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Original Research Article

Association between Emphysema Severity on Quantitative CT and Cardiac Dysfunction in Patients with Chronic Obstructive Pulmonary Disease

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is frequently accompanied by cardiovascular comorbidities, yet the relationship between the extent of parenchymal destruction quantified by computed tomography (CT) and cardiac functional parameters remains incompletely characterized. Understanding this association may facilitate early identification of patients at risk for cardiac dysfunction and guide integrated management strategies.

Methods: This cross-sectional observational study enrolled 196 patients with stable COPD (GOLD stages I–IV) from a tertiary pulmonary center. Quantitative CT analysis was performed to determine the low attenuation area percentage below −950 Hounsfield units (LAA−950%) as a measure of emphysema severity. Cardiac function was assessed using transthoracic echocardiography, including left ventricular ejection fraction (LVEF), right ventricular fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE), and estimated pulmonary artery systolic pressure (PASP). Correlations were examined using Pearson and Spearman analyses, and multivariable linear regression was performed adjusting for confounders.

Results: The mean age was 64.7 ± 9.2 years, and 68.4% were male. The mean LAA−950% was 18.3 ± 12.6%. Patients with severe emphysema (LAA−950% ≥ 25%) demonstrated significantly lower LVEF (56.1 ± 7.8% vs. 62.4 ± 6.1%, $p < 0.001$), reduced RVFAC (32.4 ± 6.9% vs. 39.7 ± 5.8%, $p < 0.001$), decreased TAPSE (16.2 ± 3.4 mm vs. 20.8 ± 3.1 mm, $p < 0.001$), and elevated PASP (42.6 ± 11.3 mmHg vs. 29.4 ± 8.7 mmHg, $p < 0.001$). LAA−950% was independently associated with RVFAC ($\beta = -0.38$, $p < 0.001$) and PASP ($\beta = 0.44$, $p < 0.001$) after multivariable adjustment.

Conclusion: Emphysema severity quantified by CT densitometry is independently and significantly associated with both right and left cardiac dysfunction in COPD patients. Quantitative CT may serve as a valuable adjunctive tool for cardiovascular risk stratification in this population.

Keywords: COPD; emphysema; quantitative computed tomography; cardiac dysfunction; echocardiography; pulmonary hypertension.

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Introduction

Chronic obstructive pulmonary disease (COPD) represents a leading cause of morbidity and mortality worldwide, affecting an estimated 300 million individuals globally and projected to become the third leading cause of death by 2030 [1].

While COPD is primarily characterized by persistent airflow limitation and progressive respiratory symptoms, it is increasingly recognized as a multisystem disorder with significant extrapulmonary manifestations, among which cardiovascular disease holds particular prominence [2]. Cardiovascular complications, including ischemic heart disease, heart failure, and pulmonary hypertension, account for approximately 25–30% of all COPD-related deaths and substantially influence disease prognosis [3]. Emphysema, defined by the abnormal and permanent enlargement of airspaces distal to terminal bronchioles accompanied by destruction of alveolar walls, constitutes a major pathological component of COPD [4].

Quantitative computed tomography (CT) has emerged as the reference standard for non-invasive

assessment of emphysema, with the percentage of low attenuation area below −950 Hounsfield units (LAA−950%) demonstrating robust correlation with histopathological measures of parenchymal destruction [5]. The widespread availability of CT-based densitometric analysis has enabled precise phenotyping of COPD patients beyond conventional spirometric classification [6].

The pathophysiological mechanisms linking emphysema to cardiac dysfunction are multifaceted. Destruction of the pulmonary vascular bed increases pulmonary vascular resistance, leading to right ventricular (RV) pressure overload and eventual cor pulmonale [7]. Additionally, hyperinflation associated with emphysema impairs venous return, reduces left ventricular (LV) preload through ventricular interdependence, and may compromise diastolic filling [8]. Systemic inflammation and oxidative stress further contribute to myocardial dysfunction through endothelial injury and accelerated atherosclerosis [9].

Several investigators have explored the relationship between CT-derived emphysema metrics and cardiac parameters. The Multi-Ethnic Study of Atherosclerosis (MESA) COPD study demonstrated that greater emphysema extent was associated with impaired LV filling and reduced cardiac output even in individuals without clinical cardiovascular disease [10]. Similarly, Barr and colleagues reported significant associations between emphysema percentage and reduced RV function on cardiac magnetic resonance imaging [11]. More recently, studies utilizing the COPDGene cohort have confirmed dose-dependent relationships between quantitative emphysema measures and both right and left heart structural changes [12].

