Post Covid Patients Cardiac Health

Vanita Arora*, Pawan Suri[1]

1. Dr Pawan Suri, MD; DM; FRCP (Edin); FESC; FSCAI

Chief Cardiologist, SGL Multispecialty Hospital, Jalandhar, Punjab, India.

Corresponding Author: Dr Vanita Arora, MD; DNB (Card); FRCP (Edin); FHRS ; FESC

Cardiac Electrophysiologist & Interventional Cardiologist, New Delhi, India.

Introduction:

Covid 19 originated in Wuhan, China in late December 2019 and within a few months, it became a 'Public Health Emergency of International Concern' (1). Covid 19 causes mild symptoms in the majority (81%) of patients and substantial symptoms like dyspnoea, respiratory rate of 30/min, blood oxygen saturation of 93% in 14%, while severe symptoms like respiratory failure, septic shock, or multiple organ dysfunction or failure in 5% of critically ill patients. The overall case fatality rate on average is between 2-3% but an individual case fatality rate is highest among those with associated cardiovascular disease (10.5%) and associated comorbidities like hypertension (6%), obesity and diabetes (7.3%) (2).

SARS-CoV-2 causing Covid 19 is a member of the family Beta coronavirus and has four structural proteins (S, E, M, N) with the S protein having a novel furin cleavage site that makes it more pathogenic and capable of causing multi-organ damage due to its higher affinity to human ACE2 receptors. ACE2 is highly expressed at the tissue level in lungs, kidneys, heart, blood vessels and although lung injury and acute respiratory distress syndrome have been at a center stage as the most dreadful complications

of Covid 19, the heart damage has recently emerged as yet another grim outcome in the virus's repertoire of possible complications.

The cardiac involvement occurs as a direct invasion of cardiomyocytes by the virus via the ACE2 receptors and also indirectly by the inflammatory reaction due to the cytokine storm (3). Cardiac symptoms in form of myocarditis, acute coronary syndrome, arrhythmias and thromboembolism can be the first clinical manifestation especially in those patients who present without the typical symptoms of fever, cough, etc. Covid 19 not only increases the severity of already pre-existing heart disease but also unmasks the cardiac symptoms in previously undiagnosed heart disease as well.

It may be a new onset myocardial injury either due to myocarditis or Acute Coronary Syndrome. Acute myocardial injury is associated with a significantly worse prognosis and in the majority of patients, the typical signs of myocarditis like segmental wall motion abnormality, reduced left ventricular ejection fraction (LVEF) or mild pericardial effusion may not be seen (5). The patients of Covid 19 who develop the clinical features of heart failure have elevated levels of amino-terminal pro-B-type natriuretic peptide (4). Although the actual incidence is unknown, there are reports that Covid 19 can cause ACS with or without the presence of systemic inflammation. The plaque rupture, coronary spasm or microthrombi due to the direct endothelial or vascular injury leading to thrombus formation are the proposed mechanisms (6).

Cardiac-specific biomarkers (CK, CK MB, Troponin, Myoglobin, and BNP) are important in recognizing patients of Covid-19 with early cardiac involvement as the presence of myocardial injury has been associated with over 50% mortality. The elevation in troponin is seen in almost 12% of Covid-19 patients and is believed to reflect a non-coronary disease due to a decrease in oxygen availability compared to an increased need in presence of cytokine storm in critically unwell Covid-19 patients. The elevated troponin also has a potential prognostic value as it is associated with a mortality of 59.6% compared to 8.9% in patients with normal levels. High mortality was seen in patients with elevated levels even without a history of CVD (37.5% vs 69.4%) (7).

Almost all types of atrial and ventricular arrhythmias ranging from brady to tachyarrhythmias have been seen in Covid 19 patients either related to the direct myocardial injury or the systemic causes such as fever, hypoxia, electrolyte abnormalities and sepsis. The usage of antiviral medications and few antibiotics in the treatment of Covid 19 are also known to induce arrhythmias in few patients.

The coagulation abnormalities that can lead to both venous and arterial thromboembolism are also associated with the Covid 19. An elevated level of D-dimer, fibrinogen, factor VIII, modestly reduced platelet counts and slightly prolonged prothrombin time due to severe inflammatory response and endothelial damage in combination with underlying comorbidities is seen in these patients.

