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Cardiac Manifestations of Coronavirus (COVID-19)-An Updated Review for Healthcare Professionals

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Abstract

Coronavirus disease 2019 (COVID-19), caused by the SARS-CoV-2 virus, primarily affects the respiratory system, but also presents significant cardiac manifestations. While acute respiratory symptoms are typical, COVID-19 is linked to numerous cardiovascular complications such as ischemic heart disease, arrhythmias, heart failure, and myocarditis. These complications, particularly in individuals with preexisting cardiovascular conditions, are associated with poorer outcomes.This review aims to comprehensively explore the cardiac manifestations of COVID-19, evaluate their pathophysiology, and assess current treatment strategies for managing these complications.This updated review consolidates findings from recent studies on the cardiovascular impact of COVID-19, drawing from clinical, epidemiological, and pathophysiological data. Literature sources were reviewed from various academic and clinical studies focusing on the prevalence, diagnosis, and management of cardiac issues associated with COVID-19.COVID-19-related cardiac complications, including acute cardiac injury, myocarditis, arrhythmias, and heart failure, have been reported across multiple studies. The prevalence of cardiac injury among hospitalized COVID-19 patients ranges from 22% to 30%, with a notable increase in those over 60 years old. Acute coronary syndrome and arrhythmias are particularly common in older patients with preexisting cardiovascular risk factors. Post-mortem studies reveal myocardial necrosis, edema, and endothelial damage, suggesting a multifactorial pathophysiology involving inflammation, thrombosis, and viral invasion.COVID-19 presents a complex challenge for healthcare professionals due to its significant cardiovascular impact, particularly in patients with pre-existing heart conditions. Cardiac complications must be closely monitored, and appropriate management strategies are critical in improving patient outcomes. Future studies should focus on refining diagnostic methods and exploring long-term cardiac health in post-COVID-19 patients.

Keywords: *COVID-19, Cardiac Manifestations, Myocarditis, Arrhythmias, Heart Failure, Acute Cardiac Injury, Pathophysiology.*

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Introduction

Coronaviruses belong to a large family of single-stranded positive-sense, enveloped RNA viruses capable of infecting various animal species. Human coronaviruses can be categorized based on their pathogenicity, with highly pathogenic types including SARS-CoV, MERS-CoV, and the currently circulating novel SARS-CoV-2 virus [1]. The onset of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections occurred in December 2019, subsequently being termed as coronavirus disease 2019 (COVID-19). By March 2020, the World Health Organization (WHO) officially declared it a global pandemic [2]. Although COVID-19 is primarily a respiratory illness, it is linked to numerous cardiac complications, with cardiac injury being one of the most prevalent. Long-term cardiac issues arising from COVID-19 encompass ischemic heart disease, heart failure, arrhythmias, and myocarditis [3]. Extensive studies have consistently demonstrated that underlying cardiovascular disease in COVID-19 patients, along with the development of acute cardiac injury due to the infection, significantly correlates with worse clinical outcomes [4]. This activity aims to review the clinical manifestations of cardiac complications associated with COVID-19, alongside their evaluation and treatment protocols.

