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REVIEW ARTICLE

A Systematic Review on the Incidence of Major Coronavirus-associated Cardiovascular Diseases and Therapeutic Approaches during the COVID-19 Pandemic

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KEYWORDS

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ABSTRACT: During the COVID-19 pandemic, people with cardiovascular disease were at increased risk of serious complications and death from the virus. The SARS-CoV-2 virus can directly affect the heart and blood vessels, exacerbating conditions such as inflammation and blood clotting. In addition, pandemic-related restrictions and lifestyle changes have increased risk factors for heart disease. This review aims to examine the impact of COVID-19 on patients with cardiovascular disease. Numerous studies have shown that individuals with cardiovascular disease are more susceptible to the virus, with cardiac problems being particularly prevalent in this patient population. In addition to the increased risk of cardiovascular complications, these patients also face a higher risk of mortality. For this purpose, scientific articles related to COVID-19, coronavirus, SARS-CoV-2, and cardiovascular diseases were collected and analyzed from reputable scientific sources, including PubMed, Scopus, Web of Science, and other scientific databases. The literature review revealed that coronavirus and COVID-19 caused various diseases and complications during the COVID-19 pandemic, including vascular complications, arrhythmias, pericarditis, myocarditis, and general cardiac involvement. Based on these findings, this study underscores the need for increased attention to patients with cardiovascular disease during the COVID-19 pandemic and highlights the need for specific and effective treatments for these patients based on the latest scientific evidence.

INTRODUCTION

Coronaviruses are a large and diverse family of viruses that can infect a wide range of organisms, including mammals and birds [1]. The SARS-CoV-2 virus is a single-stranded RNA virus that possesses key spike-like proteins as well as a membrane and nucleocapsid that allow it to enter human cells [1]. This virus continuously undergoes mutations that can alter its transmission characteristics, disease severity, and ability to evade the immune response [2]. New

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variants, such as alpha, delta, and omicron, with specific mutations in the spike protein, are more transmissible and pose challenges for vaccines and treatments [2]. Bats are recognized as the primary natural host for these viruses and exhibit high genetic diversity [2]. The novel coronavirus (SARS-CoV-2), the causative agent of COVID-19, was first identified in late December 2019 in Wuhan, China. This virus primarily affects the respiratory tract, particularly the lungs, resulting in the infectious disease COVID-19 [1,2]. The pathophysiology of cardiovascular diseases associated with SARS-CoV-2 infection is multifaceted, involving both direct and indirect mechanisms. The virus primarily enters cells through the ACE2 receptor, which is abundantly present in cardiac and vascular tissues. This interaction may result in direct harm to the heart, such as myocarditis (inflammation of the myocardial tissue), compromised left ventricular function, and in some cases, heart failure [3]. Additionally, the systemic inflammation induced by the viral infection contributes to endothelial dysfunction, which can damage the blood vessel walls, elevate blood pressure, and promote conditions such as atherosclerosis, further increasing the risk of cardiovascular events like heart attacks or strokes [3]. SARS-CoV-2 can impair the autonomic nervous system, disrupting blood pressure and heart rate regulation. These disruptions, along with inflammation and clotting abnormalities, increase the risk of thromboembolic events [4]. In patients with conditions like hypertension, diabetes, or cardiovascular disease, these complications are often more severe. The long-term effects of COVID-19, or "Long COVID," may include persistent cardiovascular symptoms, such as shortness of breath, fatigue, and ongoing cardiac dysfunction [4]. In sum, the cardiovascular impact of SARS-CoV-2 infection involves a complex interplay of direct viral effects, inflammatory responses, endothelial damage, and dysregulated coagulation, all of which contribute to the development of both acute and chronic cardiovascular complications. Understanding these mechanisms is crucial for effective management and treatment of affected individuals [4].

