Heliyon 10 (2024) e27683

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon

Research article

Differential decline of lung function in COPD patients according to structural abnormality in chest CT

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ARTICLE INFO

Keywords:
Forced expiratory volume
Respiratory function tests
Bronchiectasis
Pulmonary emphysema
Pulmonary disease
Chronic obstructive
Cohort studies

ABSTRACT

Background: Different progressions or prognoses of chronic obstructive pulmonary disease (COPD) have been reported according to structural abnormalities based on chest computed tomography (CT). This study aimed to investigate whether different structural abnormalities independently affect annual lung function changes and clinical prognosis in patients with COPD.

Methods: This longitudinal multicenter observational study was conducted using the KOCOSS cohort (NCT02800499) database in Korea from January 2012 to December 2019. For COPD patients with chest CT findings at baseline enrolment and longitudinal spirometric data, annual forced expiratory volume in 1 s (FEV_1) decline rate (mL/year) and clinical outcomes were compared according to structural abnormalities, including emphysema, bronchiectasis (BE), and tuberculosis-destroyed lung (TDL). We estimated the adjusted annual FEV_1 changes using a mixed-effect linear regression model.

Results: Among the enrolled 237 patients, 152 showed structural abnormalities. Emphysema, BE, and TDL were observed in 119 (78.3%), 28 (18.4%), and 27 (17.8%) patients, respectively. The annual decline in FEV_1 was faster in COPD patients with structural abnormalities than those without ($\beta = -70.6$ mL/year, P-value = 0.039). BE/TDL-dominant or emphysema-dominant structural abnormality contributed to an accelerated annual FEV_1 decline compared to no structural abnormality (BE/TDL-dominant, $\beta = -103.7$ mL/year, P-value = 0.043; emphysema-dominant, $\beta = -84.1$ mL/year, P-value = 0.018). Structural abnormalities made no significant differences in acute exacerbation rate and mortality.

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https://doi.org/10.1016/j.heliyon.2024.e27683
Received 7 June 2023; Received in revised form 2 March 2024; Accepted 5 March 2024
Available online 12 March 2024

Conclusion: The lung function decline rate in COPD differed according to structural abnormalities on CT. These findings may suggest that more focus should be placed on earlier intervention or regular follow-up with spirometry in COPD patients with BE or TDL on chest CT.

1. Introduction

1.1. Background

Chronic obstructive pulmonary disease (COPD) is characterized by irreversible airflow limitation, causing progressive loss of lung function, reduced quality of life, and increased risk of acute exacerbation and mortality [1]. The clinical features of COPD often vary because the diagnosis is mainly based on lung function impairment, regardless of the cause or mechanism of disease development. Indeed, different lung function decline rates, response to treatment, and clinical prognosis have been reported in patients with COPD [2,3]. Recently, several subgroups based on clinical phenotypes have been identified in COPD patients. A personalized approach based on different COPD subtypes is now suggested to achieve a better response to treatment [4].

Previous studies have reported different progression or prognosis of COPD according to structural abnormalities, based on high-resolution chest computed tomography (CT) [5,6]. The increase in bronchial wall area or thickness is associated with a higher risk of acute exacerbation [7] and mortality [8], and reduced lung function [9]. The increase in bronchial wall area or thickness is associated with bronchiectasis due to the chronic inflammatory processes that contribute to structural changes in the airways. A history of tuberculosis is considered one of the main causes of bronchiectasis [10]. Bronchiectasis is significantly associated with poor prognosis in COPD patients [11]. Another structural abnormality in COPD is emphysema, resulting from alveolar and parenchymal destruction due to an imbalance between proteases and antiproteases [12]. There were differences in lung function changes [13] and mortality [8] according to the pattern and extent of emphysema in patients with COPD. However, there remains insufficient evidence to classify COPD patients with structural abnormalities into distinct subtypes. There are limited longitudinal studies on the differences in lung function decline rate and clinical prognosis according to different structural abnormalities in patients with COPD.

Therefore, we aimed to investigate whether different structural abnormalities independently affect annual lung function changes and clinical prognosis in patients with COPD.

2. Material and methods

Our study followed the guidelines of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [14].

