REVIEW ARTICLE

A Review of Cardiac Complications Associated With COVID-19 and Its Vaccination

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Abstract

This review comprehensively examines cardiovascular complications associated with coronavirus disease 2019 (COVID-19) infection and its vaccination, focusing on myocarditis, pericarditis, and other cardiovascular adverse events. We synthesize data from epidemiological investigations, clinical case reports, and global health databases (e.g., World Health Organization, Centres for Disease Control) to assess the prevalence, mechanisms, and clinical outcomes of cardiac injury in hospitalized COVID-19 patients and vaccinated individuals. Acute myocardial injury was observed in 6.9–36% of hospitalized patients, with elevated cardiac troponin levels and left ventricular dysfunction being common; these patients exhibited higher mortality rates and complications such as acute respiratory distress syndrome. Moreover, rare but significant cardiovascular events, particularly myocarditis, were reported post-vaccination, with an incidence of ~1 in 20,000 in young males, often presenting with chest pain and fever within 2–3 days. Cardiac magnetic resonance imaging findings supported these diagnoses, though biopsy results were occasionally inconclusive. The proposed mechanisms for vaccine-induced myocarditis involve immune-mediated responses and spike protein interactions with angiotensin-converting enzyme 2 receptors, while management strategies include corticosteroids and non-steroidal anti-inflammatory drugs, with most cases resolving without long-term sequelae. This review addresses the vaccine hesitancy and emphasises the need for further research to clarify relationships and optimize patient care.

Key words: Angiotensin-converting enzyme 2 receptor, cardiac complications, coronavirus disease 2019, immune response, mRNA vaccine, myocarditis, severe acute respiratory syndrome coronavirus 2, spike protein

INTRODUCTION

The coronavirus disease 2019 (COVID-19) pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus, has had a profound effect on global health, notably through cardiovascular complications. Beyond respiratory symptoms, COVID-19 frequently leads to cardiac issues such as acute myocardial infarction, arrhythmias, and heart failure.[1] Myocardial injury, often indicated by elevated cardiac troponin levels, has been observed in 6.9–36% of hospitalized patients, with higher prevalence in severe cases and those with comorbidities.[2] These patients face poorer outcomes, including acute respiratory distress syndrome (ARDS) and coagulation disorders. The causes of cardiac injury are multifactorial, involving viral invasion, inflammation, hypoxia-induced stress, and endothelial dysfunction.[3]

In addition to direct infection-related effects, rare cardiovascular adverse events, particularly myocarditis and pericarditis, have been observed after administration of mRNA-based COVID-19 vaccination. These events are more common in young males, with an estimated prevalence of

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1–13.3 cases for 100,000 vaccinations, typically presenting with chest pain and fever shortly after the second dose.[4] While most cases are mild and resolve with supportive care, proposed mechanisms include immune-mediated reactions and molecular mimicry.[5]

Comparative studies have shown that the risk of myocarditis is significantly increased after COVID-19 infection than after vaccination, up to 16-fold, along with increased risks of other complications such as arrhythmias and pericarditis in unvaccinated individuals. These findings underscore the importance of risk–benefit analysis in addressing vaccine safety concerns and guiding public health policy.

INTRODUCTION AND BACKGROUND OF COVID-19

The onset of COVID-19 as a worldwide health emergency commenced with the detection of a novel beta coronavirus genome in bronchoalveolar lavage fluid samples from patients in Wuhan, China. On January 3, 2020, researchers from the National Institute of Viral Disease Control and Prevention validated the existence of the virus through extensive epidemiological and etiological studies.[6] The virus, initially termed 2019-nCoV, was subsequently renamed SARS-CoV-2 by the International Committee on Taxonomy of Viruses due to its genetic resemblance to the SARS-CoV virus responsible for the 2003 outbreak. This terminology denotes their common receptor binding mechanism through angiotensin-converting enzyme 2 (ACE2), which enables viral entry into host cells.[7]