Human coronaviruses are transmitted primarily by respiratory droplets from coughing or sneezing or by contact with contaminated surfaces [4]. The most common symptoms of COVID-19 include cough, difficulty breathing, fever over 100°F (38°C), chills, fatigue, muscle aches, loss of taste or smell, and gastrointestinal symptoms such as heartburn, diarrhea, or vomiting [5]. To prevent coronavirus infection, wash your hands with soap and water or disinfectant for at least 20 seconds, and cover your mouth and nose with a tissue or your elbow when you cough or sneeze. Avoid close contact with sick people and crowded places. If symptoms develop, stay home to avoid spreading the virus to others [6]. Prevention is the best approach; therefore, it is necessary to receive the COVID-19 vaccine if eligible. The vaccine can reduce the risk of long-term health problems caused by COVID-19 infection [7]. In addition to lung damage, coronavirus can cause severe damage to other organs such as the brain, heart, kidneys, and pancreas. These injuries can lead to chronic problems such as heart failure, stroke, kidney inflammation, and neurological changes that may persist even after recovery from COVID-19 [8]. Vaccination against COVID-19, especially in patients with heart conditions or those who have previously been infected with the virus, can reduce the risk of cardiac complications. Vaccines help protect against cardiovascular issues by reducing the severity of the illness and preventing long-term heart-related complications. Although, in rare cases, vaccines may lead to temporary myocarditis, the benefits of preventing serious complications and death due to COVID-19 far outweigh these risks [9]. This review specifically examines cardiovascular disease caused by SARS-CoV-2 infection and its effects on the cardiovascular system, including heart failure, arrhythmias, and myocardial infarction [10]. In addition, the virus may increase the risk of cardiovascular disease through mechanisms such as inflammation, blood clotting disorders, and changes in body oxygen levels [10, 11]. The aim of this study is to examine the impact of COVID-19 on patients with cardiovascular diseases and its complications, such as inflammation, blood clotting, arrhythmias, pericarditis, and myocarditis. The study emphasizes the importance of paying special attention to these patients during the

pandemic and the need for specific and effective treatments.

MATERIALS AND METHODS

Inclusion and Exclusion Criteria

The aim of this review study was to investigate the incidence of cardiovascular disease during the COVID-19 pandemic. Specific criteria were applied to select relevant articles, including publication in reputable scientific databases such as SID, PubMed, Scopus, Google Scholar, and Web of Science. The selected articles needed to address the impact of cardiovascular disease in patients with COVID-19 or the relationship between the SARS-CoV-2 virus and heart disease. They also had to include keywords such as COVID-19, coronavirus, SARS-CoV-2, cardiovascular diseases, myocarditis, acute coronary syndromes, heart failure, and ischemic heart disease, and be published in either English or Persian. Articles in other languages were excluded. The review considered articles published from the start of the COVID-19 pandemic (December 2019) until the date of the search. Articles that were not directly or indirectly related to the topic, such as non-research articles, those unrelated to cardiovascular disease or COVID-19, or low-quality studies with poor research design or non-generalizable results, were excluded. Additionally, duplicate articles or those published before the start of the COVID-19 pandemic were excluded from the final compilation. However, the article does not clearly address the evaluation of the quality of the studies reviewed. Providing further explanation on how the quality of studies was assessed and the criteria used to determine high or low-quality articles would enhance the paper’s credibility and strengthen its methodology. This would also reassure the reader that the studies selected are appropriate and reliable.

RESULTS

The literature review indicates that the coronavirus and COVID-19 pandemic have led to various diseases and complications, including vascular complications, arrhythmias, pericarditis, myocarditis, acute coronary syndrome, and cardiac involvement. COVID-19, caused by the SARS-CoV-2 virus, can have severe cardiovascular effects. The virus binds to heart and lung cells through ACE2 receptors, leading to myocardial inflammation, arrhythmias, and an increased risk of heart failure. In addition, the stimulation of the immune system and resulting cytokine storm can further damage the heart muscle and disrupt the body's coagulation system, increasing the risk of blood clots, heart attack or stroke. Individuals with pre-existing heart disease are particularly at risk. The results related to these findings are detailed in Tables 1 to 5.

Acute Coronary Syndrome

Acute coronary syndrome (ACS) encompasses a group of medical conditions resulting from a sudden and severe reduction in blood flow to the heart muscle, primarily due to blockage of the coronary arteries. ACS includes conditions such as unstable angina and myocardial infarction (MI). Detailed information on the trends and treatments of acute coronary syndrome is provided in Table 1.

Table 1. Overview of Acute Coronary Syndrome Caused by COVID-19: Mechanisms, Risks, and Treatments.

