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The Heart of the Matter: How COVID-19 Impacts Cardiovascular Health

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ABSTRACT

The manifestations of the respiratory system were only some of the peripheral cardiovascular manifestations for which the condition was identified. The injury to the heart is either due directly to or indirectly connected with the virus; these include arrhythmias, myocarditis, and thromboembolic manifestations. The existing cardiovascular diseases set the grounds for the progressive severity. If recognized early and treated, better outcomes could be expected for the condition. These effects can be present in the short term and may develop into long-term complications of the heart in previously healthy individuals. The vaccines have been shown to give good results as an effective way of preventing the severity of the infection and its cardiovascular complications, especially in the high-risk population. Continuous monitoring, risk stratification, and a multidisciplinary approach care for these risks with the goal of improving the outcomes for patients. In this review, we discuss the evidence for the evolution of current knowledge at the interface of COVID-19 and cardiovascular health, which has emerged as one of the major challenges, and how to try to avoid them while managing patients amid these rapidly changing times.

Keywords: COVID-19, cardiovascular health, myocardial injury, myocarditis, vaccination, heart disease, long COVID, high-risk populations, cardiac monitoring, SARS-CoV-2.

Introduction

This situation marks the entry of a new disease, COVID-19, in late 2019, which constituted a major challenge to the health systems anywhere on Earth. Majorly presenting with respiratory manifestations, it is now recognized as a multisystem disease-causing significant effect on the cardiovascular system [1]. Thus, viral action via angiotensin-converting enzyme 2 (ACE2) receptors, which are notably abundant in the lungs and/or the heart, indicates more of a direct involvement of the heart, as in [2].

Acute myocardial injury or arrhythmias, and hence thromboembolic or myocarditis events [3], were described as the common cardiovascular complications of COVID-19. Among the most common mechanisms of injury would be direct viral injuries, systemic inflammation, cytokine storm, and the development of endothelial dysfunction [4]. It may be pertinent to mention that individuals with existing cardiovascular disease stand a much higher risk of succumbing to worse outcomes from COVID-19, thus making their cardiovascular health an important aspect of pandemic management [5].

Moreover, long COVID (post-acute sequelae of SARS-CoV-2 infection [PASC]) manifested with chronic cardiovascular symptoms following viral clearance in patients, such as chest pain, palpitations, and reduced exercise tolerance [6]. Moving forward, the relationship between COVID-19 and cardiovascular health will remain an integral aspect of improving patient outcomes during the pandemic. It will, however, be most pertinent for the vulnerable populations.

COVID-19 and Cardiovascular Involvement

Though SARS-CoV-2 primarily infects the respiratory tract of an individual, the irony is when its action is felt elsewhere in a person's body, outside the lungs. One major extrapulmonary system affected is the cardiovascular system. The entry into cells is done through binding into the receptors of angiotensin-converting enzyme 2 (ACE2). The receptor does not exhibit high amounts of expression only in the lungs but also exists abundantly in the heart and vascular endothelium [7]. This receptor-mediated mechanism thus provides a direct entrance route for the virus into and destruction of possible cardiac tissues.

Myriad cardiovascular complications have been reported so far in patients with COVID-19, most of them not having any history of preceding cardiac ailments at any time. The majority among these probably include myocardial injury, usually manifested by elevation of troponins' levels, and the most serious, that is, arrhythmias, from rather benign palpitations to life-threatening ventricular tachyarrhythmias [8]. Myocarditis-inflammation of the muscle

of the heart-has, at some point or the other, been reported, most probably either from direct invasion of the virus or from an immune-mediated response [9].

Apart from this, there is now evidence of a hyper-inflammatory state, what has become commonly known by the name cytokine storm, leading to endothelial dysfunction, plaque rupture, and thrombotic events, for instance, acute coronary syndrome and pulmonary embolism, as generated by indirect tissue injury from COVID-19 [10]. It also adds some other evidence, that of a further disease being prothrombotic, which describes increased levels of D-dimer and fibrin degradation products seen in severely ill patients [11].