Etiology

Coronaviruses are classified within the Nidovirales order [5], consisting of four genera: Alphacoronavirus, Betacoronavirus, Bammacoronavirus, and Delta coronavirus. These viruses are prevalent in birds and mammals, being ubiquitous and among the most common causes of community-acquired upper respiratory infections [5]. In most cases, they induce mild, self-limiting diseases transmitted via respiratory aerosols or droplets. The virus responsible for the COVID-19 outbreak is SARS-CoV-2, a betacoronavirus with 76.4% amino acid sequence homology in its spike protein compared to the SARS-CoV virus from the 2003 outbreak in China [5]. Morphologically, SARS-CoV-2 is a positive-sense single-stranded RNA virus with an approximate genome length of 30 kb, encoding four structural proteins (surface (S), envelope (E), membrane (M), and nucleocapsid (N)) as well as several accessory proteins. Nonstructural proteins facilitating replication and transcription are also present. The virus enters host cells by binding its spike proteins to angiotensin-converting enzyme 2 (ACE-2) receptors [5]. While the respiratory system is the primary site of infection, the presence of ACE-2 receptors in other organs may lead to direct injury in these areas. In humans, ACE-2 receptors are abundantly present in lung type II alveolar cells, gastrointestinal enterocytes, endothelial cells, smooth muscle cells, cortical neurons, and glial cells [5]. As the virus replicates, it can infiltrate cardiac cells, leading to direct damage and resultant cardiac issues such as myocarditis [6]. However, clinical evidence has not consistently supported direct cardiomyocyte death due to infection [7]. Alternative mechanisms include systemic inflammation (the cytokine storm), which leads to increased oxygen demand amid persistent hypoxia, coagulopathy, and arrhythmias [6]. This is particularly concerning for individuals with preexisting cardiovascular conditions, who have limited physiological reserve to handle this stress. Clinical data indicates a notable correlation between cardiovascular diseases (e.g., hypertension and coronary artery disease) and the increased severity of COVID-19 illness [8]. Although the precise pathogenic causes of this association remain unclear, potential explanations include reduced physiological reserve, enhanced inflammatory responses, and SARS-CoV-2-induced endothelial dysfunction [9]. Numerous studies have revealed that, among hospitalized COVID-19 patients, preexisting cardiovascular disease and cardiac injury are significant predictors of higher mortality [9]. According to the Centers for Disease Control and Prevention (CDC), cardiovascular conditions such as heart failure, coronary artery disease, and cardiomyopathies are linked to an elevated risk of severe COVID-19 and its poor outcomes, with "conclusive data" supporting this connection. A large case series involving 5700 COVID-19 patients across 12 hospitals in New York found that the prevalence of hypertension, diabetes, and coronary artery disease was 57%, 34%, and 11%, respectively [10]. A meta-analysis from 2020 reported that patients with chronic heart disease are more susceptible to acute cardiovascular events when infected with SARS-CoV-2 [11]. Age is another significant risk factor for cardiac injury following SARS-CoV-2 infection. A meta-analysis of 21 studies (encompassing over 6000 patients) found a two-fold higher incidence of cardiac injury in patients over 60 years old compared to younger cohorts hospitalized for COVID-19 infection [12].

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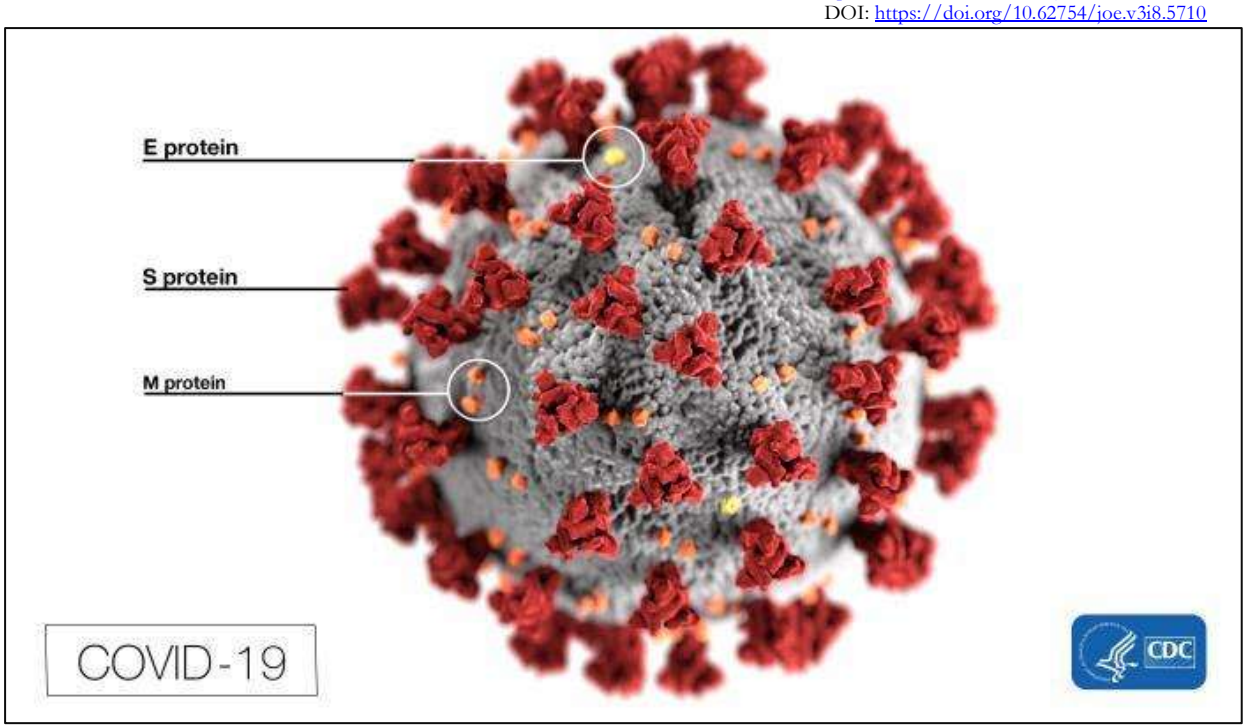