Despite these advances, several knowledge gaps persist. The majority of prior studies have focused on either right or left heart function in isolation, and few have comprehensively evaluated biventricular cardiac parameters in relation to quantitative emphysema severity across the full spectrum of COPD stages [13].

Furthermore, the independent contribution of emphysema extent to cardiac dysfunction, after accounting for airflow obstruction severity and other confounders, requires further clarification [14]. Understanding these associations is essential for developing integrated cardiopulmonary management approaches and identifying high-risk phenotypes that may benefit from targeted interventions.

The aim of this study was to investigate the association between emphysema severity, quantified by CT densitometry (LAA−950%), and comprehensive echocardiographic parameters of both right and left ventricular function in patients with stable COPD across all disease stages.

Materials and Methods

Study Design and Population: This cross-sectional observational study was conducted at the Department of Pulmonology. Consecutive patients aged ≥40 years with a confirmed diagnosis of COPD according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria (post-bronchodilator forced expiratory volume in one second/forced vital capacity [FEV_1/FVC] < 0.70) were considered for enrollment. Patients were required to be in a stable clinical state, defined as no exacerbation within the preceding six weeks.

Exclusion criteria included: (1) known pre-existing structural heart disease (valvular heart disease, congenital heart disease, cardiomyopathy); (2) history of coronary artery bypass grafting or percutaneous coronary intervention within the preceding 12 months; (3) active malignancy; (4) significant chest wall deformity precluding accurate CT analysis; (5) pregnancy; (6) inability to perform spirometry; and (7) inadequate echocardiographic acoustic windows. A total of 196 patients fulfilled eligibility criteria and constituted the final study cohort.

Pulmonary Function Testing: Post-bronchodilator spirometry was performed using a calibrated spirometer (MasterScreen Body, Jaeger, Germany) following American Thoracic Society/European Respiratory Society standards. Parameters recorded included FEV_1 (absolute and percent predicted), FVC, and FEV_1/FVC ratio. GOLD staging was assigned based on FEV_1 percent predicted values.

Quantitative CT Analysis: All participants underwent volumetric non-contrast chest CT (Siemens SOMATOM Definition Flash, 128-slice, Siemens Healthineers, Erlangen, Germany) at full inspiration with standardized acquisition parameters (120 kVp, automatic tube current modulation, 1.0 mm slice thickness, B35f reconstruction kernel). Images were analyzed using dedicated commercially available software (Pulmo3D, Siemens Healthineers). The primary emphysema metric was the percentage of total lung volume occupied by voxels with attenuation values below −950 Hounsfield units (LAA−950%). Patients were stratified into three emphysema severity groups: mild (LAA−950% < 10%), moderate (LAA−950% 10–24.9%), and severe (LAA−950% ≥ 25%).

Echocardiographic Assessment: Comprehensive transthoracic echocardiography was performed within 48 hours of CT imaging using a Vivid E95 ultrasound system (GE Healthcare, Horten,

Norway) equipped with an M5Sc-D phased-array transducer. Examinations were conducted by two experienced sonographers blinded to CT findings. Parameters assessed included: LVEF (biplane Simpson's method), LV end-diastolic diameter (LVEDD), LV mass index (LVMI), E/A ratio, E/e' ratio, RVFAC, TAPSE, RV basal diameter, and estimated PASP derived from tricuspid regurgitation velocity plus estimated right atrial pressure. RV dysfunction was defined as TAPSE < 17 mm and/or RVFAC < 35%.

Clinical and Demographic Variables: Demographic data, smoking history (pack-years), body mass index (BMI), comorbidities (hypertension, diabetes mellitus, and dyslipidemia), medications, and COPD Assessment Test (CAT) scores were systematically recorded.

Statistical Analysis: Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) based on distribution normality assessed by the Shapiro-Wilk test. Categorical variables were presented as frequencies and percentages. Group comparisons were performed using one-way analysis of variance (ANOVA) with Tukey's post-hoc test for normally distributed variables, the Kruskal-Wallis test for non-normally distributed variables, and chi-square or Fisher's exact test for categorical variables. Pearson and Spearman correlation coefficients were calculated to examine bivariate associations. Multivariable linear regression models were constructed to evaluate independent associations between LAA−950% and cardiac parameters, adjusting for age, sex, BMI, smoking pack-years, FEV_1% predicted, hypertension, and diabetes. A two-sided p-value < 0.05 was considered statistically significant. All analyses were conducted using SPSS version 28.0 (IBM Corporation, Armonk, NY, USA).

