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Cardiovascular Manifestations of COVID-19

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ABSTRACT

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Severe COVID 19 is a systemic illness characterized by hyperinflammation, cytokine storm and cardiac injury. Cardiovascular manifestations and emerging therapeutic prospects are reviewed

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Severe COVID-19 is associated with rapidly progressing systemic inflammation, a pro-inflammatory cytokine storm, and sepsis, leading to multiorgan failure and death. SARS-CoV-2 anchors on transmembrane ACE2 to enter the host cells including type 2 pneumocytes, macrophages, endothelial cells, pericytes, and cardiac myocytes leading to inflammation and multiorgan failure. In particular, the infection of endothelial cells or pericytes could lead to severe microvascular and macrovascular dysfunction. Furthermore, in conjunction with the immune over-reactivity, it can potentially destabilize atherosclerotic plaques and explain the development of the acute coronary syndromes. Infection of the respiratory tract, particularly of type 2 pneumocytes, by SARS-CoV-2 is manifested by the progression of systemic inflammation and immune cell overactivation, leading to a 'cytokine storm', which results in an elevated level of cytokines such as IL-6, IL-7, IL-22, and CXCL10. Subsequently, it is possible that activated T cells and macrophages may infiltrate infected myocardium, resulting in the development of fulminant myocarditis and severe cardiac damage. This process could be further intensified by the cytokine storm. The viral invasion could cause cardiac myocyte damage directly leading to myocardial dysfunction and contribute to the development of arrhythmia. Acute Cardiac injury varies from 1-4% in non ICU cases to 1/3 rd to 2/3 rd cases in severe cases in various early studies.[1] There is a delay between initiation of symptoms and myocardial damage in studies reported so far.

COVID-19 and cardiac arrhythmia

In a report on 138 hospitalized COVID-19 patients, 16.7% of patients developed arrhythmias, which ranked only second among serious complications after ARDS.[2] Arrhythmia was observed in 7% of patients who did not require ICU treatment and in 44% of subjects who were admitted to an ICU.[3] Further details of these manifestations included atrial fibrillation, conduction block, ventricular tachycardia, and ventricular fibrillation. These arrhythmias are also observed in viral myocarditis.

COVID-19 and myocardial injury and heart failure

A number of studies indicate that cardiac complications, including fulminant myocarditis, are potential outcomes of SARS-CoV-2 infection. Reports indicate that 7.2%[2] to 17%[4] of hospitalized COVID-19 patients sustain acute myocardial injury. This may be in the form of acute myocarditis or injury secondary to an oxygen supply/ demand mismatch [type 2 myocardial infarction (MI)]. Approximately 52% of non-survivors had heart failure as compared to 12% of survivors.[4] Transient ECG changes are common and may help to detect the presence and severity of myocardial injury. Myocarditis may progress to conduction block, tachyarrhythmias, and impairment of left ventricular function.

Hence patient monitoring should include a number of laboratory tests. In a study of 191 patients, presence of NT pro BNP and D-dimer also denote key relationship with a requirement for ICU care and mortality.

Myocarditis appears in COVID-19 patients after a prolonged period (up to 10–15 days) after the onset of symptoms. Moreover, investigators in China point to a lack of viral particle identification on endomyocardial biopsy. Given that acute myocardial injury is said to begin 2 weeks after the onset of symptomatic COVID-19, adaptive T-cell-mediated immunity or dysregulated innate effector pathways are likely to play a pivotal in the development of myocardial inflammation.[5]

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COVID-19 and ischaemic heart disease

Several pathways associated with viral diseases may contribute to destabilize plaques in COVID-19 patients. Viral illness can potentially destabilize atherosclerotic plaques through systemic inflammatory responses & cytokine storm. It is important to consider that type 2 MI is the most common subtype in viral conditions, thus the usefulness of invasive management with a view towards coronary revascularization (especially in type 2 MI) is limited. Hence the decision for invasive vs non-invasive management of patients with an ACS and COVID-19 illness should be carefully considered.

COVID-19 and coagulation abnormalities

Disseminated intravascular coagulation (DIC) and pulmonary embolism, characterized by increased D-dimer levels and fibrin degradation products, are highly prevalent in COVID-19. DIC has been observed in 71.4% of non-survivors.[5] Massive pulmonary embolism has also been reported. Experience from China indicates that a D-dimer increase is highly predictive of adverse outcomes in COVID-19 and elevated D-dimer levels (>1 g/L) were strongly associated with in-hospital mortality.

Cardiovascular effects of potential therapies for COVID-19

The antimalarial drugs Chloroquine and Hydroxychloroquine have recently received considerable attention and interest for the treatment and possible prophylaxis of COVID-19. However, the data to date in support of these drugs are weak and cardiac toxicities are considerable. A systematic review of the literature performed on patients treated with these drugs, for an extended period of time (median 7 years) demonstrated conduction disorders as the main side effect (85%). Other adverse cardiac events included ventricular hypertrophy, hypokinesia, heart failure, pulmonary arterial hypertension, and valvular dysfunction.

Impact of COVID-19 on routine and emergency cardiovascular care

In preparation for the COVID-19 pandemic, many healthcare providers have had to scale down outpatient services and also defer elective cardiac procedures and surgeries. The long-term clinical impact of scaling down outpatient activity, reduced access to diagnostics, and deferral of routine procedures is likely to be significant. Similarly, the perceived risk of being exposed to COVID-19 has led to a decline or a delay in presentation of acute cardiac emergencies which is likely to contribute to cardiac mortality and morbidity.

END NOTE

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