AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 01, Issue 10, 2023 ISSN (E): 2993-2149

Heart Failure in Coronavirus Infection

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Abstract: In the context of globalization, a new type of coronavirus infection has led the entire healthcare system of all countries to a high alert mode, which made it possible to comprehensively study this disease and its complications. This article summarizes changes in the cardiovascular system based on the publications of European and Chinese scientists.

Keywords: Coronavirus infection, COVID-19, cardiovascular system, myocarditis, arrhythmia, cardiogenic syndrome, stroke.

SARS-CoV-2 infection has serious cardiovascular consequences, including myocardial injury, myocarditis, acute coronary syndrome, pulmonary embolism, stroke, arrhythmias, heart failure, and cardiogenic shock.

Cardiac manifestations of COVID-19 may be associated with adrenergic effects, systemic inflammatory response, macrophage activation syndrome, direct viral infection of myocardial and endothelial cells, hypoxia due to respiratory failure, electrolyte imbalance, fluid overload, and side effects of some COVID-19 medications.

The effect of a previous infection caused by SARS-CoV-2 on long-term consequences in patients with cardiovascular pathology remains not fully understood. At the same time, an equally important issue is the risk of occurrence, the course of diseases of the cardiovascular system and the choice of therapy after suffering COVID-19.

Myocardial damage. Myocardial injury is defined by elevated troponin levels and can be caused by both ischemic and non-ischemic factors. One of the possible mechanisms of acute myocardial injury caused by SARS-CoV-2 infection may be its affinity for ACE2, which is widely expressed in the heart, which leads to direct damage to it [1,2]. Other hypothesized factors are: cytokine storm caused by dysregulation of T helper types 1 and 2, hyperreactivity of the sympathetic nervous system, anemia, and hypoxic damage to myocardial cells caused by respiratory dysfunction (type 2 myocardial infarction) [3]. According to studies from China, myocardial injury occurs in 7-20% of hospitalized patients with COVID-19 [4]. SARS-CoV-2-associated myocardial injury occurred in 5 of 41 COVID-19 patients (12.2%) in Wuhan, as identified by elevated levels of highly sensitive troponin I (>28 pg /mL) [5]. In a small meta-analysis (4 studies, 341 patients), troponin I levels were significantly higher in patients with severe symptoms associated with COVID-19 [6] compared with moderate patients. Myocardial injury, present in 19.7% of patients with COVID-19, was associated with higher levels of inflammatory cytokines, severe lung injury, and a high need for noninvasive and invasive ventilation. These patients were more likely to develop ARDS, acute kidney injury, impaired coagulation hemostasis, and were associated with a higher risk of death. [7]

Myocarditis and pericarditis In a series of reports of 68 deaths in a cohort of 150 patients with COVID-19, in 33% of cases, myocardial inflammation could play a role in the cause of death.

Patients with COVID-19 with non-acute myocardial injury may experience either transient viremia or migration of infected macrophages from the lungs. The available data do not exclude the classic manifestation of myocarditis (i.e., direct infection of myocardial cells with a virus), although there is an assumption that myocardial involvement in COVID-19 is rather caused by a cytokine storm [8]. Patients with COVID-19 have an aberrant T cell and monocyte response leading to a systemic hyperinflammatory response characterized by increased production of pro-inflammatory cytokines and chemokines (tumor necrosis factor, IL-2, IL-6, IL-7, CCL2, etc.) . Which leads to myocardial damage through infiltration of mononuclear cells into cardiomyocytes of patients with fulminant myocarditis and a high viral load of SARS-CoV-2. Pericarditis is described in the literature by isolated cases, as an acute manifestation of COVID-19, and as long-term complications after SARS-CoV-2 infection [9, 10].

Acute coronary syndrome. COVID-19 increases the risk of coronary plaque rupture as a result of an intense inflammatory response [11]. As previously reported by Kwong et al . patients with acute respiratory infections have an increased risk of subsequent development of acute myocardial infarction both after influenza and after other viral diseases, including COVID-19 [12]. The actual prevalence of ACS during COVID-19 infection is unknown given the gaps in identification of SARS-CoV-2 observed in many countries during the early stages of the pandemic, especially in the absence of typical symptoms suggestive of COVID-19 infection [13].

In 28 Italian Patients with ST Elevation Myocardial Infarction (STEMI) and COVID-19 Stefanini et al . reported that ST segment elevation is one of the most common cardiovascular complications of COVID-19 (85.7%) [14]. It should be noted that angiography demonstrated the absence of coronary artery obstruction (CHD) in 39.3% of cases. Similar data was presented by researchers from the United States Bangalore et al . who found that one third of patients with ACS clinic had no obstruction of the coronary arteries according to angiography. In these patients with STEMI , the hospital mortality rate was 72% [15]. In addition to type 2 myocardial infarction, "myocarditis" and stress cardiomyopathy , microvascular thrombosis has also been proposed as a mechanism underlying certain cases mimicking manifestations, ST elevation without obstructive CAD, given the endothelial dysfunction and hypercoagulable state associated with COVID-19 [16].