The clinical importance of SARS-CoV-2 arises from its distinctive biological traits. The virus has a single-stranded RNA genome that encodes structural proteins, including the spike (S) glycoprotein, which facilitates host cell attachment through ACE2 receptors that are highly expressed in pulmonary alveoli and cardiovascular tissues.[8] On February 11, 2020, the World Health Organization named the disease caused by SARS-CoV-2 as COVID-19, thereby standardizing global reporting and research terminology.[9] This designation aligned with the acknowledgement of COVID-19 as a global public health emergency, indicating its swift transnational dissemination and capacity for grave consequences across all demographics. The pandemic declaration on March 11, 2020, initiated unparalleled global research endeavors to delineate the virus's clinical manifestations and complications.[10]

The clinical spectrum of COVID-19 extends from asymptomatic infection to severe respiratory failure and multi-organ dysfunction. Cardiovascular involvement became a significant factor in disease severity, with initial reports from Wuhan revealing that 19.7% of hospitalized patients exhibited signs of myocardial injury.[11] These observations initiated a thorough examination of the virus's cardiovascular affinity and the mechanisms responsible for cardiac complications, establishing the foundation for current knowledge of COVID-19-associated heart disease.

CLINICAL MANIFESTATIONS AND CHARACTERISTICS

The clinical manifestations caused by COVID-19 are diverse, indicating the systemic nature of SARS-CoV-2 infection. Respiratory symptoms predominate in the initial phase of the disease, with cough occurring in 68% of cases and dyspnea reported in 19–34% of instances.[12] Fever, prevalent in 83–98% of hospitalized patients, demonstrates variable patterns, with certain elderly or immunocompromised individuals displaying atypical afebrile presentations.[13] Constitutional symptoms, including fatigue (38–70%) and myalgia (15–44%), often coexist with respiratory issues, whereas anosmia (41%) and ageusia (38%) present as notable neurological characteristics.[14]

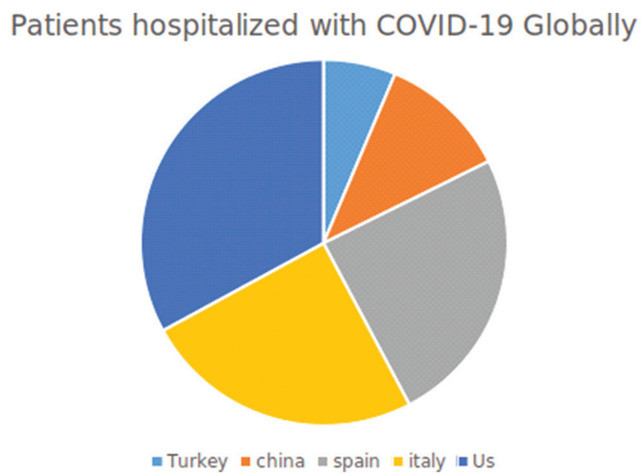
Gastrointestinal involvement is observed in roughly 18% of cases, presenting as diarrhea (13%), nausea/vomiting (10%), or anorexia (40%).[15] Consistently observed laboratory abnormalities include lymphopenia (83%), elevated C-reactive protein (86%), and increased lactate dehydrogenase (76%), indicating the virus's effect on immune and metabolic pathways.[13]

SARS-CoV-2 belongs to the *Coronaviridae* family, characterized by a diameter of 60–140 nm and distinctive spike proteins measuring 9–12 nm that extend from the viral envelope.[16] The virus possesses a single-stranded positive-sense RNA genome consisting of approximately 30,000 nucleotides, which encodes four main structural proteins (spike [S], envelope [E], membrane [M], and nucleocapsid [N]) along with 16 non-structural proteins implicated in replication.[17] The S protein mediates entry into host cells by attaching to ACE2 receptors, which are abundant in respiratory epithelium, cardiomyocytes, and vascular endothelium.[18] Diagnostic confirmation reverse transcription polymerase chain reaction (RT-PCR) testing and analysis of nasopharyngeal swabs, exhibiting sensitivity between 70% and 90% contingent upon specimen quality and viral load.[19] The virus replicates rapidly in the upper respiratory tract during the initial infection phase, with maximum viral shedding occurring 1–2 days before symptom onset, thereby enabling pre-symptomatic transmission.[20] This trait, along with an estimated incubation period of 5–6 days (ranging from 2 to 14 days), presents considerable obstacles for containment initiatives.[21]