In addition, certain antiviral medications and other therapies used in these patients are potential contributors to thrombus formation (8). Based on the data of patients with recovered myocarditis, MI, or another cardiac injury, it is expected that some patients may have subclinical and possibly overt cardiovascular abnormalities. There have been reports of delayed manifestations weeks or months after recovery in form of thromboembolism, arrhythmias, acute coronary syndrome and more drastically sudden cardiac arrest. In addition, patients who have recovered from the acute stage also seem to be at high risk of recurrent events. While current treatment appropriately focuses on acute recovery, it is unknown whether the treatment given during the acute illness may affect future CV abnormalities. Keeping in view the long-term impact of Covid 19 and cytokine storm, it is imperative to determine if acute delivery of antifibrotic therapy, anti-inflammatory therapy, cell-based therapy, or antiviral therapy affects long-term as well as short-term cardiac outcomes (9).

More than a year and a half since the first reported cases in 2020, in the global pandemic we find ourselves at a critical point. With survivors outnumbering those infected and with vaccines in distribution, we pay attention to the long-term cardiovascular effects of COVID-19. Nonetheless, as surges continue worldwide owing to new variants and a lag in vaccine distribution, our understanding of the impact of COVID-19 on the cardiovascular system is constantly evolving.

Recent findings have shown that the early case reports described a spectrum of cardiovascular manifestations of COVID-19, which are myocarditis, stress cardiomyopathy, myocardial infarction, and arrhythmia. But in most cases, the myocardial injury appears to be predominantly mediated by the severity of critical illness rather than direct injury to the myocardium from viral particles. While cardiac magnetic resonance (CMR) imaging remains a powerful tool for diagnosing acute myocarditis, it should be used judiciously in light of the low baseline prevalence of myocarditis.

"Long COVID" or post-acute sequelae of SARS-CoV-2 infection has been described. The reported symptoms include fatigue, palpitations, chest pain, breathlessness, brain fog, and dysautonomia including postural tachycardia syndrome (POTS). Management of POTS/dysautonomia primarily centers on education, exercise, and salt and fluid repletion. (10)

Unraveling the mysteries of COVID-19 has been a challenging exercise in science and experimentation. The latest observational, pathologic, imaging and clinical studies have clarified the short-term and long-term impacts of COVID-19 on cardiac health.

References

1. Wu F, Zhao S, Yu B, Chen YM, Wang W, Song ZG, et al. "A new coronavirus associated with human respiratory disease in China". Nature 2020;579:265-69.

2. Clerkin KJ, Fried JA, Raikhelkar J, Sayer G, Griffin JM, Masoumi A, et al. "COVID-19 and cardiovascular disease". Circulation 2020;141:1648-55.

3. Walls AC, Park YJ, Tortorici MA, Wall A, McGuire AT, Veesler D. "Structure, function, and antigenicity of the SARSCoV- 2 spike glycoprotein". Cell 2020;181:281-92.

4. Mehra MR, Ruschitzka F. "COVID-19 illness and heart failure: a missing link?" JACC Heart Fail 2020;8:51214 .

5. Shi S, Qin M, Cai Y, Liu T, Shen B, Yang F, et al. "Characteristics and clinical significance of myocardial injury in patients with severe coronavirus disease 2019" Eur Heart J 2020;41:2070-79.

6. Stefanini GG, Montorfano M, Trabattoni D, Andreini D, Ferrante G, Ancona M, et al. "ST-elevation myocardial infarction in patients with COVID-19: clinical and angiographic outcomes". Circulation 2020;141:2113-16.

7. Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, et al. "Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19)". JAMA Cardiol 2020;5(7):811-18.

8. Connors JM, Levy JH. "Thromboinflammation and the hypercoagulability of COVID-19". J Thromb Haemost 2020;18:1559-61.

9. Shafi AMA, Shaikh SA, Shirke MM, Iddawela S, Harky A. "Cardiac manifestations in COVID 19 patients - a systematic review". J Card Surg 2020;35:1988-2008.

10. Michael Chilazi, Eamon Y Duffy, Aarti Thakkar, Erin D Michos "COVID and Cardiovascular Disease: What We Know in 2021" Current Atherosclerosis Reports Vol 23; Article 37 (2001)