 2019 and termed Coronavirus disease 2019 (COVID-19). By March 2020, it was declared a global pandemic by the World Health Organization (WHO). Although primarily a respiratory disease, COVID-19 has been associated with many cardiac complications. Cardiac injury is recognized as one of the most frequent complications of the disease. Long-term cardiac complications following COVID-19 include ischemic heart disease, heart failure, arrhythmias, and myocarditis. Studies have consistently shown that underlying cardiovascular disease in patients with COVID-19 and the development of acute cardiac injury due to COVID-19 illness is associated with significantly worse outcomes. This activity reviews the clinical manifestations of cardiac complications related to COVID-19 infections and outlines their recommended evaluation and treatment.

Keywords: SARS-CoV-2, Pathogenicity, COVID-19, Ischemic

Introduction

Corona viruses are a large family of single-stranded positive-sense, enveloped RNA viruses that can infect many animal species. Human corona viruses can be divided based on their pathogenicity. The types with high pathogenicity include SARS-CoV, MERS-CoV, and the current novel SARS-CoV2 viruses. Severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2) infections were first identified in December 2019 and termed Coronavirus disease 2019 (COVID-19). By March 2020, it was declared a global pandemic by the World Health Organization (WHO).

Although primarily a respiratory disease, COVID-19 has been associated with many cardiac complications. Cardiac injury is recognized as one of the most frequent complications of the disease. Long-term cardiac complications following COVID-19 include ischemic heart disease, heart failure, arrhythmias, and myocarditis.

Studies have consistently shown that underlying cardiovascular disease in patients with COVID-19 and/or the development of acute cardiac injury due to COVID-19 illness is associated with significantly worse outcomes.

Etiology

Corona viruses comprise a family within the order *Nidovirales*. They consist of four genera, Alpha coronavirus, Beta coronavirus, Gamma coronavirus and Delta coronavirus.

Corona viruses are common in birds and mammals. They are ubiquitous and are among the most common causes of community-acquired upper respiratory tract infections. They mainly cause mild, self-limiting illnesses and are transmitted by respiratory aerosol/droplets. The virus responsible for the COVID-19 outbreak is called SARS-CoV-2, which is a Beta coronavirus and has 76.4% amino acid sequence homology in its spike protein compared to the SARS-CoV virus that caused the 2003 outbreak in China.

Morphologically it is a positive-sense single-stranded RNA virus with an approximately 30 kb genome that transcribes four structural proteins (surface (S), envelope (E), membrane (M), and nucleocapsid (N)) and some accessory proteins. Nonstructural proteins involved in replication and transcription are also present. The spike proteins facilitate entry into host cells by binding to angiotensin-converting enzyme 2 (ACE-2) receptors.

Although the respiratory system is the main site of entry and infection in humans, the presence of ACE-2 receptors in other organ systems may lead to direct injury at these sites. In humans, the ACE-2 receptors are abundantly present in the lung type II alveolar cells, entrecotes of the gastrointestinal tract, endothelial cells, smooth muscle cells, cortical neurons, and glial cells. As the virus replicates, it can enter cardiac cells and cause direct injury, which results in cardiac manifestations such as myocarditis. However, clinical data has not consistently supported direct cardiomyocyte death secondary to cardiomyocyte infection[1].

Epidemiology

Cardiovascular complications, as evidenced by acute cardiac injury, new-onset systolic heart failure, pericardial effusion, and acute myocarditis, have been reported in hospitalized patients since the beginning of the pandemic. A meta-analysis from 2021 reported a cardiac injury prevalence of 22% among all hospitalized patients with COVID-19. In patients with severe COVID-19 illness, they reported that 28% had documented cardiac injury. In patients older than 60 years, the prevalence of COVID-19-associated cardiac injury rose to 30%.

Common cardiac complications of COVID-19 include acute coronary syndrome and arrhythmias. These tend to occur in older patients with known cardiovascular risk factors such as hypertension, diabetes mellitus, and coronary artery disease. Myocardial infarction has been noted in up to 1.3% of all COVID-19 patients. The prevalence of myocardial infarctions in patients who died from COVID-19 illness rises to 4.9%. Arrhythmias have been reported in up to 10.4% of patients affected with moderate to severe COVID-19 illness. The most commonly noted arrhythmia in these patients is atrial fibrillation[2].

