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# Myocarditis Associated with Covid-19 Pneumonia

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The covid-19 pandemic is one of the most serious worldwide concerns, which has created many problems in health, economics, and other aspects of human life around the world. Coronavirus has shown to have a lot of manifestations during the infection, among which some of them are more critical. Cardiac complications can be considered one of the major and serious problems caused by coronavirus infection... .

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Myocarditis is a well-known cardiac complication of viral infections. Human immune cells (mainly T cells) are responsible for this problem [1]. In addition, pro-inflammatory cytokines

like Interleukin-6 (IL-6) are inflammation mediators by cytokine storm [2]. Myocarditis is normally diagnosed by histopathologic criteria and magnetic resonance imaging (MRI), but unfortunately, it is usually undiagnosed until the appearance of dilated cardiomyopathy (DCM), [3].

Several different case reports reported myocarditis as a consequence of covid-19 infection [4-6]. This increases the importance of considering myocarditis as a serious side effect of this viral infection.

The possible physiopathology of coronavirus-induced myocarditis is by angiotensin-converting enzyme 2 (ACE2). This membrane protein is expressed in the type II pneumocytes and cardiomyocytes. So, it could be inferred that the expression of ACE2 in both these cells is responsible for coronavirus-induced myocarditis. TMPRSS2 receptor is also necessary for coronavirus spikes to cleave before binding to ACE2 [7]. Coronavirus accessory proteins probably are responsible for its infective properties, like impairing the stress granule of the infected cells; therefore, the replication of the virus is accelerated [8]. Heart-produced hepatocyte growth factor (HGF) interaction with its receptor on naïve T cells (c-Met) during cardiotropism [9] and cell-mediated cytotoxicity by CD8+ T cells (which primed by antigen-presenting cells) are capable of inducing the cardiomyocytes inflammation [10]. Proinflammatory cytokines like IL-6 also cause a positive feedback loop during the cytokine storm.

Viral induced myocarditis presentations can be a spectrum from mild fatigue and dyspnea [11] to fulminant myocarditis with ventricular dysfunction, tachycardia, and heart failure [1, 12].

Other possible cardiac problems can also be considered with these symptoms. It is important to set them aside for the proper treatment of these patients. Differential diagnoses are acute coronary syndrome, sepsis-related cardiomyopathy, and takotsubo cardiomyopathy. Cardiac-related blood tests (troponin, BNP, NT-proBNP) and inflammatory biomarkers like erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and procalcitonin are probable helpful tests to distinguish myocarditis from other diseases with the same clinical symptoms [10].

Long-lasting complications of viral cardiomyopathy are still unknown. Further studies are necessary to determine its most probable long-term side effects.

According to American Heart Association (AHA) protocol, fulminant myocarditis should be managed by the cardiogenic shock based on inotropic agents, vasopressors, and respiratory support by mechanical ventilation [13].

There is another challenge during the covid-19 pandemic, which is the interference of therapeutic plans of different diseases and covid-19 infection, especially in heart-associated diseases (e.g., NSAIDs and QTc-prolonging agents) [14, 15].

Overall, since covid-19 is a serious contagious disease, the frequency of post-viral inflammatory diseases would increase rapidly. Thus, myocarditis as a consequence of covid-19 should be taken into account to reduce its complications and delayed disabilities.

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