Result	Explanation	Ref
Probability of acute coronary syndrome in COVID-19	Like other infectious diseases such as SARS and influenza, COVID-19 can lead to acute coronary syndrome. However, the incidence of acute coronary syndrome in COVID-19 patients remains uncertain.	[12]
Mechanisms underlying COVID-19-induced acute coronary syndrome	- Atherosclerotic plaque rupture - Coronary artery spasm - Microthrombosis induced by systemic or elevated cytokines	
The role of macrophages in plaque destruction	Active inflammation causes macrophages to degrade collagen, the primary component of the inner lining of plaques, leading to plaque rupture.	
The role of tissue factor in thrombus formation	Activated macrophages secrete tissue factor, a potent procoagulant that contributes to thrombus formation after plaque rupture.	
Damage to the endothelium or blood vessels	Direct endothelial or vascular damage induced by SARS-CoV-2 infection may increase the risk of thrombosis and acute coronary syndrome.	
Treatment of coronary syndrome	Treatment includes the use of anticoagulants and painkillers, as well as cardiac procedures such as angioplasty and stenting. In severe cases, coronary bypass surgery may be required.	

Pericarditis

Acute pericarditis in developed countries often occurs without a clear cause and can be attributed to viral infections or severe inflammatory responses, such as the cytokine storm associated with COVID-19. Anti-inflammatory treatment, particularly the use of colchicine, can effectively reduce inflammation and mortality risk in these patients. Table 2 provides comprehensive information on the trends and management of pericarditis.

Table 2. Overview of Covid-19-induced Pericarditis: Mechanisms, Risks and Treatments.

Result	Explanation	Ref
Unknown cause of pericarditis in developed countries	In developed countries, most cases of pericarditis are idiopathic, but are often associated with viral agents such as parvovirus, coxsackievirus, and echovirus.	[13-15]
Acute pericarditis and the inflammatory response	Acute pericarditis is caused by an inflammatory response to injury to the mesothelial cells of the pericardial layers, initiated by the release of cellular debris.	
Role of NLRP3 receptor in pericardial inflammation	The NLRP3 protein receptor is activated in response to viral infection, resulting in amplification of local and systemic inflammation.	
SARS-Covid-19 and the cytokine storm	The SARS-CoV-2 virus induces overproduction of pro-inflammatory cytokines, culminating in a cytokine storm that can lead to cardiovascular complications.	
Report of pericarditis in patients with covid-19	In one study, a COVID-19 patient was diagnosed with pericardial effusion and thickened pericardium containing mesothelial cells and active histologic sites.	
Electrocardiographic changes in acute pericarditis	In acute pericarditis, electrocardiographic changes occur in four distinct phases: ST elevation, PR segment depression, normalization, and T wave inversion.	
Anti-inflammatory treatment for acute pericarditis	The primary treatment of acute pericarditis is the use of non-steroidal anti-inflammatory drugs (NSAIDs) or colchicine, which inhibit the activity of the NLRP3 inflammasome.	
The effect of colchicine in reducing mortality	Observational studies have suggested that colchicine may reduce mortality in COVID-19 patients.	

Ischemic heart diseases (myocarditis)

Myocardial damage in COVID-19 patients results from direct viral effects, inflammation induced by cytokine storms, and hemodynamic and electrolyte disturbances. The ACE2 receptor in cardiac cells serves as an entry point for the virus, potentially leading to myocardial damage and various cardiac disorders. For comprehensive information on trends and treatments for ischemic heart disease (myocarditis), see Table 3.

Table 3. Overview of covid-19-induced Myocarditis: Mechanisms, Risks and Treatments.

Result	Explanation	Ref
Ischemic cardiovascular disease	Ischemic cardiovascular disease, including myocardial infarction (heart attack), is the leading cause of death in Iran and many other countries.	[16-20]
Risk factors for heart disease	Hypertension, dyslipidemia, diabetes, obesity, and metabolic syndrome are modifiable risk factors for myocardial infarction.	
Definition of myocarditis and causes	Myocarditis is an inflammation of the heart muscle, typically caused by viral, bacterial, or parasitic infections.	
Role of SARS-CoV-2 in the development of myocarditis	SARS-CoV-2 can cause acute myocardial injury and myocarditis characterized by elevated cardiac biomarkers and electrocardiographic abnormalities.	
Myocardial damage in patients with covid-19	Approximately 7.9% to 9.9% of COVID-19 patients develop acute myocardial injury, typically characterized by elevated cardiac troponin levels.	
Poor prognosis of myocardial damage in covid-19	The presence of myocardial damage in COVID-19 patients is associated with poorer prognosis and higher mortality.	
Mechanisms of myocardial damage in covid-19	Myocardial injury may result from direct viral invasion of the myocardium, cytokine storm, microangiopathy, or an imbalance between oxygen supply and demand.	
The role of the ACE2 receptor in myocardial injury	SARS-CoV-2 infiltrates myocytes and vascular endothelial cells via the ACE2 receptor, resulting in myocardial damage.	
Other potential mechanisms of cardiac injury	Cytokine storm and intracellular hypoxia-induced eclampsia are additional mechanisms contributing to cardiac injury in COVID-19 patients.	

