Right Ventricular Dysfunction and Its Association With Mortality in Coronavirus Disease 2019 Acute Respiratory Distress Syndrome: Do Not Focus on Not Reliable Markers!

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To the Editor:

We read with great interest the recently published study by Chotalia et al (1) in *Critical Care Medicine*, which showed the great importance of right ventricle (RV) dysfunction or failure in coronavirus disease 2019-related acute respiratory distress syndrome. Authors report an occurrence rate of 49% of RV dysfunction in their cohort (172 patients) attested by RV dilatation (RV end-diastolic area/left ventricle [LV] diastolic area > 0.6, with or without septal dyskinesia). The main finding of their study is that RV dilatation with systolic impairment was independently associated with mortality, whereas either disease state alone was not (RV dilatation without systolic impairment or systolic impairment alone without RV dilatation). Several considerations must be addressed to moderate the authors' conclusion.

First, pathophysiology of RV failure in acute respiratory distress syndrome (ARDS) is an uncoupling of the RV systolic function with the pulmonary circulation, which induces RV dilatation and negative diastolic interaction with the LV due to ventricular competition for space within the indistensible pericardium, leading to the decrease of the cardiac output and the coronary blood flow with negative systolic interaction (2). Therefore, studying RV systolic markers could be relevant. However, authors defined RV systolic impairment with RV fractional area change (FAC) less than 35% or tricuspid annular plane systolic excursion (TAPSE) less than 17 mm. In their subgroup analysis, patients were mainly identified with systolic impairment due to low FAC. Due to complex RV geometry, RV-FAC, S′ wave, and TAPSE have been shown poor markers of RV failure (3) as defined by Vieillard-Baron et al (2). Indeed, the last-stage RV uncoupling with pulmonary circulation is acute cor pulmonale and is responsible for acute circulatory failure, one of the main causes of death in ARDS. It would have been interesting to give at list left cardiac index rather than instant vasopressor dose or instant urine output.

Secondly, transthoracic echocardiography was performed 6 days in median after the start of invasive ventilation, and only 172 patients other 268 could be evaluated, introducing a large selection bias and making difficult the interpretation of the results. Transesophageal echocardiography has been shown more accurate to evaluation of RV function (4) and, if performed, could have changed drastically the results.

Finally, all patients in the subgroup analysis did not have standard treatment like prone positioning (< 65% in the group RV dilatation with normal systolic function). Results could reflect only variation of care and support in context of large workload during pandemic, of just difference in the severity of the patients or comorbidities.

DOI: 10.1097/CCM.0000000000005328

Despite these comments, we strongly agree with the authors: evaluation of RV function in ARDS is one of the cornerstones of the care and support of such patients, and RV protective measures such as prone position, inhaled nitric oxide, and extracorporeal membrane oxygenation could improve prognosis. Measurement of RV free-wall strain could help identify an early RV dysfunction in ARDS, rather than RV systolic impairment markers.

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The authors have disclosed that they do not have any potential conflicts of interest.

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We thank Petit and Bidar (1) for their insightful comments on our article (2), recently published in *Critical Care Medicine*. Our aim was to identify a common right ventricular (RV) phenotype that associated with mortality, such that RV protective measures trialed in this subgroup may improve outcomes. The problem lies in defining this phenotype: should we use RV dilation, systolic impairment or both to classify RV dysfunction (RVD)?

American and British Echocardiography Societies and numerous critical care studies have defined RVD through RV systolic impairment (low RV fractional area change [RVFAC], tricuspid annular plane systolic excursion, and S′) with no consideration of RV size. However, as Petit and Bidar (1) highlight, due to the complex geometry of the RV, these parameters have been demonstrated as inaccurate markers of RV failure (3). In our study, patients with RV systolic impairment "without" RV dilation had a low mortality rate. This may be because the degree of RV systolic impairment was overestimated through using RVFAC when RV size was small. Newer markers of RV systolic function, such as RV free wall longitudinal strain, demonstrate promise (4), but are not routinely measured in current clinical practice.

If not RV systolic impairment, then what of RV dilation? Mekontso Dessap et al (5) demonstrated that RV dilation (RV:left ventricular end-diastolic area [LVEDA] > 0.6) with septal dyskinesia (termed acute cor pulmonale) did not independently associate with mortality in over 700 prospective acute respiratory distress syndrome (ARDS) patients. Our results support this:

DOI: 10.1097/CCM.0000000000005391