The pathogen's stability across diverse environments enhances its transmissibility, with viable virus detectable on plastic surfaces for up to 72 h and in aerosol particles for 3 h under experimental conditions. Nonetheless, actual transmission predominantly transpires through close contact with respiratory droplets, as demonstrated by household attack rates of 10–20%.[22] The virus's mutation rate, although inferior to that of influenza, has resulted in many variants of concern (Alpha, Beta, Gamma, Delta, Omicron) characterized by unique transmissibility and immune evasion attributes.[23]

CARDIAC COMPLICATIONS OF COVID-19

Cardiac injury is a common complication in hospitalized COVID-19 patients, with global prevalence rates between 6.9% and 36% across various populations.[24] Regional disparities are significant, with Italy exhibiting a 27% incidence, Spain 26.9%, the United States 36%, Turkey 6.9%, and China recording rates ranging from 12.4% to 19.7%.[25] This myocardial injury primarily presents as elevated cardiac troponin levels, noted in 12-20% of cases, acting as a sensitive biomarker for cardiomyocyte stress and necrosis.[26]



The pathophysiological mechanisms of COVID-19-related cardiac injury include both direct viral impacts and systemic repercussions of the infection. SARS-CoV-2 infiltrates cells through ACE2 receptors, which are abundant in cardiomyocytes and vascular endothelial cells.[27] The viral replication in these cells causes direct cytopathic effects, while the spike protein's engagement with ACE2 disturbs the homeostasis of the renin-angiotensin system, potentially worsening myocardial dysfunction.[28] Simultaneously, the systemic inflammatory response marked by increased cytokines (IL-6, TNF-α) and C-reactive protein facilitates endothelial barrier disruption and microvascular thrombosis, thereby further impairing cardiac perfusion.[29]

Advanced imaging techniques reveal structural and functional cardiac abnormalities in COVID-19 patients. Echocardiographic analyses indicate left ventricular wall motion abnormalities in 39% of instances, with global left ventricular dysfunction observed in 10–15% of cases. Right ventricular dysfunction is a prevalent finding (35–50%), likely resulting from pulmonary vascular involvement and elevated afterload due to respiratory failure. Pericardial effusion, noted in 22% of hospitalized patients, may indicate systemic inflammation or direct viral pericarditis.[30] The structural changes are associated with clinical deterioration, as patients with cardiac abnormalities experience extended hospitalizations and increased oxygen demands.

Clinical outcomes are markedly inferior for COVID-19 patients with cardiac involvement relative to those without. Individuals with myocardial injury demonstrate a 3.4-fold elevated risk of getting ARDS and a 4.2-fold increased probability of necessitating mechanical ventilation.[31] Further complications encompass acute kidney injury (31% compared to 5% in non-cardiac injury patients), electrolyte imbalances (58% vs. 22%), and coagulation disorders presenting as pulmonary embolism or deep vein thrombosis (28% against 9%).[32] Mortality rates significantly differ between groups, with patients suffering cardiac injury exhibiting a 51.2% in-hospital mortality rate compared to 4.5% in those without myocardial involvement.[33]

Arrhythmic manifestations constitute a significant aspect of cardiac complications associated with COVID-19. New-onset atrial fibrillation arises in 8–12% of hospitalized patients, whereas ventricular arrhythmias (comprising ventricular tachycardia and fibrillation) manifest in 5.9% of cases admitted to the ICU.[34] The electrical disturbances are likely attributable to several factors: direct viral infection of cardiac conduction tissue, cytokine-mediated ion channel dysfunction, and metabolic abnormalities resulting from systemic illness.[35] Thromboembolic events represent a notably fatal complication, as autopsy studies have identified cardiac microthrombi in 62% of deceased COVID-19 patients.[36] The hypercoagulable state caused by SARS-CoV-2 encompasses endothelial dysfunction, platelet activation, and excessive activation of the complement system, resulting in a prothrombotic environment.[37] Clinically significant venous thromboembolism manifests in 21–31% of critically ill patients despite prophylactic anticoagulation, whereas arterial thrombotic events (including myocardial infarction and stroke) impact 3–5%.[38] These findings have necessitated modifications to antithrombotic protocols, leading numerous centers to implement therapeutic-dose anticoagulation for high-risk patients. These findings have transformed clinical practice, highlighting the necessity for regular cardiac monitoring in hospitalized patients and management of cardiovascular risk factors post-infection.