Figure 1: Covid-19 Virus.

Epidemiology

Cardiovascular complications, such as acute cardiac injury, new-onset systolic heart failure, pericardial effusion, and acute myocarditis, have been reported in hospitalized COVID-19 patients since the onset of the pandemic. A 2021 meta-analysis indicated a 22% prevalence of cardiac injury among all hospitalized COVID-19 patients [12]. In severe cases, 28% of patients exhibited cardiac injury, while in those over 60 years old, the prevalence rose to 30% [12]. Common cardiac complications associated with COVID-19 include acute coronary syndrome and arrhythmias, which are predominantly observed in older patients with known cardiovascular risk factors like hypertension, diabetes mellitus, and coronary artery disease. Myocardial infarction has been reported in up to 1.3% of all COVID-19 patients, with this figure rising to 4.9% among patients who succumbed to the infection [13]. Arrhythmias are comparatively more frequent, occurring in up to 10.4% of patients with moderate to severe COVID-19 illness, with atrial fibrillation being the most commonly observed arrhythmia. Congestive heart failure is reported in 2.8% of patients with COVID-19, but the incidence increases to 24% among those who die from the infection [13]. Acute myopericarditis is observed more frequently in younger individuals without pre-existing cardiovascular risk factors. However, the incidence of de novo cardiac complications in this group remains low. COVID-19-related acute myocarditis has been reported at a rate of 2 to 4 per 1000 hospitalizations and may occur with or without concurrent pneumonia [7]. Despite the low incidence, the potential for mortality warrants immediate clinical attention when symptoms suggest cardiac injury [3].

Pathophysiology

Numerous mechanisms have been proposed to explain cardiac damage in COVID-19, although the precise pathogenic pathways remain inadequately defined. Potential causes include cytokine-driven injury, microvascular thrombosis, and direct myocardial damage due to viral invasion of cardiac tissue [14]. Catecholamine-induced microvascular dysfunction, stemming from the intense inflammatory response triggered by COVID-19, is thought to underlie the occurrence of COVID-19-associated Takotsubo cardiomyopathy [14]. Autopsy studies have identified small vessel endothelial damage and neutrophilic vasculitis as contributing factors to multisystem inflammatory syndrome following COVID-19 infection in certain cases [15]. These studies indicated that "cardiac myocytes did not appear to be the target of the inflammatory process." Other post-mortem investigations similarly noted endotheliitis in various organs

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and inflammatory cell death [16]. Furthermore, these studies observed viral particles within the affected organs, alongside a host inflammatory response, which resulted in endothelial damage [16]. Patients with pre-existing cardiovascular disease exhibit chronic activation of inflammatory pathways, which may amplify the inflammation induced by COVID-19 [9]. This pre-existing inflammation, coupled with endothelial dysfunction, can destabilize coronary plaques, which has been suggested as a key mechanism for cardiovascular complications in individuals with COVID-19 [9]. Additionally, patients with cardiovascular disease have limited physiological reserves, making them more vulnerable to cardiac damage when exposed to external stressors [9].