2.1. Study design and eligibility criteria

This retrospective longitudinal study was performed using the Korea COPD Subgroup Study (KOCOSS) cohort database (NCT02800499), which prospectively enrolled patients diagnosed with COPD from January 2012 to December 2019 at 54 hospitals in South Korea. Detailed information on the methodology of the KOCOSS was described in a previous study [15]. COPD was diagnosed based on the spirometric criteria in the Global Initiative for Chronic Obstructive Lung Disease reports, which is a post-bronchodilator annual forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) ratio <0.7 [1]. The eligibility criteria were as follows: 1) age $\geq$40 years, 2) available information on baseline chest CT scan, 3) spirometric evaluation at baseline examination, and 4) follow-up with spirometric examination for 3 years.

2.2. Baseline information and lung function

In the baseline assessment, we obtained the demographic information of the eligible subjects, including age, sex, body mass index (BMI), smoking status, Charlson comorbidity index, and previous history of asthma. We acquired information on baseline symptoms or quality of life, including the COPD assessment test (CAT) score, St George's Respiratory Questionnaire for COPD Patients (SGRQ-C) score, and 6-min walking distance. We identified a history of acute exacerbations, including total and severe exacerbations.

Baseline spirometric information was obtained, including post-bronchodilator FEV_1 and FVC (mL and % of the predicted value), diffusing capacity for carbon monoxide (DLCO) (%), DLCO/alveolar volume (VA) (%), and total lung capacity (mL and % of the predicted value). Regarding radiologic evaluation, we investigated structural abnormalities, including emphysema, bronchiectasis, and tuberculosis-destroyed lung (TDL), on chest CT. The structural abnormalities were identified based on the radiologist's reports.

2.3. Study outcomes

The primary outcome was the annual change in post-bronchodilator FEV_1 (mL/year) according to different structural abnormalities. The secondary outcome was the comparison between the 3-year total number of moderate-to-severe exacerbations, the annual rate of moderate-to-severe exacerbations, and mortality events according to different structural abnormalities.

2.4. Patient groups

Eligible patients were classified into two groups according to the radiological structural abnormalities on chest CT. Structural abnormalities on chest CT include bronchiectasis, TDL, and emphysema. TDL is commonly accompanied by bronchiectasis, and it is often difficult to clearly distinguish which is predominant between TDL and bronchiectasis in clinical practice. Therefore, the radiologic features of bronchiectasis or TDL were grouped together and defined as bronchiectasis or TDL (BE/TDL)-dominant structural abnormalities. The radiological features of emphysema were defined as emphysema-dominant structural abnormalities. Patients with both BE/TDL-dominant and emphysema-dominant structural abnormalities were excluded from the comparative analyses between TDL-dominant and emphysema-dominant structural abnormalities.

2.5. Statistical analyses

We used Student's t-test or Wilcoxon rank-sum test to compare continuous variables, and chi-square test or Fisher's exact test to compare categorical variables. We performed univariate and multivariate logistic regression analyses using the stepwise selection method to determine clinically important variables. We excluded variables with a variance inflation factor >4.0, which is considered significant multicollinearity. We calculated the annual changes in post-bronchodilator FEV_1 (mL/year) at the individual level using the first and last follow-up spirometric results. We assessed the variables affecting repeated FEV_1 measurements using a linear mixed-effect model. Statistical significance was set P-values <0.05. R statistical software, version 4.1.1 (R Core Team [2021], Vienna, Austria), was used for the statistical analyses.

2.6. Ethics

The Institutional Review Board at each hospital (Seoul Metropolitan Government-Seoul National University Boramae Medical Center IRB No. 06-2012-36) approved the study protocol. Written informed consent was obtained for all participants at enrolment. This study was conducted in accordance with the principles of the Declaration of Helsinki.

3. Results

Among 2180 patients diagnosed with COPD, 1324 patients underwent one or more spirometry examinations. Of these, 518 patients were followed up for more than 3 years with spirometry, baseline chest CT was carried out for 237 patients. Eventually, the eligible 237 patients were classified into two groups: 85 without structural abnormalities and 152 with structural abnormalities (Fig. 1). Among patients with structural abnormalities, emphysema, bronchiectasis, and TDL were found in 119 (78.3 %), 28 (18.4%), and 27 (17.8%) patients, respectively.