TREATMENT FOR COVID-19

The management of COVID-19 has markedly progressed since the pandemic's inception, with treatment strategies now addressing various facets of the disease's pathophysiology. Supplemental oxygen is fundamental to supportive care, necessary for about 75% of hospitalized patients to ensure sufficient tissue oxygenation.[39]

Anti-inflammatory therapies are crucial in alleviating the cytokine storm associated with severe COVID-19. Dexamethasone, a powerful glucocorticoid, has demonstrated a consistent reduction in mortality (17% relative risk decrease) in patients necessitating oxygen support, especially when given at a dosage of 6 mg daily for up to 10 days.[40] Enhanced immunomodulation utilizing interleukin-6 inhibitors such as tocilizumab and sarilumab offers supplementary advantages in swiftly advancing cases, decreasing 28-day mortality by 4–7% when administered alongside corticosteroids.[41] These agents are generally designated for patients demonstrating significant inflammatory markers (CRP >75 mg/L or ferritin >1,000 ng/mL) and a deteriorating respiratory condition.

Anticoagulation strategies have significantly evolved as knowledge of COVID-19-related coagulopathy has advanced. Low-molecular weight heparin (e.g., enoxaparin 1 mg/kg bi-daily) is currently advised for hospitalized patients exhibiting elevated D-dimer levels (>2–3 times the upper limit of normal) or additional thrombosis risk factors.[42] This method has shown a 30% decrease in thrombotic incidents relative to prophylactic dosing, without a substantial rise in the risk of major bleeding.[43]

Antiviral therapies constitute another essential treatment component, with remdesivir demonstrating limited clinical efficacy (a 5-day regimen correlating with a 30% accelerated recovery) in hospitalized patients not necessitating mechanical ventilation.[44] Novel oral agents such as nirmatrelvir/ritonavir and molnupiravir exhibit significant efficacy in high-risk outpatients, decreasing hospitalization or mortality by 88% and 30%, respectively, when commenced within 5 days of symptom onset.[45]

Monoclonal antibody therapies, although less employed against current variants due to diminished neutralization efficacy, previously significantly contributed to the prevention of disease progression. Convalescent plasma, although initially promising, has demonstrated variable outcomes in clinical trials, leading current guidelines to recommend its use solely in particular immunocompromised populations.[46]

Addressing cardiac complications associated with COVID-19 necessitates specialized strategies. Treatment for myocarditis generally includes non-steroidal anti-inflammatory drugs (NSAIDs) for mild cases and corticosteroids (prednisone 1 mg/kg/day) for more severe instances, accompanied by vigilant hemodynamic monitoring.[47] Arrhythmias may require beta-blockers or antiarrhythmic medications, whereas heart failure is treated in accordance with established protocols utilizing ACE inhibitors/ARBs and diuretics.[48]

Recent data indicate that COVID-19 vaccination, notwithstanding infrequent cardiac side effects, continues to be predominantly advantageous in averting severe illness. The occurrence of vaccine-related myocarditis (approximately 1 in 20,000 among young males) should be considered in relation to the significantly greater cardiac risks posed by SARS-CoV-2 infection itself.[49]