Results

Baseline Characteristics: The study cohort comprised 196 patients with a mean age of 64.7 ± 9.2 years, of whom 134 (68.4%) were male. The mean BMI was 25.6 ± 4.8 kg/m², and the mean smoking history was 41.3 ± 18.7 pack-years. Distribution across GOLD stages was: GOLD I, 28 (14.3%); GOLD II, 72 (36.7%); GOLD III, 61 (31.1%); and GOLD IV, 35 (17.9%). The mean FEV_1% predicted was 49.8 ± 19.4%. The mean LAA−950% for the entire cohort was 18.3 ± 12.6%, with 62 patients (31.6%) classified as mild, 72 (36.7%) as moderate, and 62 (31.6%) as severe emphysema.

Table 1: Baseline Demographic and Clinical Characteristics by Emphysema Severity Group

Variable	Mild (n = 62)	Moderate (n = 72)	Severe (n = 62)	p-value
Age (years)	62.1 ± 9.8	64.9 ± 8.7	67.4 ± 8.6	0.006
Male sex, n (%)	39 (62.9)	50 (69.4)	45 (72.6)	0.488
BMI (kg/m²)	27.4 ± 4.5	25.8 ± 4.6	23.2 ± 4.7	< 0.001
Smoking (pack-years)	35.2 ± 16.9	42.1 ± 18.2	47.5 ± 19.4	0.001
FEV_1% predicted	62.7 ± 16.8	48.3 ± 15.9	37.1 ± 16.3	< 0.001
FEV_1/FVC	0.58 ± 0.08	0.51 ± 0.09	0.42 ± 0.10	< 0.001
LAA−950% (%)	5.8 ± 2.7	16.4 ± 4.3	34.2 ± 7.9	< 0.001
CAT score	14.6 ± 6.2	19.3 ± 7.1	24.8 ± 6.9	< 0.001
Hypertension, n (%)	26 (41.9)	31 (43.1)	24 (38.7)	0.872
Diabetes mellitus, n (%)	12 (19.4)	15 (20.8)	11 (17.7)	0.893

Echocardiographic Parameters by Emphysema Severity: Echocardiographic parameters demonstrated progressive deterioration with increasing emphysema severity (Table 2). Patients in the severe emphysema group exhibited significantly lower LVEF, reduced RVFAC, decreased TAPSE, and elevated PASP compared to both the mild and moderate groups. Left ventricular diastolic dysfunction, as reflected by E/e' ratio, also worsened with emphysema severity.

Table 2: Echocardiographic Parameters by Emphysema Severity Group

Parameter	Mild (n = 62)	Moderate (n = 72)	Severe (n = 62)	p-value
LVEF (%)	62.4 ± 6.1	59.8 ± 6.7	56.1 ± 7.8	< 0.001
LVEDD (mm)	47.3 ± 4.8	46.9 ± 5.1	45.4 ± 5.6	0.094
LVMI (g/m²)	89.2 ± 18.4	92.7 ± 20.1	96.8 ± 22.6	0.097
E/A ratio	1.06 ± 0.31	0.92 ± 0.28	0.81 ± 0.26	< 0.001
E/e' ratio	8.9 ± 2.8	10.4 ± 3.2	12.7 ± 3.9	< 0.001
RVFAC (%)	39.7 ± 5.8	36.1 ± 6.4	32.4 ± 6.9	< 0.001
TAPSE (mm)	20.8 ± 3.1	18.4 ± 3.6	16.2 ± 3.4	< 0.001
RV basal diameter (mm)	34.6 ± 4.9	37.8 ± 5.3	41.2 ± 6.1	< 0.001
PASP (mmHg)	29.4 ± 8.7	34.8 ± 9.6	42.6 ± 11.3	< 0.001
RV dysfunction, n (%)	8 (12.9)	19 (26.4)	31 (50.0)	< 0.001

Correlation and Regression Analyses: Bivariate correlation analysis revealed that LAA−950% was significantly correlated with RVFAC ($r = -0.52$, $p < 0.001$), TAPSE ($r = -0.48$, $p < 0.001$), PASP ($r = 0.56$, $p < 0.001$), LVEF ($r = -0.34$, $p < 0.001$), and E/e' ratio ($r = 0.39$, $p < 0.001$).