Arrhythmia

Common arrhythmias in COVID-19 patients include ventricular arrhythmias (tachycardia and ventricular fibrillation) and atrial arrhythmias (atrial fibrillation). These arrhythmias can result from electrolyte imbalances, hypoxia, and cytokine storms. Arrhythmias in COVID-19 patients are typically caused by several pathophysiological factors, including direct viral myocardial damage, electrolyte disturbances induced by the virus or treatments, hypoxia (lack of oxygen), and cytokine storms, which collectively alter the electrical function of the heart and increase the risk of arrhythmias. Effective management of arrhythmias in these patients requires careful consideration of the patient's hemodynamic status, including control of fever, management of hypoxia with oxygen therapy, and monitoring of medications. It is also important to note that antiviral and antibiotic treatments for COVID-19 may increase the risk of arrhythmias. Therefore, accurate and timely management is critical. Table 4 provides comprehensive information on arrhythmia trends and management.

Table 4. Overview of arrhythmias caused by Covid-19: Mechanisms, Risks, and Treatments

Component	Detail	Ref
Common arrhythmias in covid-19	-Ventricular Arrhythmias: Ventricular Tachycardia, Ventricular Fibrillation	[21-25]
	-Atrial Arrhythmias: Atrial Fibrillation	
Pathophysiological mechanisms of arrhythmias	-Electrolyte disorders: Abnormalities in sodium and potassium levels in COVID-19 patients may increase the risk of arrhythmias.	
	-Hypoxia: Lack of oxygen in the blood disrupts the function of the junctional areas of the heart, leading to atrial fibrillation.	
	-Cytokine storm: Elevated levels of interleukins and TNF-α can lead to fatal arrhythmias.	
Effects of drugs on arrhythmias	-Medications such as chloroquine and hydroxychloroquine can prolong the QT interval and increase the risk of ventricular arrhythmias (torsade de pointes).	
	-In addition, antivirals and antibiotics can indirectly cause arrhythmias.	
Management of arrhythmias	-In patients with stable hemodynamic status, reducing the heart rate is unnecessary.	
	-Supportive care, including oxygen therapy and fever control, may reduce the risk of atrial fibrillation.	
	-Anticoagulant therapy is recommended in patients with coagulation disorders.	
Therapeutic drugs	-Beta-blockers are recommended for patients who are hemodynamically stable and do not have heart failure.	
	-Amiodarone is used to treat acute heart failure or malignant ventricular arrhythmias.	
Arrhythmia risk prediction	-Patients with elevated troponin T levels are at increased risk for malignant arrhythmias.	
	-Individuals with inherited arrhythmia syndromes, such as long QT syndrome and Brugada syndrome, are predisposed to arrhythmias.	

Vascular complications

Coagulation disorders associated with COVID-19, especially in severe cases, increase the risk of thrombosis and pulmonary venous embolism. These patients often have molecular changes similar to pulmonary vascular disease, increased coagulability, and microthrombosis. Venous thrombosis is highly prevalent in COVID-19 patients, necessitating the use of diagnostic techniques such as CT angiography to identify these complications. Table 5 provides comprehensive information on trends and management of vascular complications.

Table 5. Examination of Vascular Complications Induced by COVID-19: Mechanisms, Risks, and Treatments

