Phenotypic Characterization of Right Ventricular Dysfunction and Prognostication in COVID-19–Induced Acute Respiratory Distress Syndrome

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To the Editor:

The prognostication of coronavirus disease 2019 acute respiratory distress syndrome based on phenotypic characterization of the right ventricle (RV) by Chotalia et al (1) published in the recent issue of *Critical Care Medicine* unravels several interesting and paradoxical findings related to echocardiographic assessment of the systolic function of the RV and left ventricle (LV).

The dissociation between tricuspid annular plane systolic excursion (TAPSE) and right ventricular fractional area shortening (RV-FAC) needs worth attention. A decrease in TAPSE (longitudinal motion of annulus and free wall) has been consistently associated with a decrease in RV-FAC and systolic dysfunction (2, 3). A substantial reduction in RV-FAC in RV systolic dysfunction group despite a normal TAPSE and normal movement of the interventricular septum (good LV ejection fraction) appears indeed surprising.

All three RV phenotypes with variable grades of dilatation and systolic dysfunction had a normal LV systolic function. All three RV phenotypes may produce widely different LV end-diastolic volume, and LV contractility is likely to differ with a change in LV preload (4). RV dilatation with normal RV-FAC could have produced higher RV forward flow and LV end-diastolic volume than RV phenotypes with impaired systolic function or RV dilatation with normal function. Interestingly, RV dilatation with normal systolic function group required higher vasopressor or inotrope than RV systolic impairment with normal size (0.07 vs 0.01 μg/kg/min). Therefore, labeling RV dilatation with normal systolic function does not seem appropriate as inotropes and vasopressor could have improved RV-FAC and RV end-diastolic volume by increasing contractility and raising mean systemic filling pressure.

RV dilatation due to RV volume or pressure overload is invariably associated with an abnormal LV eccentricity index in systole and diastole. Surprisingly, the authors did not find a difference in the incidence of abnormal LV eccentricity in RV dilatation with normal systolic function group and normal RV size with systolic dysfunction group. Based on the incidence of abnormal LV eccentricity, it appears that in the majority of patients, volume or pressure overload was not present in these two phenotypes. In contrast, the peak tricuspid regurgitation velocity (2.7 m/s) indicates a definite pressure overload in the majority of the patients. Therefore, we believe that phenotypic characterization was not absolute and rigorous.

Defining systolic or diastolic dysfunction based on a single criterion may not be appropriate and are liable to misinterpretations (5). As TAPSE and RV-FAC

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are positively correlated except in patients with RV wall motion abnormality and the authors could have been stratified patients with RV systolic dysfunction based on the reduction in both TAPSE and RV-FAC. Furthermore, stratification between RV dilatation and normal RV size should have been based on both RV:LV end-diastolic area (RV:LVEDA) and abnormal LV eccentricity. Furthermore, the assessment of RV diastolic dysfunction could have helped refine of the phenotypic characterization of RV.

Like LV, systolic dysfunction may precede or follow volume overload. In regurgitant lesions, volume overload is generally followed by systolic dysfunction. Pressure overload may result in acute chamber dilatation and positive inotropy due to the Anrep effect in the beginning and, subsequently result in systolic and diastolic dysfunction (6). We suggest phenotypic characterization of RV dysfunction and RV dilatation based on minimum of two echocardiographic criteria for prognostication and patient-centric management. Therefore, RV systolic dysfunction should include TAPSE and RV-FAC, and RV dilatation should include abnormal LV eccentricity and RV:LVEDA based on the guidelines to improve prognostication and tailor patient-specific management. Additionally, like LV, incorporation of RV diastolic function assessment along with RV systolic function could have helped in better prognostication.

We believe that a larger sample size is required for our proposed phenotypic characterization, which could not have been possible in the study by Chotalia et al (1). However, phenotypic characterization based on at least two echocardiographic criteria to define systolic and diastolic dysfunction may obviate overlapping and misinterpretations.

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