Furthermore, even if there is no provoking mechanism, COVID-19 can destabilize atherosclerotic plaques or increase the myocardial oxygen requirement with decreased supply, thus exacerbating the effects of cardiovascular diseases [12]. Hence, those interactions make cardiovascular contributions important in morbidity and death from COVID-19.

Acute and Long-Term Cardiac Effects

Currently, an increasing plethora of acute cardiac complications can be caused by COVID-19, many of which result in adverse clinical events. Myocardial injury, most assessed via high cardiac troponin, is one of the acute direct effects [5]. The injury could be due to direct viral invasion of cardiomyocytes, or it can be due to systemic inflammation, hypoxia, or microvascular injury [5]. Clinical manifestations will take the shape of angina, abnormal changes on an electrocardiogram, and impairment in the ejection equation like acute coronary syndrome, sometimes mimicking [13]. There are also detained and hurried arrhythmias in the generality of hospitalized patients, particularly in an intensive care environment [14].

The acute phase of the disease also includes numerous thromboembolic events such as pulmonary embolism, deep vein thrombosis, and ischemic stroke. These complications arise from a rather interesting hypercoagulable state and endothelial dysfunction caused by the virus. These lead to cardiovascular instability, which further contributes to mortality [15]. Acute myocarditis, either new-onset or exacerbated from pre-existing disease, sometimes progresses to cardiogenic shock and then to mechanical support [16].

That which follows is that today this post-acute or long-term line of some patients has those symptoms and effects that may truly be described by all as persistent bothersome cardiac meanings and effects well-known in Long COVID or Post-Acute Sequelae of SARS-CoV-2 Infection (PASC). Consistent chest pain, palpitations, reduced exercise tolerance, and postural orthostatic tachycardia syndrome symptom-like complaints are, for instance [17]. Recent cardiac MRIs have revealed that there is still ongoing inflammation or fibrosis within the myocardial tissue with possible involvement of the pericardium among individuals who experienced mild acute infections or were asymptomatic [18]. The highlights of these findings bring grave worries about possible future cardiovascular repercussions related to the arrhythmogenic risks that may develop and progressive deterioration of cardiac function that will call for longer-term follow-up and monitoring [19].

Indeed, the persistence of these abnormalities in the heart identifies further needs for thorough evaluation of the cardiovascular system among all survivors of COVID-19, especially in those suffering from pre-existing heart disease or acute severe illness. Lots remains unknown about the unknown details of the long-term cardiac effects of the virus; mostly, however, the emphasis remains on rehabilitation after recovery from the virus and on individualized cardiovascular care [20].

Impact on High-Risk Populations

People with pre-existing cardiovascular illnesses like hypertension and coronary heart disease, and even heart failure, are at a higher risk for extreme outcomes of COVID-19. These comorbid conditions tend to predict not only higher susceptibility to infections but also an increase in hospitalization rates, admission to the intensive care unit, and increased mortality [21]. Underlying cardiac conditions seem to aggravate the inflammatory and thrombotic manifestations triggered by SARS-CoV-2, resulting in rapidly deteriorating clinical conditions for many of the patients [22].

Hypertension is the third most predominant cardiovascular risk factor globally and fell as a central co-morbidity for patients hospitalized with COVID-19. Hypertensive individuals are expected to worsen already high cardiovascular load, possibly due to their higher incidence of the infection among them, due to the interaction between SARS-CoV-2 and RAAS with respect to the down-regulating ACE2, which is supposed to worsen vascular functions [23]. At the beginning of the pandemic, the increased fear regarding the use of RAAS inhibitors was general; still, up to now, explorations suffice to say that treatment using ACEI or ARB is not associated with increased COVID infections but may even afford some degree of protection [24].