Histopathology

Although the precise mechanisms of pathogenesis remain unclear, post-mortem evaluations offer valuable insights into the cardiovascular complications linked to COVID-19. Post-mortem findings have most frequently revealed myocardial necrosis and edema [17]. The study also indicated that chronic changes such as myocardial hypertrophy, coronary artery disease, and fibrosis were prevalent. Notably, cardiac amyloidosis was more commonly observed among those who succumbed to COVID-19, suggesting it may contribute to worse outcomes [17]. A review of thrombotic complications in COVID-19 patients showed a 36.2% prevalence of microvessel thrombosis, 22.2% for pulmonary embolism, and 11.8% for acute myocardial infarction [17]. Microvascular thrombosis is considered a significant factor in the cardiac complications of COVID-19, which explains the lack of an identifiable culprit lesion in over 40% of patients undergoing coronary angiography when acute myocardial infarction is suspected [17].

History and Physical

Patients who experience cardiac complications from acute COVID-19 infection exhibit a wide range of symptoms. While most present with typical COVID-19 symptoms such as cough, fever, and dyspnea, some remain asymptomatic. A small fraction of patients display symptoms indicative of heart disease. Early epidemiologic studies found that the occurrence of cardiac symptoms as the primary presentation of COVID-19 was low, with one study reporting palpitations in 7.3% of patients and chest pain in only 2% [18][19]. The majority of patients presented with cough, fever, myalgias, and headache, which are characteristic symptoms of COVID-19 infection [20]. Critically ill patients require vigilant cardiac monitoring, as cardiac injury in these individuals is often linked to the severity of their illness. As a result, classical symptoms of acute coronary syndrome are rarely observed. A retrospective study from November 2020 revealed that the incidence of myocardial injury was similar between COVID-19 patients and those with acute respiratory distress syndrome (ARDS) due to non-COVID-19 causes [21]. The study further suggested that myocardial injury in critically ill COVID-19 patients correlated with baseline risks, comorbidities, and the multisystem organ dysfunction induced by acute illness. Among patients with severe COVID-19 who required mechanical ventilation, 50% showed signs of myocardial injury based on serum markers. Interestingly, similar high rates of myocardial injury were also observed in non-COVID-19-related critical illnesses. The study concluded that myocardial injury in severe COVID-19 cases was comparable to that in the general ARDS cohort [21]. Patients with arrhythmias, acute coronary syndrome, or myocarditis generally present with typical signs and symptoms associated with these conditions, without any distinct features specific to COVID-19. Acute heart failure in COVID-19 patients has an incidence of approximately 23% [22], with most of these patients exhibiting elevated cardiac biomarkers. A small proportion may experience Takotsubo syndrome [22].

Evaluation

Cardiac biomarkers such as troponin and B-type natriuretic peptide (BNP) are typically elevated in patients with COVID-19, indicating potential cardiac injury, even in the absence of overt symptoms [3]. Electrocardiography and echocardiography are essential diagnostic tools for evaluating suspected myocardial injury, as they aid in distinguishing acute myocardial infarction resulting from atherothrombotic disease from demand ischemia. Specific cardiac testing is generally unnecessary unless clinical indications arise. For individuals with post-COVID-19 syndrome experiencing persistent symptoms, cardiac testing becomes crucial in identifying those who may require cardiopulmonary rehabilitation [23].

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Cardiopulmonary testing conducted on patients attending post-COVID-19 care clinics for fatigue, myalgias, and dyspnea demonstrated a notable decrease in peak oxygen uptake, with these patients exhibiting an average reduction of 30% below predicted values, and fewer than 10% maintaining normal levels [23]. In acutely ill patients, especially those with elevated cardiac markers, caution is advised when interpreting troponin levels to avoid misdiagnosis. A retrospective study in 2022 reviewed 2152 patients and found that 88% underwent high-sensitivity troponin-T testing, with 57% exceeding the 99th percentile. However, further examination revealed that only 47% of these patients had a primary cardiac etiology for their elevated troponin levels [22]. This underscores the necessity for precise diagnostic testing to ascertain the cause of elevated cardiac biomarkers. Notably, patients with a primary cardiac etiology experienced higher all-cause mortality (28%) compared to those with elevated troponin levels without a cardiac origin (16%), while patients with normal troponin levels had a much lower mortality rate of 3.4% [22].