VACCINE DEVELOPMENT AND MECHANISMS

The extraordinary rapidity of COVID-19 vaccine development signified a transformative change in vaccinology, condensing a process that usually spans 5–10 years into under 12 months while maintaining safety standards.[50]

mRNA vaccines (Pfizer-BioNTech BNT162b2 and Moderna mRNA-1273) use lipid nanoparticles to transport synthetic nucleoside-modified mRNA that encodes the spike (S) protein of SARS-CoV-2. Following intramuscular administration, host cells translate this mRNA to synthesise spike proteins that stimulate the immune system, generating both antibody-mediated (humoral) responses and T-cell-mediated (cellular) immunity involving CD4" and CD8' T cells.[51] The platform offers rapid adaptability to new variants, with a redesign capability of 6–8 weeks, and lacks viral components or adjuvants.[52]

Viral vector vaccines (AstraZeneca ChAdOx1 nCoV-19 and Janssen Ad26.COV2.S) utilise replication-deficient adenoviruses engineered to transport spike protein DNA.[53] These vectors infiltrate host cells to produce spike antigens, eliciting strong immune responses through both transgene expression and vector-specific immunity.[54] In contrast to mRNA vaccines, viral vector products exhibit stability at 2–8°C for extended periods, thereby enhancing distribution in tropical areas.[55]

Protein subunit vaccines (Novavax NVX-CoV2373) comprise recombinant spike proteins combined with Matrix-M adjuvant to augment immunogenicity.[56] These nanoparticles replicate the native viral architecture, stimulating antibody production in the absence of genetic material or viral constituents.[57] The platform utilizes proven vaccine technologies (e.g., hepatitis B and HPV vaccines), providing superior safety profiles and formulations that are stable at refrigeration temperatures. Nonetheless, the intricacy of manufacturing and diminished T-cell responses relative to mRNA/vector vaccines poses potential constraints.[58]

Inactivated virus vaccines (Sinovac CoronaVac and Bharat Biotech Covaxin) employ chemically inactivated complete SARS-CoV-2 particles to trigger an immune response. These conventional vaccines offer extensive antigenic exposure but generally necessitate adjuvants and multiple doses to attain sufficient protection.[59] Although exhibiting substantial real-world efficacy against severe disease, their neutralizing antibody levels are generally inferior to those of mRNA/vector vaccines, especially concerning variants.[60] The spike protein is the principal immunological target across all platforms because of its critical function in viral entry through the ACE2 receptor binding.[61] Antibodies induced by vaccines primarily focus on the receptor-binding domain, inhibiting viral adhesion to host cells.[62] T-cell responses target spike epitopes displayed by MHC molecules, offering cross-protection against variants exhibiting antibody escape mutations.[63]

Post-vaccination immune responses exhibit predictable kinetics: Immunoglobulin M emerges within 7 days, immunoglobulin G peaks between 28 and 42 days, and memory B and T cells endure for several months.[64] The emergence of variants has required booster doses to sustain immunity, especially for non-mRNA platforms.[65]

CARDIOVASCULAR ADVERSE EVENTS FOLLOWING VACCINATION

The World Health Organization database recorded the highest incidence of cardiovascular adverse events in individuals receiving the BNT162b2 (Pfizer-BioNTech) COVID-19 vaccine, offering essential insights into post-vaccination complications. Among 17,636 individuals who reported cardiovascular events post-mRNA vaccination, 17,192 cases were linked to BNT162b2, whereas 444 cases were connected to mRNA-1273 (Moderna).[66] Thrombosis was identified as the most commonly reported adverse event associated with all mRNA vaccines, representing a significant share of cases.