Cardiac arrhythmias In 138 hospitalized patients with COVID-19, arrhythmias were the leading complication (19.6%) and were more common in patients requiring ICU transfer (44.4% vs 6.9%). Guo et al . showed that in 187 patients with COVID- 19, malignant ventricular arrhythmias occurred twice as often in the presence of elevated troponin levels (11.5% vs. 5.2%). Ventricular arrhythmias may also represent the first clinical manifestation of SARS-CoV-2 infection. In 136 patients with COVID-19 who underwent cardiac arrest in the hospital, Shao et al . found that the most common initial rhythm was asystole in 89.7% of cases [17]. Pulseless electrical activity was detected in 4.4%, while a rhythm requiring pacing was detected in only 5.9% of patients. In 4 provinces of Italy Baldi et al . reported a 58% increase in out-of-hospital cardiac arrest in the 40 days of the COVID-19 outbreak compared to the same period in 2019 [18].

Under the conditions of COVID-19, arrhythmias can be caused by the following mechanisms: direct viral damage to myocardial cells and/or conduction system; worsening of pre-existing heart disease or conduction disorders; electrolytic disturbances; adrenergic stress leading to electrical instability; and ACS with ongoing ischemia [19]. The high inflammatory activity characteristic of COVID-19 is another potentially important proarrhythmic factor. Inflammation is a new risk factor for long QT syndrome and torsades de pointes , primarily due to the direct effect of cytokines, in particular, IL-1, IL-6 and TNF-α on the myocardium, disrupting the functioning of cardiomyocyte channels (K + and Ca ++) [20].

Heart failure and cardiogenic shock. Concomitant heart failure has been observed in 23–49% of patients infected with COVID-19 [21,22]. It was associated with a worse prognosis, as it

occurred almost 5 times more often in patients who did not survive hospitalization (51.9% vs. 11.7%) [23]. As with troponin , elevated levels of B-type natriuretic peptides (BNP/NT- proBNP) are associated with poor outcomes in patients with ARDS [24]. In the setting of COVID-19, heart failure may be associated with either an exacerbation of underlying cardiovascular disease or a new onset of cardiomyopathy (especially myocarditis or stress cardiomyopathy). Isolated right ventricular failure can occur with pulmonary hypertension associated with severe ARDS or pulmonary embolism [25].

Moreover, in elderly people with cardiovascular disease, left ventricular hypertrophy and diastolic dysfunction are often observed. Thus, these patients may be prone to developing pulmonary edema if they are given copious intravenous fluids to maintain blood pressure or as a means of parenteral drug administration [26].

Venous thromboembolism and pulmonary embolism. Patients with COVID-19 have an increased risk of venous thromboembolism (VTE). In addition to prolonged immobilization, endothelial injury and vascular inflammation contribute to the development of a hypercoagulable state . In a multicentre Chinese study, an increase in D- dimer levels (>1000 μg/L) was an independent predictor of hospital death [27]. In a study of 184 patients with severe COVID-19 from 3 centers in the Netherlands, 31% of patients developed VTE despite pharmacological prophylaxis [28]. Poissy et al . reported an incidence of pulmonary embolism (PE) of 20.4% (95% CI, 13.1–28.7%) in patients with severe or very severe COVID-19 [28]. In 90.1% of cases, PE occurred in patients already receiving prophylactic antithrombotic treatment. The incidence of thromboembolism in this cohort was significantly higher than that observed before the pandemic in patients with various conditions of similar severity [29].

Stroke. Ischemic stroke has been recognized as a complication of severe forms of COVID-19, which is thought to be associated with a highly prothrombotic state and severe infection-induced endothelial dysfunction [30]. Beyrouti et al . reported a series of cases of stroke in 6 patients with COVID-19; large vessel occlusion with markedly elevated D- dimer levels (≥1000 μg/l) was observed in all patients. Three patients had multifocal stroke; 2 had concurrent VTE; and in 2 cases, ischemic stroke occurred during anticoagulant therapy [31]. Similarly, Oxley et al . reported 5 young patients with COVID-19 (aged 33 to 49 years) who had suffered large vessel ischemic stroke [32].

Conclusion: With coronavirus , cardiovascular complications we show an average of 20 to 49 percent, depending on the type of pathology . The most common among which are inflammatory diseases of the myocardium and a complication of concomitant pathology from the cardiovascular system. This high trend obliges medical staff to more closely examine the cardiovascular system in patients with coronavirus infection. We believe that a screening study of biochemical markers of the cardiovascular system in patients with coronavirus infection can help to reduce mortality among patients and speed up the recovery process

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