Electrocardiograms are imperative for all critically ill patients presenting with potential myocardial injury due to an underlying infection. Common electrocardiographic findings in COVID-19 patients include tachycardia (mean heart rate 90 ± 19 bpm) and a mean Bazett-corrected QT interval of 449 ± 144 ms [24]. Sinus rhythm predominates in most patients, with atrial fibrillation/flutter occurring in 5.6% of cases. Abnormal intraventricular conduction was noted in 11.8%, with right bundle branch block in 7.8%, left bundle branch block in 1.5%, and nonspecific intraventricular block in 2.5% [24]. In one study, 13.9% of patients had a prior Q-wave myocardial infarction, and approximately 8.4% developed new-onset tachyarrhythmia or block during hospitalization for acute COVID-19 illness [22]. Echocardiography is a key diagnostic tool in patients presenting with electrocardiographic or serum biomarker evidence of cardiac injury. Increased prevalence of significant echocardiographic abnormalities, including left ventricular (LV) wall motion abnormalities, global LV dysfunction, diastolic dysfunction, right ventricular (RV) dysfunction, and pericardial effusions, has been observed in patients with myocardial injury from acute COVID-19 illness [25]. However, elevated cardiac biomarkers do not always correlate with echocardiographic abnormalities. A study revealed that most patients with elevated troponin levels had an LV ejection fraction (EF) greater than 50%, with only 22% exhibiting wall motion abnormalities. RV dysfunction was detected in 17% of patients, and pericardial effusion was found in 6.6% [22]. The clinical significance of obtaining an echocardiogram in patients with elevated cardiac biomarkers is reinforced by findings that myocardial injury is linked to higher mortality when accompanied by echocardiographic abnormalities. A study showed that the presence of RV dysfunction or LV wall motion abnormalities in over 50% of myocardial segments independently predicted mortality [22]. A diagnosis of myocarditis necessitates tissue sampling, as radiographic imaging, such as cardiac magnetic resonance imaging (MRI), can only suggest ongoing myocardial inflammation post-COVID-19 infection. However, autopsy analysis indicates that clinical criteria, including electrocardiograms and cardiac biomarkers, tend to overestimate the incidence of myocarditis. Postmortem cardiac examinations revealed a histologically confirmed myocarditis prevalence of only 0.5% [22].

Treatment / Management

Optimizing cardiac care for COVID-19 patients involves early detection and management of cardiac complications while simultaneously implementing strategies to triage cases and minimize COVID-19 exposure [26]. The optimal management approach for myocardial injury in acute COVID-19 remains undefined. In the absence of specific guidelines, patients with demand ischemia or clinically suspected myocarditis are generally treated with supportive care, hemodynamic management, and symptom control. For those with COVID-19-associated heart failure or LV systolic dysfunction, standard goal-directed therapy for congestive heart failure should be administered. Pharmacologic or surgical interventions for arrhythmias should be considered based on the specific conduction or rhythm abnormalities present. Initially, controversy surrounded the safety of ACE inhibitors (ACEI) and angiotensin receptor blockers (ARB) in COVID-19 patients. The current consensus favors the continued use of these medications [27][28]. The BRACE CORONA trial demonstrated no significant difference in the number of days alive and out of the hospital among patients receiving continuous ACEI/ARB therapy versus those temporarily discontinuing the medications during hospitalization for COVID-19 [29]. Current guidelines from the United States National Institutes of Health recommend continuing ACEI and ARB therapy for patients

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with underlying cardiac indications unless acute circumstances, such as renal failure, necessitate withholding them. A joint statement from the American Heart Association, the Heart Failure Society of America, and the American College of Cardiology asserts that these medications should not be added or removed based solely on a COVID-19 diagnosis [28]. Clinical decisions regarding ACEI and ARB therapy should be made based on the patient's individual hemodynamic and clinical condition. If patients are on these medications for preexisting cardiac or vascular conditions, they should continue as prescribed, even with COVID-19. For patients with post-COVID syndrome and compromised cardiopulmonary function, physical and/or occupational therapy with a graded exercise program is recommended [23]. Supervised exercise within cardiopulmonary rehabilitation programs is preferred during the initial phase of recovery [23].

