EDITORIAL

COVID 19 and Cardiovascular Disease

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Coronavirus disease 2019 (Covid 19) originated in Wuhan, China in late December 2019 and in a time duration of few months only has become a 'Public Health Emergency of International Concern' (1). Covid 19 causes no or mild symptoms in a significant majority (81%) of patients, substantial symptoms like dyspnoea, respiratory rate e"30/min, blood oxygen saturation d"93% in 14% and 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 an 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).

SARSCoV-2 causing Covid 19 is a member of the family Beta corona virus 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 centre 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 an already pre-existing heart disease but also unmasks the cardiac symptoms in a previously undiagnosed heart disease as well. Apart from this the drugs that are repurposed and are supposed to have a preventive or a therapeutic potential are also supposedly causing cardiac side effects.

Approximately, one fourth of patients admitted with Covid 19 develop the clinical features of heart failure, evidenced by elevated levels of amino-terminal pro-B-type natriuretic peptide (4). The heart failure might be the result of an exacerbation of pre-existing conditions or a new onset myocardial injury either due to myocarditis or Acute Coronary Syndrome. Acute myocardial injuryis associated with a significantly worse prognosis and in 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). 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 on to thrombus formation are the proposed mechanisms (6).

Cardiac specific biomarkers (CK, CK MB, Troponin, Myoglobin, and BNP) are important in recognizing patients

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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 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 exemplified by Guo *et al*, that revealed that elevated troponin level was 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 to the systemic causes such as fever, hypoxia, electrolyte abnormalities and sepsis. The usage of antiviral medications and few antibiotics in treatment of Covid 19 are also known to induce arrhythmias in few patients.

The coagulation abnormalities that can lead to both the 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 the potential contributors to thrombus formation (8).

Based on the data of patients with recovered myocarditis, MI, or other cardiac injury, it is expected that some patients will 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 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).

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