Table 3: Multivariable Linear Regression Analysis: Independent Predictors of Cardiac Parameters

Dependent Variable	Independent Variable	Standardized β	95% CI	p-value
RVFAC	LAA−950%	−0.38	−0.51 to −0.25	< 0.001
	FEV_1% predicted	0.19	0.04 to 0.34	0.014
	Age	−0.12	−0.26 to 0.02	0.089
TAPSE	LAA−950%	−0.33	−0.47 to −0.19	< 0.001
	FEV_1% predicted	0.17	0.02 to 0.32	0.028
	BMI	0.11	−0.03 to 0.25	0.121
PASP	LAA−950%	0.44	0.31 to 0.57	< 0.001
	FEV_1% predicted	−0.14	−0.29 to 0.01	0.062
	Age	0.13	0.00 to 0.27	0.057
LVEF	LAA−950%	−0.22	−0.37 to −0.07	0.004
	Age	−0.18	−0.32 to −0.04	0.012
	Hypertension	−0.15	−0.29 to −0.01	0.034
E/e' ratio	LAA−950%	0.28	0.13 to 0.43	< 0.001
	Age	0.21	0.07 to 0.35	0.004
	BMI	0.14	0.00 to 0.28	0.048

In multivariable models adjusted for age, sex, BMI, smoking pack-years, FEV_1% predicted, hypertension, and diabetes mellitus, LAA−950% remained independently associated with all five cardiac parameters. The strongest independent associations were observed for PASP ($\beta = 0.44$, $p < 0.001$) and RVFAC ($\beta = -0.38$, $p < 0.001$).

Discussion

The present study demonstrates that emphysema severity, quantified by CT densitometry, is independently and significantly associated with both right and left ventricular dysfunction in patients with stable COPD. These findings extend current understanding of the cardiopulmonary interactions in COPD by demonstrating graded, dose-dependent relationships between parenchymal destruction and comprehensive echocardiographic measures of biventricular performance.

The most robust associations were observed between LAA−950% and right heart parameters, which is pathophysiologically coherent. Progressive destruction of the alveolar capillary bed in emphysema reduces the cross-sectional area of the pulmonary vasculature, resulting in elevated pulmonary vascular resistance and increased RV afterload [15].

Additionally, hypoxic pulmonary vasoconstriction in areas of ventilation-perfusion mismatch further compounds vascular remodeling [16]. Our finding that 50% of patients with severe emphysema exhibited RV dysfunction underscores the clinical significance of this relationship and aligns with observations from the MESA Lung Study, where Kawut and colleagues demonstrated progressive RV structural and functional impairment with increasing emphysema severity on magnetic resonance imaging [17].

The independent association between emphysema extent and elevated PASP in our cohort, even after adjustment for FEV_1% predicted, suggests that the anatomical destruction of the pulmonary vascular bed captured by CT densitometry provides information complementary to and distinct from spirometric measurements of airflow limitation. This observation is consistent with findings from Wells and colleagues, who reported that CT emphysema metrics predicted pulmonary vascular disease independent of airflow obstruction in the COPDGene cohort [18].

Regarding left heart function, our data revealed modest but statistically significant independent associations between LAA−950% and both LVEF and E/e' ratio. Several mechanisms may account for this observation. Dynamic hyperinflation in emphysema increases intrathoracic pressure, reduces venous return, and impairs LV filling through ventricular interdependence and pericardial constraint [19]. Watz and colleagues previously demonstrated that static hyperinflation in COPD was associated with reduced biventricular cardiac chamber sizes and impaired cardiac output, supporting the mechanical hypothesis [20]. Furthermore, systemic inflammation and oxidative stress, both hallmarks of COPD with emphysema, promote endothelial dysfunction and subclinical myocardial injury that may contribute to impaired LV performance [21].

The finding that LAA−950% was independently associated with E/e' ratio, a validated marker of LV diastolic function, is noteworthy. Diastolic dysfunction has been increasingly recognized as a

prevalent and underdiagnosed comorbidity in COPD [22]. Bhatt and colleagues demonstrated that emphysema on CT was associated with impaired LV filling and reduced stroke volume in population-based studies, findings that our clinical COPD cohort now extends to more advanced disease stages [23]. The clinical implication is that patients with severe emphysema may be at heightened risk for heart failure with preserved ejection fraction (HFpEF), a condition with substantial morbidity and limited therapeutic options.