Differential Diagnosis

The differential diagnosis of acute myocardial injury in patients with COVID-19 infection involves a broad range of potential conditions [22]. Among the most significant are chronic troponin elevation, pulmonary embolism, noncardiogenic shock, sepsis, and renal failure. Chronic troponin elevation may result from persistent cardiac stress or damage unrelated to an acute event, and it can complicate the interpretation of cardiac biomarkers in these patients. Pulmonary embolism, which occurs when a blood clot obstructs a pulmonary artery, can mimic symptoms of myocardial injury, such as chest pain and dyspnea, and should be considered in the differential diagnosis. Noncardiogenic shock, characterized by inadequate tissue perfusion in the absence of primary cardiac dysfunction, can also present with elevated cardiac biomarkers, further complicating the clinical picture. Sepsis, a systemic inflammatory response to infection, can lead to myocardial dysfunction through inflammatory mediators, often resulting in elevated troponin levels without direct cardiac injury. Renal failure, which is common in critically ill patients, can contribute to the elevation of cardiac biomarkers, as impaired renal clearance may lead to the accumulation of troponins. In the context of COVID-19, it is essential for clinicians to evaluate these conditions thoroughly when interpreting elevated cardiac biomarkers, as each can affect the management and prognosis of affected patients. Accurate diagnosis and differentiation between these conditions are crucial for optimizing patient care and ensuring appropriate treatment.

Prognosis

As mentioned earlier, cardiac injury in patients with acute COVID-19 illness is strongly associated with an increased risk of all-cause mortality [12]. Even in patients without a primary cardiac cause for elevated cardiac biomarkers during acute COVID-19 infection, the mortality rate is still significantly higher compared to those without elevated biomarkers (16% versus 3.4%) [22]. The prognostic significance of cardiovascular disease is clearly demonstrated in a cohort of 191 patients, where 30% had hypertension, accounting for 48% of nonsurvivors, whereas 8% had cardiovascular disease, constituting 13% of nonsurvivors [30]. In a report of 44,672 confirmed COVID-19 cases from the Chinese Center for Disease Control and Prevention, the overall case-fatality rate was 2.3% for the entire cohort, though it was significantly higher for patients with hypertension (6%), diabetes (7%), or cardiovascular disease (11%) [31]. Additionally, COVID-19-associated acute myocarditis is often complicated by shock, affecting 38.9% of cases [7]. However, isolated myocarditis tends to have a better prognosis than myocarditis complicated by concurrent pneumonia due to COVID-19 [7].

Complications

The United States Department of Veterans Affairs (VA) database demonstrated a heightened burden of cerebrovascular disorders, arrhythmias, ischemic heart disease, heart failure, and thrombotic disorders following COVID-19 infection [13]. While these complications were observed in individuals not hospitalized for acute COVID-19 infection, they were most pronounced in patients who required intensive care. Atrial fibrillation and heart failure contributed most to this increased burden, occurring in more than 10 additional individuals per 1,000 people [13]. The impact of disease extended beyond the acute phase, as new-onset hypertension and heart failure were present in 2% of patients who were over a year post-acute COVID-19 infection [13]. Additionally, new right-sided heart failure, in the absence of hypertension or left-sided heart failure, was seen in 2.7% of these patients. Acute myocardial injury is also linked to persistent

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symptoms 12 months after the initial COVID-19 infection, contributing to a higher hospital readmission rate [13]. This is likely attributed to ongoing myocardial dysfunction after the initial infection. Short-term follow-up (2 to 3 months post-infection) showed that 78% of patients had abnormalities on cardiac magnetic resonance imaging (CMR). Studies assessing right ventricular (RV) function in patients recovering from severe COVID-19 revealed that 42% had impaired RV longitudinal strain, although overt RV dysfunction was not observed based on tricuspid annular plane systolic excursion (TAPSE) or RV fractional shortening [13].

