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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:

I read with great interest the article by Karagodin et al., which confirmed the high incidence of cardiac dysfunction in hospitalized patients with COVID-19 and the usefulness of transthoracic echocardiography (TTE) for the management of patients with more severe COVID-19.

After repeated observations that left ventricular (LV) dysfunction was rarely a major cause for COVID-19-related heart failure and that most patients with high troponin had preserved LV function, considering the justifiable safety concerns, the role of TTE was at least initially quite controversial. Karagodin et al. also found no association between LV ejection fraction (LVEF) and in-hospital mortality, which is not surprising, because in 83% of their patients the LVEF was >50% (although 59% of the study population already had cardiac problems before COVID-19), whereas only 3% had an LVEF < 30%. Although the more sensitive LV longitudinal strain appeared to be independently associated with in-hospital mortality, its positive predictive value (PPV) for COVID-19-related death was low.

Much more frequent and life-threatening than the worsening of LV function appears to be a virus infection-associated extensive pulmonary thrombotic microangiopathy with severe hypoxemic respiratory failure, high resistance in the pulmonary circulation, and acute right ventricular (RV) failure, which emerged as a distinctive feature of COVID-19 with a major negative impact on patient survival. The significant correlations of D-dimer levels with pulmonary arterial systolic pressure (PAPs), RV dysfunction, and RV dilation plus the correlation between troponin levels with both PAPs and RV dysfunction underline the pathogenetic significance of the COVID-19-associated pulmonary microvascular thrombosis-induced RV afterload increase. Among hospitalized patients with COVID-19-related cardiac dysfunction without significant myocardial injury, 48% revealed isolated RV dysfunction. Karagodin et al. identified RV dilation in 33% of their study population, and the incidence of RV dysfunction was 50% higher compared with that of LV dysfunction.

Although in the study by Karagodin et al. both the RV free wall longitudinal strain and the basal RV diameter were found to be independently associated with in-hospital mortality, these highly load-dependent parameters revealed no relevant PPV for COVID-19-related death. This is not surprising because in severe COVID-19, pulmonary tissue damage and small-vessel thrombosis with consequent hypoxemia and excessively increased resistance in the pulmonary circulation are the cause of both acute respiratory failure and RV failure, which makes reliable prediction of mortality solely by RV dilation and/or RV dysfunction impossible.

Given the low PPV for death in relation to severe COVID-19 found in their study for both LV longitudinal strain and RV free wall longitudinal strain, plus the fact that a significant number of patients did not have sufficient images to assess the left or right ventricle (from their 402 patients treated at intensive care units, acceptable echocardiographic recordings for the left and right ventricle were obtainable only in 326 (81%) and 234 (58%) patients, respectively), can the authors identify scenarios where their imaging protocol is of benefit?

The guidelines for appropriate use of echocardiography should be applied in COVID-19 just as in routine practice. However, if there are indications for TTE in patients with COVID-19 (particularly in those with severe disease), it is particularly important to examine carefully the right heart (cavity dimensions, RV function, tricuspid regurgitation, and PAPs).

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