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Editorial

Covid-19 Bites the Lung but does not Leak the Heart

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COVID-19 infection and heart

SARS-CoV-2 causes COVID-19 which is the severe acute respiratory syndrome (SARS). The COVID-19 pandemic is in full swing for last two years in successive waves causing a significant mortality and morbidity. Most of the cases are mild. Only less than 10% of COVID-19 suffers from SARS. Covid-19 infection progresses through the three phases. The 3rd phase is the cytokinin storm phase which directly affects liver, kidney, and heart in addition to the SARS. Though the Covid-19 infections spans over a period of 3 to 4 weeks but in some patients, the course of disease is unpredictable, leading to the death in a few days depending upon the age, gender, comorbidity, immune response, the strength of cytokinin storm [1], vaccination status [2] and treatment.

Involvement of multiple organs are seen because of cytokine storm. COVID-19 bites the lung but does not leak the heart. When there are existing underlying heart diseases prior to the COVID-19, cardiovascular involvement goes beyond direct myocardial injury like vasculitis, increased incidence of the thrombosis and pulmonary thromboembolism [3,4]. Persistent cardiac injury even after SARS, which is evident by the elevated troponin level, causes a greater number of deaths [5].

In the patients who have a greater number of comorbidities, the cardiovascular manifestations take severe course [6].

The spectrum of cardiovascular system involvement includes acute coronary syndromes sometimes unmasking the occult underlying cardiovascular diseases, arrhythmias, myocarditis, vascular inflammation, and thrombosis [7,8].

Myocarditis is seen up to the extent of 27% of cases, myocardial ischemia is seen up to the extent of 22% of cases and both are seen in 6% of cases of COVID-19 which explains the basis for type I and type II myocardial infarction and inducible ischemia beyond myocarditis [8]. Inflammatory cells have seen in the myocardium during the autopsy of the patients with COVID-19 but its true incidence yet to be documented [9]. There is a direct relationship between the level of cardiac troponin and death [10-12] but this correlation needs further observation in children [13].

Both the arterial and venous thrombosis are quite common due to several mechanisms. Immune-mediated thrombotic mechanisms, complement activation, macrophage activation syndrome, antiphospholipid antibody syndrome, hyperferritinemia and renin-angiotensin system dysregulation have been proposed. Many of these pathogenic processes can be evaluated by the measurement of the relevant biomarkers which carries potential prognostic value in COVID-19 [14].

Pulmonary thromboembolism(PTE) is detected in 29% of patients who underwent contrast enhanced pulmonary angiogram(CTPA) scanning because of suspicion of PTE [15].

The ECG change is quite dynamic in Covid-19 and may be the marker for the critical status of the patient. These changes include sinus tachycardia, PVC, bradyarrhythmia and ST-T changes simulating myocardial infarction [16,17]. The ECG changes and the elevated level of the biomarkers creates the illusion of the acute coronary syndrome which needs proper evaluation before revascularization.

The echocardiographic changes during COVID-19 include dilatation of the cardiac chambers like the right ventricle, left ventricle, right atrium, and inferior vena cava. Left ventricular ejection fraction and right ventricular fractional area are reduced. Pericardial effusion is quite common. The severity of the right ventricular dilation directly proportionates to the mortality [18].

Like the empirical use of steroid, immune modulators, and monoclonal antibodies along with the anti-viral and antibiotic therapy in moderate to severe form of the COVID-19, the patient also needs intravenous or oral anticoagulation because of high risk of arteriovenous thrombosis resulting in the type I and II myocardial infarction [19]. Depending upon the cardiovascular manifestations, the patient needs specific care starting from the evaluation by CT coronary angiogram, cardiac MRI or invasive coronary angiogram to up the treatment i.e., the revascularization. Some patients who have the myocarditis or pericarditis may be benefited by colchicine [20]. As the incidence of atheromatous plaque rupture is frequent and there is increased risk thrombosis, type I MI is also quite common in the COVID-19 in which the angioplasty is preferred to thrombolysis .The proper infection control measure is required to reduce the transmission of covid-19 infection to the least .However ,the revascularization strategy depends upon the match between disease severity, availability of resources and the institutional COVID-19 protocol [21-23].

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