Patient Education

Patient education plays a crucial role in preventing COVID-19 infection and its associated cardiac complications. The primary strategy to reduce these complications is through vaccination. COVID-19 vaccines have been shown to be highly effective in decreasing hospitalization rates, ICU admissions, and emergency department visits related to the virus [32]. Even though vaccine efficacy is somewhat reduced in elderly individuals and those with multiple comorbidities, they still provide significant protection against severe disease and death [33]. It is important to educate patients, especially the unvaccinated, about the benefits of receiving the vaccine. Evidence indicates that unvaccinated adults are at a significantly higher risk of hospitalization compared to those who have received the vaccine [34]. By informing patients about the safety, effectiveness, and importance of vaccination, healthcare providers can help reduce the spread of COVID-19 and mitigate its impact on vulnerable populations. In addition to vaccination, other preventive measures such as maintaining good hygiene, wearing masks, and practicing social distancing should be emphasized. Empowering patients with knowledge about these measures not only aids in preventing COVID-19 infection but also contributes to overall public health efforts. Ensuring that patients understand the full scope of protection that vaccines offer can help to build trust in vaccination programs and improve compliance, ultimately reducing the strain on healthcare systems and decreasing the incidence of severe COVID-19-related complications.

Enhancing Healthcare Team Outcomes

Cardiac complications associated with COVID-19 illness encompass a diverse range of conditions, which may either be cardiac or noncardiac in nature. Regardless of the underlying cause, these complications are linked to an increased disease burden and elevated mortality rates. While they are most commonly observed in patients with pre-existing cardiac conditions and/or advanced age, such complications can also affect individuals without prior cardiac disease. Recognizing the significance of these conditions in the context of COVID-19 is crucial for ensuring accurate diagnosis and timely intervention. Healthcare professionals, including clinicians and other members of the interprofessional healthcare team, must maintain a high level of vigilance for the potential development of these complications, particularly given that typical symptoms of cardiac disease may not always be evident in these patients. The role of clinical nurses is vital in closely monitoring cardiac rhythms and rates in patients admitted with acute COVID-19 infection to detect these complications early and facilitate appropriate management. Clinical pharmacists are also integral in assisting healthcare providers with the administration of guideline-recommended therapies, particularly when complications such as arrhythmias or heart failure arise. Both nurses and pharmacists must be empowered to promptly notify clinicians of any changes in patient status or other concerns that may emerge. Furthermore, it is important to recognize that patients may experience residual dysfunction for extended periods after the acute phase of illness. Many of these individuals will benefit from cardiopulmonary rehabilitation, which includes supervised physical and occupational therapy to optimize recovery. A well-coordinated interprofessional team of clinical providers can significantly enhance the outcomes for patients affected by cardiac complications stemming from acute COVID-19 infection.

Conclusion

COVID-19 has emerged as a global health crisis, with its impact extending far beyond respiratory illness to affect multiple organ systems, including the cardiovascular system. The connection between COVID-19 and various cardiac complications has been well-documented, with the most common manifestations being

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acute cardiac injury, arrhythmias, myocarditis, and heart failure. The severity of these complications varies, but they are most prevalent in older patients and those with underlying cardiovascular disease. The increased risk of severe outcomes in these patients highlights the importance of recognizing and managing cardiac complications early in the course of the disease. The pathophysiology of COVID-19-induced cardiac damage remains complex and multifactorial. While viral invasion of cardiac tissue may contribute to direct myocardial injury, other mechanisms such as cytokine storms, microvascular thrombosis, and systemic inflammation play a significant role in exacerbating cardiac damage. Studies have shown that these mechanisms often result in ischemic events, arrhythmias, and structural heart changes such as myocarditis and pericardial effusions. The inflammatory response triggered by the virus, coupled with pre-existing cardiovascular risk factors, creates a particularly vulnerable patient population that requires careful monitoring and management. Clinical evaluations of patients with COVID-19-related cardiac complications involve a combination of biomarkers, electrocardiography, and echocardiography. Elevated cardiac biomarkers, such as troponin, are common in these patients and are used to assess the extent of myocardial injury. Electrocardiograms often reveal arrhythmias and conduction abnormalities, while echocardiography can help identify structural changes in the heart. Despite the challenges in diagnosing cardiac injury, timely and accurate assessments are crucial for guiding treatment decisions, particularly in critically ill patients who may require intensive care. The management of cardiac complications in COVID-19 patients involves a multifaceted approach, including the use of medications to manage arrhythmias, heart failure, and inflammation. Additionally, the identification of high-risk patients with pre-existing cardiovascular conditions is critical to improving outcomes. Post-acute care for COVID-19 patients should include monitoring for long-term cardiac sequelae, as some individuals may experience ongoing symptoms such as fatigue, dyspnea, and decreased exercise capacity. As the pandemic continues, ongoing research into the long-term effects of COVID-19 on cardiovascular health is essential to guide effective treatment and rehabilitation strategies. In conclusion, the cardiovascular impact of COVID-19 is profound and demands increased attention from healthcare professionals. While early diagnosis and treatment can mitigate some of the risks associated with these complications, a comprehensive approach to patient care, from acute management to long-term follow-up, is essential in optimizing outcomes for individuals affected by this novel virus.