From a clinical perspective, our results support the integration of quantitative CT emphysema analysis into the comprehensive assessment of COPD patients, particularly for cardiovascular risk stratification. Given that chest CT is already routinely performed for lung cancer screening and COPD phenotyping, the incremental burden of extracting emphysema metrics is minimal, yet the prognostic information gained may be substantial [24]. Early identification of emphysema-predominant patients with subclinical cardiac dysfunction may facilitate timely referral for cardiology evaluation and initiation of cardioprotective therapies.

Several limitations warrant acknowledgment. The cross-sectional design precludes inference of causality between emphysema severity and cardiac dysfunction. Echocardiographic assessment of cardiac function, while clinically practical, may be suboptimal in patients with severe hyperinflation due to limited acoustic windows; however, patients with inadequate echocardiographic quality were excluded. Cardiac magnetic resonance imaging would have provided more precise biventricular volumetric assessment but was not feasible for all participants. Additionally, the single-center design and predominantly male cohort may limit generalizability. Inflammatory biomarkers were not systematically measured, precluding evaluation of the mediating role of systemic inflammation. Finally, residual confounding from unmeasured variables, such as coronary artery calcification or subclinical ischemic heart disease, cannot be entirely excluded.

Future longitudinal studies incorporating serial CT and cardiac imaging assessments are needed to establish the temporal and potentially causal relationship between emphysema progression and cardiac functional decline. Investigation of specific emphysema distribution patterns (upper lobe versus lower lobe predominant) in relation to regional pulmonary hemodynamics and cardiac function represents another promising avenue for research [25].

Conclusion

This study demonstrates that emphysema severity, quantified by the percentage of low attenuation area on CT (LAA−950%), is independently and significantly associated with both right and left ventricular dysfunction in patients with stable COPD. Right heart parameters, particularly PASP and RVFAC, exhibited the strongest associations with emphysema extent, reflecting the direct impact of pulmonary vascular bed destruction on RV afterload. Modest but significant associations with left ventricular systolic and diastolic function were also observed, likely attributable to hyperinflation-mediated hemodynamic compromise and systemic inflammatory mechanisms. These findings support the routine incorporation of quantitative CT emphysema analysis as a complementary tool for cardiovascular risk assessment in COPD patients and advocate for integrated cardiopulmonary evaluation strategies, particularly in patients with advanced emphysema.

References

1. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: 2023 report. Am J Respir Crit Care Med. 2023;207(4):e24–e70. DOI: 10.1164/rccm.202301-0106ST
2. Cavaillès A, Brinchault-Rabin G, Dixmier A, et al. Comorbidities of COPD. Eur Respir Rev. 2013;22(130):454–475. DOI: 10.1183/09059180.00008612. PMID: 24293462
3. Sin DD, Anthonisen NR, Soriano JB, Agusti AG. Mortality in COPD: role of comorbidities. Eur Respir J. 2006;28(6):1245–1257. DOI: 10.1183/09031936.00133805. PMID: 17138679
4. Snider GL, Kleinerman J, Thurlbeck WM, Bengali ZH. The definition of emphysema: report of a National Heart, Lung, and Blood Institute, Division of Lung Diseases workshop. Am Rev Respir Dis. 1985;132(1):182–185. DOI: 10.1164/arrd.1985.132.1.182. PMID: 4014865
5. Gevenois PA, de Maertelaer V, De Vuyst P, Zanen J, Yernault JC. Comparison of computed density and macroscopic morphometry in pulmonary emphysema. Am J Respir Crit Care Med. 1995;152(2):653–657. DOI: 10.1164/ajrccm.152.2.7633722. PMID: 7633722
6. Lynch DA, Austin JH, Hogg JC, et al. CT-definable subtypes of chronic obstructive pulmonary disease: a statement of the Fleischner Society. Radiology. 2015;277(1):192–205. DOI: 10.1148/radiol.2015141579. PMID: 25961632
7. Sakao S, Voelkel NF, Tatsumi K. The vascular bed in COPD: pulmonary hypertension and