Result	Explanation	Ref
Coagulation disorders associated with covid-19	Emerging evidence suggests that COVID-19 patients have coagulation abnormalities that contribute to an increased risk of venous and arterial thromboembolism.	[26-29]
Pulmonary venous embolism in severe covid-19 patients	Pulmonary venous embolism is particularly common in patients with severe COVID-19.	
Molecular changes similar to pulmonary vascular disease	The molecular changes induced by COVID-19 are similar to those observed in pulmonary vascular disease, potentially leading to microthrombosis and pulmonary hemodynamic dysfunction.	
Increased risk of coagulopathy and peripheral vein thrombosis	In COVID-19 patients, the increased risk of coagulability leads to peripheral vein thrombosis and subsequent pulmonary embolism.	
Prevalence of venous thrombosis in patients with covid-19	Numerous studies have shown that 41% to 91% of COVID-19 patients experience venous thrombosis.	
Diagnostic methods	Pulmonary CT angiography is recommended as an appropriate diagnostic modality for the detection of pulmonary embolism in patients with severe respiratory symptoms or a positive D-dimer marker.	
Pulmonary artery thrombosis and wedge-shaped areas	In patients with severe COVID-19 infection, pulmonary artery thrombosis and wedge-shaped areas due to decreased blood supply have been observed.	
Presence of microthrombosis	In approximately half of the patients, CT scans did not show clear thrombi, indicating the possible presence of microthrombi.	

DISCUSSION

Cardiovascular disease has a bidirectional relationship with COVID-19 [30]. Chronic heart disease may increase the risk of severe COVID-19, while cardiovascular complications contribute significantly to the pathophysiology of the disease. Although there is little evidence of a direct viral effect on the heart, mild to moderate myocardial damage is a recognized adverse prognostic feature in severe COVID-19 cases [31]. Key factors such as systemic inflammation, microvascular thrombosis, hypoxia, and increased susceptibility in individuals with pre-existing heart disease are primary causes of this myocardial damage [32]. In addition, heart failure may exacerbate pulmonary problems in these patients. The incidence of morbidity is higher in COVID-19 patients with a history of cardiovascular disease. Reports from the National Health Commission of China indicate that some COVID-19 patients initially sought medical attention for cardiovascular symptoms, such as chest tightness and heart pain, rather than the typical respiratory symptoms of fever or cough [33]. Given the prevalence and significance of cardiovascular complications associated with COVID-19, medical personnel, particularly nurses who are in close contact with COVID-19 patients, should be well versed in recognizing these complications to improve patient outcomes through early diagnosis and intervention [34]. Health behaviors, diet, lifestyle, and access to healthcare in different countries can significantly impact the prevalence and outcomes of cardiovascular diseases related to COVID-19. In regions with limited access to healthcare or where unhealthy lifestyles are common, the likelihood of cardiovascular complications in COVID-19 patients is higher. Therefore, addressing these differences in healthcare policies and treatment strategies is crucial [35, 36]. These patients, due to their multiple risk factors, require specialized preventive strategies, including closer monitoring, management of

cardiovascular medications, stress reduction, and adherence to an appropriate diet. Additionally, vaccination and appropriate treatments are crucial to prevent severe COVID-19 complications. Therefore, the development of tailored care plans for these patients is essential to mitigate risks and improve therapeutic outcome [37].Future research should focus on identifying biological markers that can predict cardiovascular risks in COVID-19 patients. Long-term studies are also needed to assess the lasting effects of the infection on heart health and to evaluate the impact of different treatments on cardiovascular complications. Further investigation into the link between cardiovascular diseases and pulmonary complications is crucial. Additionally, the development of multidisciplinary treatment approaches and the exploration of genetic differences in susceptibility to COVID-19-related heart complications could improve prevention and treatment strategies.

CONCLUSIONS

Given the complex interaction between COVID-19 and cardiovascular complications, future research should focus on identifying biomarkers that can predict cardiovascular risks in patients with COVID-19. Additionally, long-term follow-up studies are crucial for understanding the lasting effects of viral infection on heart health. Research indicates a bidirectional relationship between cardiovascular diseases and COVID-19. Chronic heart conditions can increase the risk of severe COVID-19, while cardiac complications arising from the virus significantly affect the disease's prognosis. Mild to moderate myocardial damage is commonly seen in patients with cardiovascular diseases, worsened by factors such as systemic inflammation, microvascular thrombosis, and hypoxia. Furthermore, heart failure can exacerbate pulmonary complications in these patients. Evidence shows that patients with a history of cardiovascular disease have higher morbidity rates and may present with cardiac symptoms early on. Therefore, it is essential for healthcare providers, especially nurses, to be aware of the cardiac symptoms and complications associated with COVID-19, enabling early diagnosis and timely intervention, which can improve patient outcomes. For clinicians, adopting a multidisciplinary approach involving cardiologists, pulmonologists, and infectious disease specialists is critical to ensuring optimal care and minimizing complications.

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Conflict of interest

None.

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