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الاعراض القلبية لفيروس كورونا - (COVID-19) مراجعة محدثة للعاملين في الرعاية الصحية

الملخص:

الخلفية: مرض فيروس كورونا 2019(COVID-19) ، الذي يسببه فيروسSARS-CoV-2 ، يؤثر بشكل رئيسي على الجهاز التنفسي، لكنه يظهر أيضًا تظاهرات قلبية هامة. في حين أن الأعراض التنفسية الحادة هي الأعراض النموذجية، يرتبط COVID-19 بالعديد من المضاعفات القلبية مثل مرض القلب الإقفاري، واضطرابات النظم القلبية، وفشل القلب، والتهاب عضلة القلب. ترتبط هذه المضاعفات، خاصةً لدى الأفراد الذين يعانون من حالات قلبية سابقة، بنتائج أسوأ.

الهدف: تهدف هذه المراجعة إلى استكشاف التظاهرات القلبية لـ COVID-19 بشكل شامل، وتقييم فيزيولوجيها المرضية، وتقييم استراتيجيات العلاج الحالية لإدارة هذه المضاعفات.

الطرق: تجمع هذه المراجعة المحدثة نتائج الدراسات الحديثة حول التأثير القلبي لـCOVID-19 ، مستمدة من البيانات السريرية والوبائية والفيزيولوجية المرضية. تم استعراض مصادر الأدبيات من دراسات أكاديمية وسريرية متنوعة تركز على انتشار وتشخيص وإدارة المشاكل القلبية المرتبطة بـ.COVID-19

النتائج: تم الإبلاغ عن المضاعفات القلبية المرتبطة بـCOVID-19 ، بما في ذلك إصابة القلب الحادة، التهاب عضلة القلب، اضطرابات النظم القلبية، وفشل القلب في العديد من الدراسات. تراوحت نسبة الإصابة القلبية بين المرضى الذين تم إدخالهم للمستشفى بسبب COVID-19من 22% إلى 30%، مع زيادة ملحوظة لدى المرضى الذين تزيد أعمارهم عن 60 عامًا. يعتبر المتلازمة التاجية الحادة واضطرابات النظم القلبية شائعة بشكل خاص لدى المرضى الأكبر سنًا الذين يعانون من عوامل خطر قلبية موجودة مسبقًا. تكشف الدراسات بعد الوفاة عن تموت العضلة القلبية، ووجود الوذمة، وتلف الخلايا البطانية، مما يشير إلى فيزيولوجيا مرضية متعددة العوامل تشمل الالتهاب، والتخثر، والغزو الفيروسي.

الخلاصة: يشكل COVID-19 تحديًا معقدًا للعاملين في الرعاية الصحية بسبب تأثيره القلبي الكبير، خاصةً لدى المرضى الذين يعانون من حالات قلبية موجودة مسبقًا. يجب مراقبة المضاعفات القلبية عن كثب، وتعد استراتيجيات العلاج المناسبة أمرًا حيويًا لتحسين نتائج المرضى. يجب أن تركز الدراسات المستقبلية على تحسين طرق التشخيص واستكشاف صحة القلب طويلة الأمد لدى المرضى بعد التعافي من.COVID-19

الكلمات المفتاحية: COVID-19 إصابة القلب، فشل القلب، اضطرابات النظم القلبية، التهاب عضلة القلب، التظاهرات القلبية، ، القلب الحادة، الفيزيولوجيا المرضية