Pathophysiology

Multiple mechanisms have been suggested as the cause of cardiac damage; however, true pathogenic mechanisms have not been clearly elucidated thus far. Possible causes include cytokine-mediated damage, micro vascular thrombi, and/or direct cardiomyocyte

injury due to viral invasion of the myocardium.

Catecholamine-induced micro vascular dysfunction due to the intense inflammatory state caused by COVID-19 is thought to cause COVID-19-associated Takotsubo cardiomyopathy.

Autopsy evaluations have revealed small vessel endothelial damage and vacuities (neutrophilic) as the cause of multisystem inflammatory syndrome after a COVID-19 infection in one case report. They reported that the "cardiac myocytes did not seem to be the target of the inflammatory process." Other post-mortem studies revealed similar findings with endotheliitis in several organs and evidence of inflammatory cell death. These studies also noted the presence of viral bodies in affected organs and host inflammatory response leading to endothelial damage.

Pre-existing cardiovascular disease is associated with chronic activation of inflammatory pathways, which may act synergistically with COVID-19-induced inflammation. Pre-existing inflammation and superimposed endothelial dysfunction can accelerate coronary plaque instability, which has been proposed as a cause of cardiovascular complications in patients with COVID-19 illness[3].

Evaluation

Cardiac biomarkers such as troponin and B-type natriuretic peptide (BNP) are usually elevated in these patients. They may identify those with cardiac injury due to COVID-19 critical illness in the absence of symptoms. An electrocardiogram and echocardiography are warranted in evaluating patients suspected of having a myocardial injury to differentiate those with acute myocardial infarction due to atherothrombotic disease from those with demand ischemia. Specific cardiac testing is seldom warranted.

In patients with post-COVID-19 syndrome and persistent symptoms, however, cardiac testing may help identify those needing cardiopulmonary rehabilitation. Results of cardiopulmonary testing in patients seen in a post-COVID-19 care clinic due to persistent symptoms of fatigue, myalgias, and dyspnea reported decreased peak oxygen uptake in these patients. Those with persistent symptoms were noted to have mean peak oxygen uptake 30% below predicted, with less than 10% of these patients having a normal value.

In acutely ill patients, elevated cardiac markers, especially troponin levels, must be taken in context to prevent erroneous diagnoses. A retrospective study from 2022 stated that of the 2152 patients studied, 88% had high-sensitivity troponin-T testing, with 57% having a value above the 99th percentile. Further analysis of those with elevated levels revealed that only 47% of the patients had a primary cardiac etiology for the elevated troponin level[4, 5].

Conclusion

COVID-19 has been associated with many cardiac complications. Cardiac injury is recognized as one of the most frequent complications of the disease. Long-term cardiac complications following COVID-19 include ischemic heart disease, heart failure, arrhythmias, and myocarditis. Treatment options include supportive care, symptom control, and guideline therapy.

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The First Convocation 2024

About RKDF University Ranchi

It was in the year 2018, **Dr. Sunil Kapoor**, the founder decided to fulfill his dream of establishing an institute for quality education to the people and the region & beyond initiated **"Ayushmati Education and Social Society"** trust in an attempt to make the holy city of Ranchi, a recognized destination for knowledge seekers from different spheres of life and strive to become one of the best Universities in Jharkhand. This is what led to the foundation of a milestone at the karmabhoomi of the versatile and sagacious **Bhagwan Birsa Munda**.

RKDF group has been actively involved with social causes since its very inception and has drawn appreciation from one and all for its works in various facets of societal paradigms. The Group started its journey in 1994 by establishing 1st private engineering college at Bhopal, Madhya Pradesh. Now the group has 162 institutions & 6 universities (Five in Madhya Pradesh and one in Jharkhand). The six universities and social society established by Ayushmati Education are **RKDF University, Bhopal** (2011), **Sri Satya Sai University of Technology & Medical Science, Sehore** (2014), **Sarvepalli Radhakrishna University, Bhopal** (2015), **Dr. A.P.J. Abdul Kalam University, Indore** (2016), **Bhabha University, Bhopal** (2018), **RKDF University, Ranchi** (2018). RKDF Ranchi is a highly prestigious government recoginized university established under the Jharkhand Govt. Act & registered under UGC 2f 1956. RKDF University, Ranchi is a recoginized member of AIU (Association of Indian Universities) and has publication house, named IJHESM (International Journal of Humanities, Engineering, and Science & Management) with an impact factor of SJIF-5.81.

Next theme for fifth edition

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