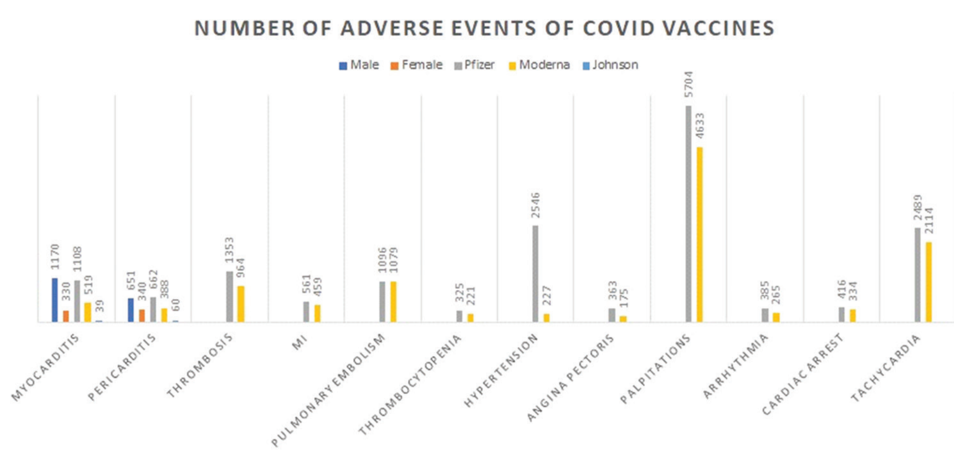
Clinical manifestations exhibited considerable variability, with pericarditis/myopericarditis occurring in 32% of cases, myocarditis in 28%, and arrhythmias in 15%, constituting the predominant cardiac complications. Additional reported occurrences comprised hypotension (9%), hypertension (7%), cardiogenic shock (4%), and pulmonary embolism (3%).[67] Symptom onset generally transpired within 2–3 days following vaccination, with chest pain (89% of myocarditis cases) and fever (33%) as the primary manifestations. Young males exhibited notable vulnerability, with 79% of myocarditis cases occurring in individuals under the age of 30.

The Advisory Committee on Immunization Practices (ACIP) examined 1,226 probable cases of myocarditis/pericarditis reported after the administration of 300 million mRNA vaccine doses up to June 2021. Notably, 67% of cases manifested after the second dose, with a median start interval of 3.8 ± 4.5 days following vaccination.[68] Hospitalization was necessary for 96% of these patients, although the average duration of stay was relatively short at 6 days. Diagnostic assessments indicated elevated cardiac enzymes in 64% of instances and abnormal imaging results in 17%, with ST-segment elevation identified as the predominant electrocardiogram anomaly (70% of recordings).[69]

An age-stratified analysis indicated a distinct risk gradient, with the 16–30 age cohort exhibiting myocarditis at a rate of 1 in 20,000 vaccinations, in contrast to 1 in 100,000 in the general population.[70] This pattern was especially evident in males aged 16–18 years, who exhibited the highest incidence of symptoms on day 2 following vaccination. The clinical trajectory was predominantly positive, with the majority of cases resolving through NSAID therapy and temporary activity limitation; however, 19% necessitated more aggressive interventions, including corticosteroids.

The pathophysiological mechanisms underlying these events are still being studied, with multiple hypotheses gaining support. Structural similarities between viral spike proteins and cardiac antigens can trigger an autoimmune reaction, whereas the vigorous cytokine release after mRNA vaccination could provoke transient myocardial inflammation. The ACE2 receptor pathway is implicated, as vaccine-induced spike protein interactions may disturb cardiovascular homeostasis through renin-angiotensin system modulation.[71]

Thrombosis with thrombocytopenia syndrome (TTS), although infrequent, constitutes a clinically important complication. This condition entails the formation of anti-platelet factor 4 antibodies post-vaccination, resulting in paradoxical thrombosis despite diminished platelet counts.[72] The reported incidence is exceedingly low (approximately 1



in 100,000 vaccinations), with the majority of cases arising within 4–28 days following vaccination.[73]

PATHOPHYSIOLOGY OF VACCINE-INDUCED MYOCARDITIS

The World Health Organization characterizes myocarditis as an inflammatory condition of the myocardium, diagnosed through established histological, immunological, and immunohistochemical criteria.[74] This condition has emerged as a potentially hazardous complication of both COVID-19 infection and vaccination, although the underlying mechanisms vary significantly between these scenarios. Myocardial injury in vaccine-associated cases is generally recognized when cardiac troponin levels exceed the 99th percentile of normal reference values; however, this biochemical marker does not specifically denote myocarditis.[75]

