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Editorial

Recommendation of Hemodynamic Stabilization and GDMT Therapies in Patients with Heart Failure: COVID-19 and Myocarditis

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Editorial

SARS-COV-2 is the causative agent of COVID-19 disease that has gained worldwide attention since December 2019 [1]. Millions of people around the world have been infected with this deadly pamdemic that has caused significant mortality.

Human-to-human transmission of SARS-COV-2 has been reported to be higher than that of other Corona viruses [4]. COVID is transmitted through respiratory particles and aerosols and respiratory droplets [10]. Symptoms may include fever, dry cough, shortness of breath, fatigue, weakness, headache, sore throat, and loss of sense of taste [8,11,13]. Corona virus is a single-stranded RNA virus with glycoprotein coatings and polysaccharide-coated spikes that are responsible for binding to angiotensin-2-converting enzyme receptors in host cells, facilitating virus entry into the target cells [9]. Angiotensin-Converting Enzyme 2 (ACE2) is abundant in type 2 pneumocytes as well as cells of cardiovascular system, kidney and gastrointestinal tract, causing multiorgan involvement and clinical manifestations of COVID-19 [6]. Patients with underlying comorbidity such as hypertension, diabetes, obesity, cardiovascular disease, lung disease and cancer are more likely to develop more severe disease and multiple organ failure [3,6,10,11]. COVID-19 has major consequences for the cardiovascular system, including myocarditis [12].

In this review, we intend to examine the association between myocarditis and the COVID-19 disease.

During this pandemic, patients with COVID were at several times higher risk of developing myocarditis than patients without COVID, and this risk varies with age and gender [21]. Diagnosis of cardiac involvement may be challenging, but it is of particular importance due to its adverse effect on clinical outcomes [8].

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Viral infections are one of the causes of myocarditis and COVID-19 is one of the infectious causes that causes inflammatory involvement of the heart muscle and can increase hospitalization for heart failure and sudden death [16]. During 2019 and 2020, the percentage of myocarditis has increased dramatically [2]. Acute myocarditis is defined by the presence of a virus in myocardial tissue. Inflammatory infiltrations and myocardial damage are rare. The presence of macrophages in myocardial interstitial cells is seen in most cases [12]. The mechanism of SARS-COV-2 infection and the development of myocarditis are not yet fully understood, and its pathophysiology is probably similar to that of other viruses [14,15].

Myocarditis due to COVID can even occur in the absence of pulmonary involvement and is merely a manifestation of the disease. Myocardial involvement may be due to a variety of mechanisms, including direct myocardial infarction caused by the COVID virus itself, electrolytic ischemia, and abnormal coronary thrombosis [17].

While the exact pathophysiology of SARS-C0V-2 myocarditis remains unknown, the proposed mechanisms include: 1. Immune-mediated 2. Autoimmune-mediated 3. Caused by the virus itself [18]. The diagnosis of COVID-induced myocarditis is similar to other cases of viral myocarditis based on clinical laboratory findings of CMR[1] and pathology [1]. Unfortunately, there is no single laboratory test for myocarditis, and elevated serum troponin in patients with COVID-19 may identify patients with severe type and myocardial involvement increases the risk of mortality [29]. Natriuretic peptides (NT) and inflammatory markers such as ESR[2], CRP[3] are other tests used that are sensitive but not specific (20). Elevated liver enzymes, creatinine, lactic acid, organ dysfunction and hypoperfusion nonspecifically suggest cardiogenic shock. Elevated troponin and NT pro BNP are seen without acute coronary involvement, so myocarditis should be suspected in patients with COVID-19 who present with acute heart failure and cardiogenic shock and have no evidence of coronary involvement [12].

Abnormal findings in ECG of patients with myocarditis include low voltage rhythm, disturbances QRS due to myocardial edema, ST-T changes and conduction abnormalities. These abnormalities are sensitive but not specific for the diagnosis of myocarditis [21,22]. Echocardiography is a non-invasive test suitable for examining the heart defects of COVID-19, the findings of which include left ventricular myocardial dysfunction, left ventricular thrombus, edema, and pericardial effusion [21]. CMR is the best test to evaluate ventricular function and evaluation in myocarditis. If available, it is recommended to show edema and interstitial fibrosis [21].

Presence of myocardial edema in T2 mapping and nonischemic myocardial injuries in T1 and late gadolinium enhancement are myocardial findings in CMR [23]. CMR should be considered as the first test in stable patients· However, in the case of myocardial biopsy, it should be borne in mind that myocardial biopsy is not routinely recommended in all patients with myocarditis COVID-19 and should be limited to severe and refractory cases [12].

Endomyocardial biopsy is often used to aid in the diagnosis of myocarditis and is based on the DALLAS criteria [23,24]. However, the clinical manifestations of myocarditis can be seen as an acute chest pain, ST-T change, arrhythmia, acute heart failure and hemodynamic instability [12].

Management

Due to the novelty of the disease and the lack of clinical data, the management of secondary myocarditis to COVID-19 is very similar to other causes of myocarditis and there is no specific strategy at present [25]. Steroid administration for viral myocarditis is controversial and may help reduce the immune response to COVID-19. In severe cases, treatment is more likely to lead to steroids [31]. The use of corticosteroids and immunomodulatory therapy routinely is not recommended and corticosteroids should be prescribed based on pulmonary conditions [12].

In immunocompromised myocarditis and damage to the myocardium and the development of dilated cardiomyopathies, the use of immunosuppressive therapies such as corticosteroids and azathioprine did not show significant clinical benefits, but there is evidence that other safety-modifying strategies may be effective [27-30]. Patients with myocardial infarction should be treated with guideline directed medical therapy (GDMT) including ACEi[4], ARB[5], ARNi[6], MRA[7] beta-blockers and diuretics [25].

There are concerns about ACEi, ARB, ARNi that no harm has been observed due to the current clinical results and it is recommended to use it [12,31,32]. In cases of myocarditis with acute heart failure and cardiogenic shock, the use of Mechanical Circulatory Support (MCS), inotropes and vasopressors is recommended [2,12]. There are limited data on the prognosis of myocarditis in patients with pre-existing heart problems and increased troponin during the course of the disease have worse outcomes, including mortality Further increase the length of hospital stay and the need for mechanical ventilation [33,34]. Finally, these findings emphasized the importance of vaccination as soon as possible to reduce these complications [35]. After the injection of COVID vaccine, cases of myocarditis following side effects of the vaccine have been reported, From December 2020, after vaccination, the risk of myocarditis following COVID decreased, especially in young people, but myocarditis and pericarditis were seen after vaccination [36]. The Vaccination Committee concluded that the benefits of COVID-19 vaccination clearly outweighed the risks of myocarditis after vaccination [35].

1 Cardiac magnetic resonance imaging

2 Erthrocyte sedimentation rate

3 C-Reactive protein

4 Angitensin-converting-enzyme inhibitors

5 Angiotensin receptor blockers

6 Angiotensin receptor neprilysin inhibitor

7 Aldosterone receptor antagonists

Summary

Myocarditis is a serious cardiac complication of SARS-COV-2 infection and has different clinical symptoms. Its prevalence is still unknown and screening should be performed in patients to be found in high-risk patients. CMR Clinical examination laboratory tests and echocardiography recommended. Supportive therapies such as inotropes and MCS are used to treat it. Hemodynamic stabilization as well as GDMT therapies are recommended in patients with heart failure. Further long-term studies are needed to evaluate the risk and complications of the disease. Routine use of corticosteroids to treat myocarditis is not recommended.

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