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Response to Letter to the Editor: Timely Identification of Hospitalized Patients at Risk for COVID-19-Associated Right Heart Failure Should Be a Major Goal of Echocardiographic Surveillance



To the Editor:

We thank Dr. Dandel¹ for his thoughtful Letter to the Editor regarding our recent paper “Echocardiographic Correlates of In-Hospital Death in patients with Acute COVID-19 Infection: The World Alliance Societies of Echocardiography (WASE-COVID) Study.”²

We agree with Dr. Dandel that the predominant cardiac manifestation of COVID-19 infection is right ventricular (RV) dysfunction, which can be due to a variety of mechanisms, including but not limited to direct angiotensin-converting enzyme 2–mediated myocardial injury from the SARS-CoV-2 virus, increased afterload from acute respiratory distress syndrome and pulmonary embolism, microvascular and macrovascular dysfunction associated with endothelitis, and negative inotropic effects of cytokines.³

The mechanism of RV injury proposed by Dr. Dandel that is related to infection-related pulmonary thrombotic microangiopathy resulting in severe hypoxemic respiratory failure and increased pulmonary vascular resistance⁴ is certainly plausible, particularly in the most critically ill patients with COVID-19 infection, but is unlikely to be the sole explanation for RV dysfunction in our patient population (only 46% in the intensive care unit).

In his letter, Dr. Dandel refers to the “positive predictive value (PPV) for COVID-19-related death.” We would like to point out that our paper reported odds ratios (as opposed to positive predictive value) for the continuous variables RV free wall strain (RVFWS) and RV global longitudinal strain, both of which were significantly associated with in-hospital mortality (not COVID-related death) in the univariate analysis, with RVFWS and left ventricular longitudinal strain (LVLS) found to be independently associated with in-hospital mortality in the multivariate analysis.

With regards to Dr. Dandel’s question about when strain-based imaging would be most useful, we would propose that given its increased availability and known value in risk stratification above and beyond ejection fraction, this imaging method would be particularly helpful in the triage of hospitalized patients with cardiac symptoms to determine which patients are at highest risk for decompensation and in-hospital mortality and to help guide further workup and management. For example, a reduced RVFWS may prompt further pulmonary workup to rule out pulmonary embolism or worsening lung disease, while a reduced LVLS may point to a cardiac etiology of the patient’s symptoms.

While the echocardiograms in our study were limited by safety concerns during the onset of the pandemic,⁵ now that most health care providers worldwide are vaccinated, we agree with Dr. Dandel

that given the paramount role of the right ventricle in the pathophysiology of COVID-19-related cardiac dysfunction, comprehensive assessment of the right heart, including cavity size, RV function, and spectral Doppler of the tricuspid valve to obtain pulmonary artery pressures, is critical in the assessment and triage of this patient population.

Interestingly, in our latest analysis of the WASE COVID-19 patient cohort, which included up to 9 months of echocardiographic follow-up, we found that in patients with baseline impaired LVLS, RVGLS, or increased RV basal diameter, there was significant improvement at time of follow-up.⁶ This suggests that cardiac abnormalities may improve over time and that echocardiography can potentially be used to monitor cardiac sequelae of COVID-19 infection.

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Conflicts of Interest: None.

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