The range of myocardial damage after COVID-19 vaccination includes multiple pathological mechanisms beyond traditional myocarditis. These encompass stress cardiomyopathy, microinfarction resulting from microvascular thrombosis, and cardiac stress associated with hypoxemia.[76] The specific timing of symptom onset, usually 2–3 days after vaccination, indicates an immune-mediated process rather than direct viral cytotoxicity seen in SARS-CoV-2 infection.[77]

Upon intramuscular administration of mRNA vaccines, the mRNA-loaded lipid nanoparticles enter cells by crossing the cell membrane through LDL receptor-mediated endocytosis.[78] Upon internalization, ribosomes translate mRNA into complete spike glycoproteins, which subsequently undergo post-translational modifications before being displayed on the cell surface or secreted extracellularly.[79] These spike proteins elicit both humoral and cellular immune responses, but may also provoke abnormal inflammatory cascades in susceptible individuals through molecular mimicry or excessive cytokine release.[80]

The binding of the spike protein with ACE2 receptors has been associated with cardiovascular complications, as this receptor is highly expressed on cardiomyocytes and vascular endothelial cells.[81] Experimental models indicate that spike protein binding can disrupt ACE2-mediated signaling pathways, potentially resulting in calcium handling abnormalities and myocardial dysfunction.[82] This mechanism may elucidate the ephemeral nature of the majority of vaccine-related cardiac events, as the spike protein is ultimately eliminated from circulation without causing a sustained infection.[83]

Histopathological findings in vaccine-associated myocarditis cases frequently diverge from those of classic viral myocarditis, typically exhibiting restricted lymphocytic infiltration and the absence of viral particles.[84] Immunohistochemical analyses indicate significant infiltration of macrophages and eosinophils, implying a hypersensitivity reaction rather than direct viral cytotoxicity.[85] These observations correspond with the swift clinical enhancement observed in the majority of cases subsequent to anti-inflammatory therapy.

The preference for young males may indicate hormonal effects on immune responses, as testosterone has been demonstrated to augment Th1 polarization and interferon-γ production.[86] Furthermore, the vigorous immune reactivity typical of younger individuals may predispose them to heightened inflammatory responses to vaccine constituents.[87] Genetic factors may also play a role, as specific HLA haplotypes could elevate the risk of vaccine-associated myocarditis.[88]

Recent evidence indicates that lipid nanoparticles may contribute to cardiac complications by inducing transient endothelial activation and prothrombotic alterations.[89] This mechanism may elucidate infrequent instances of simultaneous thrombosis and myocarditis post-vaccination, although these occurrences are exceedingly rare.[90]

The pathophysiological comprehension advances as more intricate molecular investigations are performed. Current data endorse a multifactorial model in which vaccine components interact with host factors to elicit a self-limiting inflammatory response in cardiac tissue.[91] This framework elucidates the ephemeral characteristics of the majority of cases and the favorable long-term outcomes noted in follow-up studies, while affirming the comprehensive safety profile of COVID-19 vaccination.

MANAGEMENT OF CARDIAC COMPLICATIONS INDUCED BY VACCINATION

In mild to moderate cases of myocarditis or pericarditis following COVID-19 vaccination, they are primarily managed using NSAIDs, with ibuprofen (400–800 mg every 6–8 h) or aspirin (81–325 mg daily) proving effective for symptom alleviation.[92] Colchicine (0.6 mg bi-daily) has demonstrated significant efficacy in pericarditis, decreasing recurrence rates when given for 3 months.[93]

Moderate-to-severe cases frequently require corticosteroid therapy, typically involving prednisone (1 mg/kg/day) or methylprednisolone (40–60 mg/day) as standard regimens.[94] Intravenous immunoglobulin given at a dose of 2 g/kg for 2–5 days may be contemplated for refractory cases, especially when autoimmune mechanisms are presumed.[95] Supportive care is essential, necessitating stringent activity limitation for 3–6 months in confirmed myocarditis cases to avert exercise-induced complications.[96]