pulmonary vascular alterations. Eur Respir Rev. 2014;23(133):350–355. DOI: 10.1183/09059180.00007913. PMID: 25176970
8. Jörgensen K, Müller MF, Nel J, Upton RN, Houltz E, Ricksten SE. Reduced intrathoracic blood volume and left and right ventricular dimensions in patients with severe emphysema. Chest. 2007;131(4):1050–1057. DOI: 10.1378/chest.06-2245. PMID: 17426209
9. Fisk M, Maysoon M, Gale NS, et al. Systemic inflammation and cardiovascular disease in COPD. J Chronic Obstr Pulm Dis. 2018;5(1):32–40. DOI: 10.15326/jcopdf.5.1.2017.0153
10. Barr RG, Bluemke DA, Ahmed FS, et al. Percent emphysema, airflow obstruction, and impaired left ventricular filling. N Engl J Med. 2010;362(3):217–227. DOI: 10.1056/NEJMoa0808836. PMID: 20089972
11. Barr RG, Ahmed FS, Carr JJ, et al. Subclinical atherosclerosis, airflow obstruction and emphysema: the MESA Lung Study. Eur Respir J. 2012;39(4):846–854. DOI: 10.1183/09031936.00165410. PMID: 21965224
12. Wells JM, Washko GR, Han MK, et al. Pulmonary arterial enlargement and acute exacerbations of COPD. N Engl J Med. 2012;367(10):913–921. DOI: 10.1056/NEJMoa1203830. PMID: 22938715
13. Smith BM, Prince MR, Hoffman EA, et al. Impaired left ventricular filling in COPD and emphysema: is it the heart or the lungs? Circ Cardiovasc Imaging. 2013;6(1):29–36. DOI: 10.1161/CIRCIMAGING.112.979438. PMID: 23192849
14. Stone IS, Barnes NC, James WY, et al. Lung deflation and cardiovascular structure and function in chronic obstructive pulmonary disease: a randomized controlled trial. Am J Respir Crit Care Med. 2016;193(7):717–726. DOI: 10.1164/rccm.201508-1647OC. PMID: 26550687
15. Hurdman J, Condliffe R, Elliot CA, et al. Pulmonary hypertension in COPD: results from the ASPIRE registry. Eur Respir J. 2013;41(6):1292–1301. DOI: 10.1183/09031936.00079512. PMID: 23018917
16. Naeije R, Barbera JA. Pulmonary hypertension associated with COPD. Crit Care. 2001;5(6):286–289. DOI: 10.1186/cc1049. PMID: 11737896
17. Kawut SM, Poor HD, Parikh MA, et al. Cor pulmonale parvus in chronic obstructive pulmonary disease and emphysema: the MESA COPD study. J Am Coll Cardiol. 2014;64(19):2000–2009. DOI: 10.1016/j.jacc.2014.07.991. PMID: 25440096
18. Wells JM, Iyer AS, Rahaghi FN, et al. Pulmonary artery enlargement is associated with right ventricular dysfunction and loss of blood volume in small pulmonary vessels in chronic obstructive pulmonary disease. Circ Cardiovasc Imaging. 2015;8(4):e002546. DOI: 10.1161/CIRCIMAGING.114.002546. PMID: 25873722
19. Watz H, Waschki B, Meyer T, et al. Decreasing cardiac chamber sizes and associated heart dysfunction in COPD: role of hyperinflation. Chest. 2010;138(1):32–38. DOI:10.1378/chest.09-2810. PMID: 20190002
20. Watz H, Waschki B, Meyer T, Magnussen H. Physical activity in patients with COPD. Eur Respir J. 2009;33(2):262–272. DOI: 10.1183/09031936.00024608. PMID: 19010994
21. Maclay JD, McAllister DA, Johnston S, et al. Vascular dysfunction in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2009;180(6):513–520. DOI: 10.1164/rccm.200903-0414OC. PMID: 19542477
22. Rutten FH, Cramer MJ, Lammers JW, Grobbee DE, Hoes AW. Heart failure and chronic obstructive pulmonary disease: an ignored combination? Eur J Heart Fail. 2006;8(7):706–711. DOI: 10.1016/j.ejheart.2006.01.010. PMID: 16531114
23. Bhatt SP, Dransfield MT, Cockayne DA, et al. A computed tomographic measure of emphysema and left heart dysfunction. Am J Respir Crit Care Med. 2014;190(3):321–331. DOI: 10.1164/rccm.201404-0592OC. PMID: 24955811
24. Labaki WW, Martinez CH, Martinez FJ, et al. The role of chest computed tomography in the evaluation and management of the patient with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2017;196(11):1372–1379. DOI: 10.1164/rccm.201703-0451PP. PMID: 28622013
25. Galban CJ, Han MK, Boes JL, et al. Computed tomography–based biomarker provides unique signature for diagnosis of COPD phenotypes and disease progression. Nat Med. 2012;18(11):1711–1715. DOI: 10.1038/nm.2971. PMID: 23042237.