In late December 2019, the COVID-19 pandemic emerged from Wuhan, Hubei province in China, overwhelming health-care facilities. As of this writing, COVID-19 has infected more than 20 million people worldwide.

The vast majority of COVID-19-related complications are respiratory, ranging from mild flu-like symptoms to life-threatening acute respiratory distress syndrome (ARDS). The heart is also involved, as it is in close correlation with the changes happening in the lungs. The mechanisms leading to the cardiac complications in COVID-19 are not well understood yet. It is thought to be mostly due to increased stress caused by pulmonary diseases (such as ARDS), direct myocardial infection (MI) by the virus, and thrombotic events (such as MI, strokes, and negative inotropic effect of cytokines).

In general, the echocardiogram can be used as a method to follow the function and structural changes in the heart. In the transthoracic echo examination, the chief abnormality was right ventricle (RV) dilatation in 41% with 27% RV dysfunction. Increased D-dimer and C-reactive protein levels are the indicators of RV damage. On the other hand, left ventricular function was hyperdynamic or normal in most of the patients.

More specifically, RV involvement in COVID-19 patients leads to structural and functional abnormalities in which a marked reduction in radial RV systolic function with a relative sparing of longitudinal shortening have been shown. McConnel sign (severe wall hypokinesia/akinesia and apical sparing) as it refers to acute pulmonary embolism (PE); however, it is not that specific but, it may raise the risk of having acute cor-pulmonale secondary to ARDS.

A retrospective study on COVID-19 nonsurvivors without known cardiac impairment demonstrated an increased incidence of RV dilation and dysfunction and elevated pulmonary vascular resistance (PVR). Thus, the evaluation of RV function should be considered as a predictor of mortality in COVID-19 patients.

The association between COVID-19 infection and RV impairment is well established by the current data, and this association might contribute to the rapid hemodynamic deterioration, arrhythmias, and sudden cardiac death in patients with COVID-19. Studies keep emerging to identify the causes and complications in COVID-19 patients. We discuss some of the most important ones with relation to the heart.

Respiratory complications of COVID-19 infection are the most frequent, of which ARDS is the most critical. A recent retrospective study showed that the development of ARDS in 58.5% of hospitalized patients with COVID-19 who had underlying cardiac injury. RV dysfunction specifically was found to be an independent predictor of mortality in moderate-to-severe ARDS patients.

RV failure development is attributed to increased PVR from increased vasoactive mediators, vascular remodeling, hypoxic pulmonary vasoconstriction, intravascular thrombosis, and vascular compression from atelectasis and edema. Recent autopsy reports of COVID-19 patients showed evidence of RV dilation. This may be due to the thin-walled RV which is particularly susceptible to ischemia in response to sudden increases in myocardial stress.
in afterload and/or coronary occlusion by microthrombi which in turn may compromise left ventricular function.\textsuperscript{[8]}

The other major issue is the need for a mechanical ventilator, which leads to an uncoupling between RV and pulmonary circulation, and eventually RV failure.\textsuperscript{[6]}

Reports and new data demonstrate a hypercoagulable state associated with the infection as evidenced by elevated d-dimer and fibrin degradation products, a prolonged prothrombin time, and thrombocytopenia. This is complicated by the increased incidence of PE. As a result, it adds an extra strain on the RV.

The hemodynamic response of the RV to acute PE depends on the size of the embolus, the degree of obstruction, the response to vasoactive, and cardiopulmonary baseline status. RV failure is considered the principal determinant of early mortality in acute PE. Therefore, the early detection of RV dysfunction and myocardial injury is crucial in COVID-19 patients.\textsuperscript{[8,9]}

The immunological reaction characterized by a developing the cytokine storm that contributes to RV dysfunction through their negative inotropic effects on the myocardium. Depressed RV contractility with acutely elevated PVR caused by ARDS and PE may lead to death.\textsuperscript{[6]}

CONCLUSION

RV involvement in COVID-19 patients increases the risk of its failure. Angiotensin-converting enzyme 2-mediated heart injury, the negative inotropic effect of cytokines, and the increased afterload from ARDS and PE are the suggested mechanisms of RV involvement. Identification of RV impairment might prompt physicians to limit positive end-expiratory pressure and avoid hypercapnic acidosis, which could otherwise adversely affect RV performance by inducing pulmonary arteriolar vasoconstriction and increased RV afterload.

RV dysfunction early detection and treatment will decrease the mortality and enhance the outcome of COVID-19 treatment. Further researches should be conducted to understand the precise molecular mechanisms of RV pathology in COVID-19.

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Conflicts of interest
There are no conflicts of interest.

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