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EDITORIAL

Association of cardiovascular disease with COPD: cardiac function and structure evaluation

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COPD is defined as "a common, preventable, and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation."[1] COPD is the leading cause of morbidity and mortality worldwide, and it was the third leading cause of death globally in 2019. Therefore, COPD burdens the society and economy as a whole.[1] COPD-related mortality is frequently found to be associated with cardiovascular comorbidities.[2,3] Evidence suggests that mortality due to cardiovascular disease (CVD) is more dominant than those related to respiratory failure in patients with COPD.[4,5]

The association between COPD and CVD was suggested to be due to several factors, including shared risk factors (cigarette smoking, aging), symptom overlap (dyspnea, exercise limitation), and pathophysiological processes (systemic inflammation, increased oxidative stress).[2,5-8] The heightened inflammatory response to cigarette smoke in patients with COPD contributes to atherosclerotic plaque formation, which leads to coronary heart disease.[7,8] In acute COPD exacerbations, respiratory tract infection induces an acute inflammatory stimulation, potentially leading to a high risk for an acute cardiovascular event.[7,9] In stable COPD, a higher circulating level of systemic inflammatory biomarkers was found and associated with an increased risk of cardiac injury.[10]

Other than the abovementioned chronic inflammation, several characteristics of COPD affect cardiac function and structure, such as airflow limitation, hyperinflation, and pulmonary hypoxia.[5,7,11] Lung hyperinflation resulting from chronic airflow limitation, together with increased pulmonary vascular resistance from hypoxic vasoconstriction, could directly increase pressure in the pulmonary artery (pulmonary hypertension), leading to right ventricular diastolic dysfunction.[3,11] Chronic rising pulmonary pressure in long-standing COPD leads to right ventricle (RV) dilation and hypertrophy, which usually preserve stroke volume.[5,12] During a COPD acute exacerbation, a sudden increase in RV pressure load can lead to acute *cor pulmonale*, which presents with acute RV dilation and RV failure.[12,13] Hyperinflation can also affect cardiac dimension, resulting in decreased diastolic filling and reduced systolic ejection fraction.[11]

The importance of CVD comorbidity in COPD has been recognized, and integrated care was suggested.[14] Management of CVD comorbidity in COPD has been addressed in the GOLD guidelines.[1] It has been shown that cardiac abnormalities are highly prevalent during a COPD exacerbation regardless of established cardiac disease or cardiovascular risk factors.[13] Therefore, an echocardiogram has been recommended to assess cardiac function and structure for CVD in COPD during both acute and stable stages.[5,14] Echocardiograms reveal several abnormal cardiac functions and structures, such as left ventricle systolic and diastolic dysfunction, right and left ventricle enlargement, left atrial dilatation, and pulmonary hypertension.[15] A pitfall of performing an echocardiogram for CVD in COPD is that air trapping may impede the echocardiographic acoustic window resulting in unsatisfactory images.[16]

In this issue of the *Jornal Brasileiro de Pneumologia*, Pereira et al.[17] tried to contrast cardiac function and structure between patients with stable COPD and those with recent exacerbated COPD using echocardiogram. However, the results showed no differences in cardiac function or structure between recently exacerbated (one month prior) and stable COPD patients regardless of their clinical conditions.[17] The authors suggested that the similarities in cardiac function and structure in these two populations was because the patients with a recent COPD exacerbation had no significant respiratory failure to the point that it could cause changes in cardiac function. The authors further explained that, within 30 days after an exacerbation, the cardiac changes could have been transitory and went back to normal. However, in the absence of differences in echocardiographic results between the two groups, Pereira et al.[17] found impaired left ventricular function, increased left ventricular posterior wall thickness, and reduced mitral peak early/late diastolic filling velocity ratio, which is an index to assess diastolic filling. However, whether modifications in the left ventricular structure could affect left ventricular systolic and diastolic function is still controversial. The relative results were still inconclusive when compared with previous studies.[13,15]

Pereira et al.[17] also aimed to verify the association between cardiac structure and function with exercise capacity. The authors found that exercise capacity was associated with left ventricular posterior wall thickness and right atrial volume index, suggesting ventricular stiffness and increased filling pressure, which resulted in limited exercise capacity.[17] However, the effect of cardiac structure and function on exercise capacity was not shown in this study due to the type of analysis performed (e.g., Pearson correlation coefficient). The effect of cardiac structure and function on exercise capacity should have been evaluated using regression analysis by accounting for the degree of airway obstruction and cardiac function as confounding factors.

It is known that limited exercise capacity is one of the shared symptoms found in COPD and CVD.[14] However,

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evidence supporting the association between exercise capacity and cardiac function and structure is limited. In advanced emphysema, limited exercise capacity can be a result of airflow limitation, gas exchange impairment, and muscle depletion.[18] A study[15] involving 342 COPD patients hospitalized due to their first COPD exacerbation found no association between the six-minute walk distance and the presence of echocardiography abnormalities, whereas Schoos et al.[19] found that a longer six-minute walk distance independently correlated with milder tricuspid valve regurgitation.[15,19] More studies are required about the association between cardiac function and structure and exercise capacity in patients with concomitant COPD and CVD.

The presence of cardiovascular comorbidities in COPD has long been recognized, and their coexistence yields worse outcomes in comparison with each condition alone.[3,6] Even though the precise mechanism of the influence of these two diseases is not fully understood, the management of CVD comorbidities is implied in the GOLD guidelines.[1] Therefore, a thorough assessment of the signs and symptoms of both diseases is encouraged.[5,14] An echocardiogram is one of the assessment tools suggested to be useful and should be considered in concomitant COPD and CVD.[5,14,15] Information on cardiac function and structure could lead to a better understanding of the mechanism and subsequently facilitate an improvement in the disease management and therapeutic approach.

