

The Right Ventricle in COVID-19 Patients

Subjects: Cardiac & Cardiovascular Systems

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Cardiac involvement has been described during the course of SARS-CoV-2 disease (COVID-19), with different manifestations. Several series have reported only increased cardiac troponin without ventricular dysfunction, others the acute development of left or right ventricular dysfunction, and others myocarditis.

Keywords: COVID-19 ; cardiac imaging ; cardiac disease

1. Characteristics and Importance of Right Ventricular Dysfunction

Isolated RV dysfunction in patients with COVID-19 may suggest acute pulmonary hypertension, right myocardial infarction, or focal myocarditis. Given the high incidence of acute respiratory distress syndrome (ARDS) requiring mechanical ventilation and the state of hypercoagulability responsible to the development of deep vein thrombosis and pulmonary embolism, lung parenchyma and pulmonary microcirculation damage may contribute to RV dilation and dysfunction. In addition, positive end-expiratory pressure, common ventilatory support, can acutely worsen RV function, and this persistent dysfunction correlates with an adverse prognosis ^[1].

Data on RV involvement in COVID-19 have been published. The group of Szekely and Lichter ^[2], who first conducted the above-mentioned echocardiographic study within 24 h of patients' admission, described RV dilation in 40% of patients, with or without dysfunction. The pulmonary acceleration time (AT) was reduced, demonstrating an increase in pulmonary vascular resistance, and FAC and the peaks' wave at TDI were lower than in normal values, demonstrating a reduced RV function. The reduction in AT, which suggests a RV afterload increase, assumed a prognostic value, being associated with more complex clinical pictures. Progressive clinical deterioration observed in 20% of patients and troponin increase were related to RV dilatation and dysfunction. Other investigators provided further information. Lazzeri et al. ^[3] demonstrated, in a population of 42 patients with moderate to severe ARDS, that troponin release was related to RV dysfunction. Argulian et al. ^[4] conducted an echocardiographic study on 105 consecutive hospitalized patients, of which 30% had mechanical ventilatory support at the time of the echocardiographic examination. RV dilation was observed in 31% of patients and on multivariate analysis, RV dilation was the only variable significantly associated with mortality. To evaluate the prevalence and prognostic value of pulmonary hypertension and RV dysfunction in patients admitted to non-intensive care units, Pagnesi et al. ^[5] conducted an observational echocardiographic study on 200 patients. The prevalence of pulmonary hypertension (PAPs ≥ 35 mmHg) was 12% and that of RV dysfunction (TAPSE < 17 mm or s-wave < 9.5 cm/s) 14.5%. Patients with pulmonary hypertension were older and with more comorbidities and with signs of more severe pulmonary impairment, in radiological, laboratory, and oxygen saturation terms. Patients with RV dysfunction, on the other hand, had more comorbidities but no evidence of more severe lung disease. Furthermore, patients with pulmonary hypertension, unlike those with RV dysfunction, were associated with a higher rate of in-hospital mortality and ICU transfer. On the basis of these findings, it is imperative for the clinician to pay attention to RV function in hospitalized patients, especially in patients with severe pneumonia, ARDS, and on ventilation.

2. Information from Speckle Tracking Echocardiography

In a study by Li et al. ^[6], 120 consecutive patients with COVID-19 underwent echocardiography to study RV systolic function, using both conventional parameters (such as FAC, TAPSE and peak s' wave) and the RV free wall longitudinal strain (FWLS) by 2D-STE. Patients with greater impairment of RV longitudinal deformation (strain cut-off value of -23%) had increased heart rate; elevated CRP and D-dimer values; increased incidence of acute myocardial injury, ARDS, and deep vein thrombosis; higher mortality; and received high flow oxygen and mechanical ventilation. Interestingly, RV-FWLS predicted mortality more accurately than FAC, becoming an important echocardiographic marker of patients at-risk. Most notably, patients were included without categorizing them according to their comorbid status. Furthermore, detection of RV longitudinal strain as an independent predictor of mortality might be due to the enrolment of a relatively high number of patients with significant comorbidities. Gibson et al. ^[7] determined the RV-FWLS in 32 patients receiving mechanical

ventilation for COVID-19-associated respiratory failure. They proved that abnormal RV-FWLS was present in the majority (66%) of patients and, unexpectedly, was associated with favourable lung mechanics (i.e., compliance) and lower airway pressures, suggesting that RV-FWLS in this population may not be attributable to alveolar collapse or distension during positive pressure ventilation. RV-FWLS correlated negatively with age and with serum troponin. Patients with abnormal RV-FWLS did not exhibit worse oxygenation, hypercarbia, or acidosis, and, consistent with these findings, did not have radiologic evidence of more severe lung disease to account for the RV impairment. The authors considered two alternative mechanisms to explain impaired RV function: pulmonary vascular abnormalities, such as micro or macro thromboembolic phenomena that can increase RV afterload, and direct myocardial injury causing impaired contractility. Direct myocardial damage could occur in severe COVID-19 either from inflammation or viral entry into cardiomyocytes, which, however, has not been demonstrated so far. Lastly, they found that abnormal RV-FWLS was associated with markers of reduced LV systolic and diastolic function. LV dysfunction in previously normal hearts may be seen during acute pulmonary hypertension as a consequence of ventricular interdependence. Alternatively, RV-FWLS may provide an early indication of global cardiac impairment in COVID-19 patients, as factors causing direct injury to the RV would also damage the LV.

Studies evaluating RV function during follow-up of COVID-19 patients have been published using 2D-STE. Günay et al. [8] enrolled 51 patients with COVID-19 (29 with severe and 22 with moderate pneumonia) and 32 healthy volunteers. They showed that RV dysfunction continued in the first month after discharge. RV-GLS strain and RV-FWLS were lesser in COVID-19 patients than in the control group. Another study confirmed subclinical dysfunction of RV by 2D-STE in hospitalized patients in relation to the severity of pneumonia after recovery from COVID-19 [9]. The median follow-up duration was 4 months. After recovery from COVID-19, echocardiography was performed in 79 consecutive patients. According to the recovery at home vs. hospital, patients were divided into two groups: home recovery ($n = 43$) and hospital recovery ($n = 36$). Comparisons were made with age, sex, and risk factor-matched control group ($n = 41$). In patients recovered from hospital, RV-GLS and RV-FWLS were impaired compared to the control group. In subgroup analysis, RV-FWLS was impaired in patients with severe pneumonia compared to mild-moderate pneumonia, without pneumonia and control groups. A significant correlation was detected between serum CRP level at hospital admission and both RV-GLS and RV-FWLS. Age, male gender, pneumonia on computed tomography, and need for steroid in treatment were identified as independent predictors of impaired RV-FWLS ($>-18\%$) via multivariate analysis.

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