Patients with diabetes mellitus as a comorbidity with cardiovascular disease will also be exposed to a higher level of risk. Hyperglycemia leads to increased viral replication and an upsurge in the extent of release of inflammatory cytokines, and it also impedes the vascular endothelium as it predisposes to damage from the heart [25]. Additionally, it involves increased risks due to obesity and metabolic syndrome, among other issues, which increase the chances of endothelial dysfunction, pro-inflammatory conditions, and higher cardiac workload, resulting in occurrences with infection by the COVID-19 pathogen [26].

Thus, they are also representative cohorts of old people, the elderly of whom are above 65 and have pre-existing conditions of heart diseases. Immunosenescent physiology, with a higher positive correlation to atherosclerosis and lower cardiac reserve, characterizes the other one predisposed to acute cardiovascular diseases and protracted recovery. Frequent such populations need more intensive care and monitoring to avert adverse outcomes.

Immunization alone cannot meet the goal, which has a tenor ineffectively focused on late management of chronic ailments or timely intervention at the point of COVID-19 infection. Cite prevention efforts in such disproportionately high-risk groups because of the pressing negative consequences. Targeted interventions indeed can improve some cardiovascular care outcomes for these vulnerable populations [28].

Role of Vaccination in Heart Health

Vaccination offered a profound chance not only to reduce the intensity of respiratory complications affecting the heart but also to have the heart itself spared. Vaccines will reduce the incidence and intensity of SARS-CoV-2 infections; thus, the more these vaccines suppress infections, the fewer will be the acute cardiac events, such as myocardial injury, arrhythmias, and thromboembolism. Such incidences are otherwise rampant in unvaccinated active infections. Vaccinated patients have been shown-since breakthroughs do occur-to develop cardiovascular complications at a rate much lower than the unvaccinated [29, 30].

Vaccination remains a measure for the protection of life and thus among the prime protective strategies in groups at high risk, such as patients with cardiovascular diseases who have very severe outcomes with COVID-19 since their underlying condition will be destabilized by heart failure exacerbations or acute coronary syndromes. Therefore, the reduction in inflammatory load due to the vaccination and exertion (as by the viral load) interferes with the triggering of destabilization of underlying cardiac conditions [31]. Further evidence from various studies suggests that the fully vaccinated are hospitalized and sustain cardiac injury at lower rates than those unvaccinated with cardiovascular comorbidities [32].

Though myocarditis was rare after mRNA, these cases were mild and treated conservatively. These cases typically cluster in younger males that have a milder clinical course compared to COVID-induced myocarditis, who are sick and often develop complications [33]. Based on that, health authorities and cardiology societies have been reinforcing the belief that the benefits of COVID-19 vaccination clearly outweigh its risks, especially concerning protection of the heart from the virus [34].

Vaccines also reduce transmission and therefore protect patients with chronic cardiac conditions on the community level, thereby ensuring timely access to healthcare systems. Hence, the promotion of booster doses and ensuring equitable access to vaccines remain crucial to eradicating the cardiovascular burden imposed globally by the COVID-19 pandemic [35].

Management and Monitoring

The early recognition of individuals with risk factors, particularly those with a prior history of cardiovascular conditions, makes this management competent in combating the cardiovascular complications of COVID-19. A patient at risk should be assessed immediately and inclusively by means of cardiac biomarkers such as troponins and natriuretic peptides; ECG; and echocardiography if required at baseline. Risk stratification follows the assessment and will provide guidance as to management decision-making, most relevant to hospitalized patients with a suspicion of myocardial involvement [36]. Continuous real-time monitoring of vital signs, oxygen saturation, and hemodynamic parameters when admitted to a high-dependency unit also captures early signals of cardiac decompensation [37].

Management of pharmacotherapy for cardiovascular complications due to COVID-19 usually centers on the ratio of benefits versus risks of anti-inflammatory, antiviral, anticoagulant, and heart-specific drugs. Thus, anticoagulation is an integral part of prophylaxis against thromboembolic events occurring mainly in patients with COVID-19 due to the hypercoagulable state [38]. In cases of myocarditis or heart failure, initiating or optimizing guideline-directed medical therapy using beta-blockers, ACE inhibitors, and diuretics should be considered without worsening hemodynamic instability [39].