AUTHOR CONTRIBUTIONS

SD: drafting of the manuscript. SD, WC, and PT: conception, revision, and approval of the final version of the manuscript.

CONFLICTS OF INTEREST

None declared.

REFERENCES

1. Global Initiative for Chronic Obstructive Lung Disease [homepage on the Internet]. Bethesda: GOLD [cited 2022 Oct 14]. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease 2022 report. Available from: https://goldcopd.org
2. Trinkmann F, Saur J, Borggrefe M, Akin I. Cardiovascular Comorbidities in Chronic Obstructive Pulmonary Disease (COPD)-Current Considerations for Clinical Practice. J Clin Med. 2019;8(1):69. https://doi.org/10.3390/jcm8010069
3. Rabe KF, Hurst JR, Suissa S. Cardiovascular disease and COPD: dangerous liaisons? [published correction appears in Eur Respir Rev. 2018 Nov 21;27(150):]. Eur Respir Rev. 2018;27(149):180057. https://doi.org/10.1183/16000617.0057-2018
4. Berry CE, Wise RA. Mortality in COPD: causes, risk factors, and prevention. COPD. 2010;7(5):375-382. https://doi.org/10.3109/15412555.2010.510160
5. André S, Conde B, Fragoso E, Boléo-Tomé JP, Areias V, Cardoso J; et al. COPD and Cardiovascular Disease. Pulmonology. 2019;25(3):168-176. https://doi.org/10.1016/j.pulmoe.2018.09.006
6. Cavaillès A, Brinchault-Rabin G, Dixmier A, Goupil F, Gut-Gobert C, Marchand-Adam S, et al. Comorbidities of COPD. Eur Respir Rev. 2013;22(130):454-475. https://doi.org/10.1183/09059180.00008612
7. Morgan AD, Zakeri R, Quint JK. Defining the relationship between COPD and CVD: what are the implications for clinical practice?. Ther Adv Respir Dis. 2018;12:1753465817750524. https://doi.org/10.1177/1753465817750524
8. Roversi S, Roversi P, Spadafora G, Rossi R, Fabbri LM. Coronary artery disease concomitant with chronic obstructive pulmonary disease. Eur J Clin Invest. 2014;44(1):93-102. https://doi.org/10.1111/eci.12181
9. Donaldson GC, Hurst JR, Smith CJ, Hubbard RB, Wedzicha JA. Increased risk of myocardial infarction and stroke following exacerbation of COPD. Chest. 2010;137(5):1091-1097. https://doi.org/10.1378/chest.09-2029
10. Sin DD, Man SF. Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular diseases? The potential role of systemic inflammation in chronic obstructive pulmonary disease. Circulation. 2003;107(11):1514-1519. https://doi.org/10.1161/01.CIR.0000056767.69054.B3
11. Fragoso E, André S, Boleo-Tomé JP, Areias V, Munhá J, Cardoso J; et al. Understanding COPD: A vision on phenotypes, comorbidities and treatment approach. Rev Port Pneumol (2006). 2016;22(2):101-111. https://doi.org/10.1016/j.rppnen.2015.12.001
12. Bigatello LM, Leissner KB. Cor Pulmonale. In: Parsons PE, Wiener-Kronish JP, editors. Critical Care Secrets, 5th Ed. Philadelphia: Mosby; 2013. p. 157-165. https://doi.org/10.1016/B978-0-323-08500-7.00024-2
13. Pelà G, Li Calzi M, Pinelli S, Andreoli R, Sverzellati N, Bertorelli G, et al. Left ventricular structure and remodeling in patients with COPD. Int J Chron Obstruct Pulmon Dis. 2016;11:1015-1022. https://doi.org/10.2147/COPD.S102831
14. Roversi S, Fabbri LM, Sin DD, Hawkins NM, Agustí A. Chronic Obstructive Pulmonary Disease and Cardiac Diseases. An Urgent Need for Integrated Care. Am J Respir Crit Care Med. 2016;194(11):1319-1336. https://doi.org/10.1164/rccm.201604-0690SO
15. Freixa X, Portillo K, Paré C, Garcia-Aymerich J, Gomez FP, Benet M, et al. Echocardiographic abnormalities in patients with COPD at their first hospital admission [published correction appears in Eur Respir J. 2015 Nov;46(5):1533]. Eur Respir J. 2013;41(4):784-791. https://doi.org/10.1183/09031936.00222511
16. Hawkins NM, Petrie MC, Jhund PS, Chalmers GW, Dunn FG, McMurray JJ. Heart failure and chronic obstructive pulmonary disease: diagnostic pitfalls and epidemiology. Eur J Heart Fail. 2009;11(2):130-139. https://doi.org/10.1093/eurjhf/hfn013
17. Pereira MBCM, Castello-Simões V, Heubel AD, Kabbach EZ, Schafauser NS, Roscani MG, et al. Comparing cardiac function and structure and their relationship with exercise capacity between patients with stable COPD and recent acute exacerbation: a cross-sectional study. J Bras Pneumol. 2022;48(5):e20220098.
18. Visca D, Aiello M, Chetta A. Cardiovascular function in pulmonary emphysema. Biomed Res Int. 2013;2013:184678. https://doi.org/10.1155/2013/184678
19. Schoos MM, Dalsgaard M, Kjærgaard J, Moesby D, Jensen SG, Steffensen I, et al. Echocardiographic predictors of exercise capacity and mortality in chronic obstructive pulmonary disease. BMC Cardiovasc Disord. 2013;13:84. https://doi.org/10.1186/1471-2261-13-84