Hemodynamic monitoring is crucial for patients exhibiting cardiovascular symptoms following vaccination. Continuous telemetry identifies arrhythmias, whereas echocardiography evaluates ventricular function in suspected myocarditis cases.[97] For patients exhibiting symptoms of heart failure, standard pharmacotherapy is initiated, comprising beta-blockers (e.g., metoprolol succinate) and ACE inhibitors (e.g., lisinopril), with meticulous titration to prevent hypotension.[98]

The ACIP advises postponing additional vaccine doses in people who develop myocarditis or pericarditis after the first dose, especially among younger demographics.[99] Clinical follow-up must incorporate serial cardiac magnetic resonance imaging at 3–6 month intervals to assess for late gadolinium enhancement, potentially signifying enduring myocardial fibrosis.[100]

In rare instances of thrombosis with TTS, management diverges considerably from conventional anticoagulation protocols. Non-heparin anticoagulants, such as argatroban or fondaparinux, are favored because of the risk of cross-reactivity associated with heparin-induced thrombocytopenia.[101] Administration of Intravenous immunoglobulin (1 g/kg for 2 days) together with high doses of corticosteroids (methylprednisolone 1 g/day for 3 days) may assist in modulating the immune response responsible for platelet destruction.[102]

The majority of vaccine-related cardiac complications exhibit a benign clinical trajectory, with 85–90% of myocarditis cases demonstrating complete symptom resolution and normalization of cardiac function within 90 days.[103] A minority of patients (5–10%) may encounter enduring ventricular dysfunction or arrhythmias necessitating prolonged cardiology monitoring.

The management strategy must weigh the infrequent risks of vaccine-related cardiac incidents against the recognized cardiovascular advantages of COVID-19 vaccination. Population-level data consistently indicate that SARS-CoV-2 infection presents significantly greater cardiac risks than vaccination, encompassing a higher incidence of myocarditis, arrhythmias, and thrombotic events.[104] The risk-benefit analysis is fundamental to clinical decision-making and public health guidelines concerning COVID-19 vaccination strategies.

CONCLUSION

This study has investigated the cardiac risks associated with COVID-19 infection and vaccination, providing a valuable understanding of their prevalence, mechanisms, and clinical outcomes. Our work indicates that SARS-CoV-2 infection presents significant cardiovascular risks, with myocardial injury impacting up to 36% of hospitalized patients; however, vaccine-related cardiac complications are infrequent and generally mild. The observed demographic patterns, especially the occurrence of myocarditis, are predominantly observed in young men after vaccination, underscore the necessity for customized risk communication and monitoring strategies. These findings augment the accumulating evidence affirming the overall safety and net advantages of COVID-19 vaccination, while underscoring the necessity for diligent cardiac monitoring in high-risk groups.

Multiple mechanisms, including molecular mimicry, cytokine storm, direct invasion, and autoimmunity, have been postulated to explain the occurrence of myocarditis induced by the COVID-19 vaccine. However, the connection. These factors remain circumstantial, and it is unclear whether either predominates in the execution and maintenance of myocarditis and myocardial infarction. There is a lack of comprehensive systemic studies or evidence indicating that the COVID-19 vaccine induces cardiac complications. Misinformation and extensive reporting of vaccine-related myocarditis contribute to vaccine hesitancy, despite adverse reactions being rare, easily treatable, and having a favorable prognosis. Further evidence-based research validating the beneficial effects of vaccines should be conducted and widely disseminated among the general population to dispel unnecessary suspicions and encourage greater vaccination uptake.

Comprehensive research conducted by the Indian Council of Medical Research and All India Institute of Medical Sciences on sudden adult fatalities following COVID-19 has determined no correlation between COVID-19 vaccinations and sudden deaths. Future research must emphasize longitudinal studies to elucidate the long-term cardiovascular effects of both COVID-19 infection and vaccination, especially in at-risk populations. Mechanistic studies of the immunological pathways involved in vaccine-associated myocarditis may guide the creation of safer vaccine platforms and targeted therapeutic strategies. As the pandemic progresses, continuous surveillance will be crucial to assess the cardiac effects of new variants and revised vaccine formulations, ensuring that clinical practices and public health policies are based on solid scientific evidence.

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