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EDITORIAL

Advanced Echocardiographic Techniques for the Diagnosis of Subclinical Right Ventricular Dysfunction in Chronic Obstructive Pulmonary Disease

Silvio Henrique Barberato[1]

Cardioeco, Centro de Diagnóstico Cardiovascular,[1] Curitiba, PR – Brazil

Editorial referring to the article: Chronic Obstructive Pulmonary Disease: The Role of Myocardial Deformation Indices and Right Ventricle Three-Dimensional Echocardiography

Chronic obstructive pulmonary disease (COPD) is a common and treatable disease in the general population, accounting for high morbidity and mortality rates.[1] In patients with severe COPD, hypoxemia is the main cause of complications such as pulmonary hypertension (PH) and right ventricle (RV) dysfunction.[2] However, the presence of RV vascular structural and functional changes may occur in the early stages of COPD, even before significant hypoxemia or PH.[3] Subclinical RV dysfunction often precedes the development of signs and symptoms of RV failure; thus, there is an evident need to detect it from the onset of the disease.[2]

The importance of the RV in cardiovascular physiology has been underestimated for decades. Previously considered a mere conduit, the chamber is now known to play a major role in maintaining global cardiac function intact. Impairment of RV systolic function is an essential determinant of clinical outcomes in different scenarios and should thus be considered in the individualized management of patients.[4,5]

Due to its wide availability, echocardiography is the most commonly used imaging test in clinical practice to assess RV size and function. However, that assessment can be hindered by the complex RV anatomy and method-specific limitations.[6] Hence, international societies of cardiovascular imaging have recommended the systematic addition of a multiparametric approach using several echocardiographic techniques and measurements to enable accurate analysis of RV function.[7] The strategy includes conventional parameters, such as RV basal diameter (normal ≤41 mm), tricuspid annular plane systolic excursion (TAPSE, normal ≥17 mm), and tissue Doppler-derived tricuspid lateral annular systolic velocity wave (s-wave, normal ≥9.5 cm/s), as well as advanced parameters, such as RV ejection fraction on tridimensional echocardiography (RVEF3D, normal ≥45%) and RV free wall longitudinal strain (RVFWLS, normal ≥20% in absolute value).[7] RVEF3D measurement reflects the RV systolic performance without the need for geometric assumption, resulting in greater accuracy and reproducibility, closer to the reference technique, cardiac magnetic resonance (CMR). RV strain calculated by two-dimensional speckle-tracking echocardiography (2D-STE) has been considered a superior alternative for early detection of RV systolic dysfunction because it has advantages over traditional echocardiographic techniques, such as lower angle dependency and less intra- and interobserver variability. International consensus has recommended that (1) RV strain should be measured using the specific RV focused apical four-chamber view to improve reproducibility; and (2) to report RVFWLS as a default parameter, but to allow the calculation of RV global longitudinal strain (RVGLS, which includes the ventricular septum) as an option.[8] The main limitations of the advanced techniques for evaluating RV function are the presence of poor acoustic thoracic window and arrhythmias, as well as, in the case of deformation indices, software variability of the different brands of equipment.[7]

In this issue of *International Journal of Cardiovascular Sciences*, Botelho et al.,[9] compared RV anatomy and systolic function assessed by traditional and advanced echocardiographic parameters in 20 patients, mostly older men with stable, moderate-to-severe COPD, and 20

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Mailing Address: Silvio Henrique Barberato
Cardioeco, Centro de Diagnóstico Cardiovascular. Avenida República Argentina, 1336, conj 215. Postal code: 80620-010, Curitiba, PR – Brazil.
E-mail: silviohb@cardiol.br

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controls.[9] Patients with COPD had significantly higher pulmonary artery systolic pressure and RV-free wall thickness values than controls. In contrast, they showed lower TAPSE, RVEF3D, and myocardial deformation values than controls. In 2D echocardiography, the RV linear dimensions did not differ significantly between the COPD and control groups. Only one (5%) COPD patient showed no RV-related echocardiographic abnormality. In two (10%) COPD patients, the diagnosis of RV systolic dysfunction was possible only by using advanced techniques (one with decreased RVGLS and one with decreased RVEF3D). The authors should be commended for including the measurement of RVEF3D in the study methodology. However, it was not possible to estimate RVEF3D in 3 (15%) COPD patients due to technical difficulties. In conclusion, their interesting results indicate overlooked RV dysfunction in clinically stable COPD patients, which is in line with previous reports.[3, 10]

Despite the rigorous methodology conducted by the study investigators, some issues must be addressed. The authors reported strain not only by 2D-STE, which is the current method of choice to assess myocardial deformation, but also by tissue Doppler imaging (TDI). It is worth noting that the TDI-derived strain indices appeared to be relatively unreliable for the analysis of RV function, as they had higher intra- and interobserver variability than RVGLS, RVEF3D, and even TAPSE. This is in agreement with prior clinical experience on the use of TDI for the estimation of myocardial strain, now relegated only to a historical role. Another issue to be highlighted is that the authors reported only RVGLS (including ventricular septum), but not RVFWLS (excluding the interventricular septum) in this study. Most studies have shown that RVFWLS has higher predictive power for diagnosing RV dysfunction,[10] and a more robust prognostic value in several cardiopulmonary disorders.[5] Furthermore, if the septum is included in the strain calculation, one should expect smaller values in magnitude than the RVFWLS. Finally, the lack of RV function analysis by MRI is another limitation that, although understandable, must be acknowledged.

The findings by Botelho et al.,[9] suggest that the use of advanced echocardiographic techniques for the detection of subclinical RV systolic dysfunction in stable COPD patients may select those who require careful surveillance and early intervention to prevent the development of RV failure and cardiovascular complications. Further studies are necessary to deepen the pathophysiological knowledge of RV dysfunction in the clinical context of COPD and to evaluate the role of therapeutic strategies to reduce mortality.

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