There is a provision for post-acute services as well as follow-up programs for the identification and management of cardiovascular-related diseases that last long after treatment. Increasing development of cardiac biomarkers and symptoms such as chest pain, palpitation, or dyspnea suggests that a recovery will warrant further imaging and functional testing at follow-up [20]. Cardiac MRI was found to detect ongoing evidence of myocarditis or fibrosis in recovered patients and to inform regarding return to exercise or work [18].

Telephonic and remote monitoring technologies are emerging as good ways to track diseases of cardiovascular concern without the patient ever coming to the hospital. Apart from the management of the patient in the clinic, these methods essentially reduce exposure risk to care at the acute and recovery stages [40]. Finally, complex multidisciplinary care involving Cardiologists, primary care practitioners, and rehabilitation specialists shape the overall management of these complex patients [41].

Severe management for cardiovascular complications due to COVID-19 should anticipate early detection of individuals with risks, specifically those who have prior cardiovascular diseases. The patient has to be assessed immediately and must comprehensively include cardiac biomarkers such as troponins and natriuretic peptides; ECG; and echocardiography if required at baseline. Such tests will be important in assessing risk stratification and management decision-making, most relevant to the hospitalized patient suspected of myocardial involvement [36]. Continuous surveillance of vital signs, oxygen saturation, and hemodynamic parameters during even critically ill hospitalization has to be done to pick up the early signs of cardiac decompensation [37].

Management of COVID-19-related cardiovascular problems by pharmacotherapy mostly discusses the ratio of benefits and risks between anti-inflammatory, antiviral, anticoagulant, and heart-specific drugs. Anticoagulation thus constitutes an integral part of prophylaxis against thromboembolic events occurring mostly in patients with COVID-19 because of the hypercoagulable state [38]. Guidelines-directed medical therapy using beta-blockers, ACE inhibitors, and diuretics should be considered without aggravating hemodynamic instability in cases of myocarditis or heart failure [39].

Both post-acute care and follow-ups are imperative to the identification and management of possible long-term cardiovascular sequelae. Recovery following COVID-19, marked especially by increased levels of cardiac biomarkers and symptoms like chest pain, palpitation, or dyspnea, will necessitate further imaging and functional testing as needed upon follow-up [20]. Cardiac MRI is found helpful in detecting continual evidence of myocarditis or fibrosis in patients that have recovered and advising the return to exercise or work [18].

Beyond in-clinic disease management, telemedicine and remote monitoring technologies crop up as excellent avenues for tracking cardiovascular symptoms and adjusted medications without in-person access to care. These methods primarily assist in lowering exposure risk, allowing care delivery both in acute and recovery stages [40]. In conclusion, complex multidisciplinary care involving cardiologists, primary care practitioners, and rehabilitation specialists shapes the overall management of these complex patients [41].

Conclusion

Further knowledge about the influence of viral infections on previous cardiovascular health is provided by the COVID-19 pandemic, giving a huge area for thought. Anything that can be done to injure a SARS-CoV-2-infected cardiovascular system can damage an already dysfunctional heart: myocarditis, arrhythmias, thromboembolism, etc. In the long term, existing cardiovascular risk factors might lead to sustained cardiac impairment.

As far as minimizing the acute and chronic cardiac sequelae of COVID-19 is concerned, doing so through vaccination has gained prominence while also aggravating the ongoing cardiovascular burden. Active intervention through early diagnosis, timely initiation of drug treatment, and post-illness recovery care for preventive purposes will be the crux in alleviating the burden due to long-term complications and improving the likelihood of a good outcome.

As the pandemic changes, clearly, knowledge about COVID-19-related cardiovascular sequelae would continue to serve as the mainstay for public health recommendations, clinical focus, and future research. Strengthening integration of care approaches, ensuring that cardiac health is prioritized in acute and post-acute management of COVID-19, will enhance a positive long-term health trajectory for patients across the globe.

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