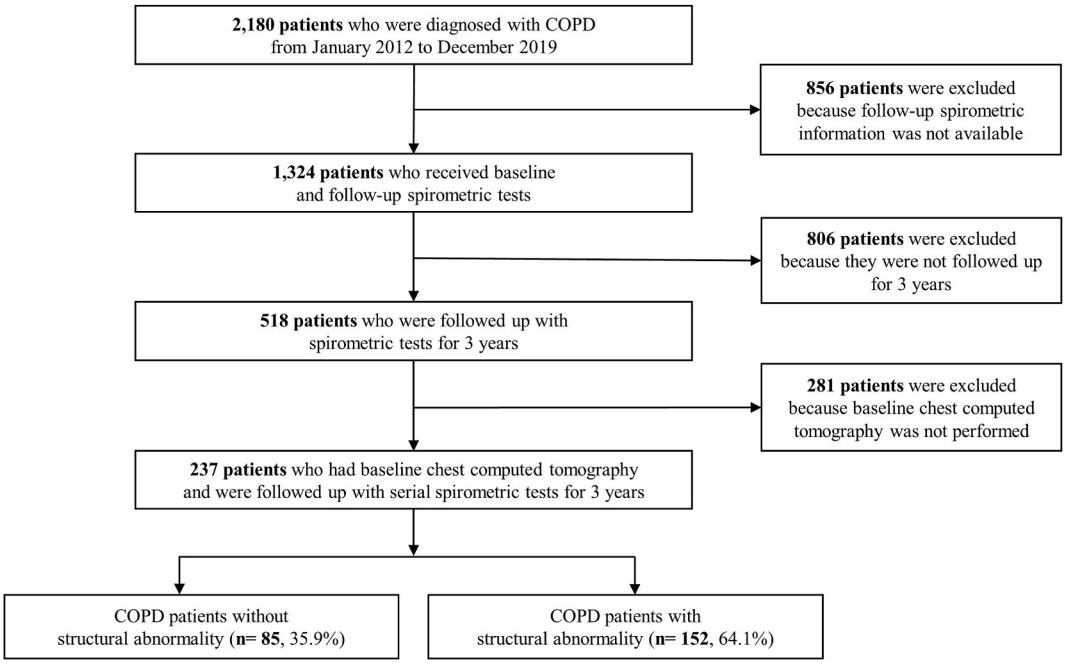

Fig. 1. Flow chart of inclusion for eligible patients BE/TDL, bronchiectasis or tuberculosis destroyed lung; COPD, chronic obstructive pulmonary disease.

3.1. Baseline characteristics and lung function

At baseline, more patients with structural abnormalities were men (Table 1). We found no significant difference in the symptomatic burden or exacerbation history between patients with and without structural abnormalities. In the baseline spirometric evaluation, FEV_1 and DL_{CO} did not differ according to structural abnormality. Whereas FEV_1/FVC was lower, and FVC (L) and total lung capacity (TLC) (L) were higher in patients with structural abnormalities (Table 2).

After excluding patients with both BE/TDL-dominant and emphysema-dominant structural abnormalities, baseline characteristics and lung function were compared between COPD patients without structural abnormalities and those with BE/TDL-dominant or emphysema-dominant structural abnormalities (Supplementary information 1 and 2). Male sex, lower BMI, ever-smoker, and higher FVC (L) or TLC (L) were more likely to be found in those with emphysema-dominant structural abnormalities than those without structural abnormalities. We found that the SGRQ-C was higher in COPD patients with BE/TDL-dominant structural abnormalities than those without structural abnormalities.

In comparing BE/TDL-dominant and emphysema-dominant structural abnormalities, male and ever-smoker patients were more likely to have emphysema-dominant structural abnormalities (**Supplementary Information 3**). Meanwhile, a higher BMI and CAT score were found in COPD patients with BE/TDL-dominant structural abnormalities. In the comparison of clinical features, those with emphysema-dominant structural abnormalities showed a higher FVC (L) or TLC (L) (**Supplementary Information 4**).

3.2. Lung function change in the overall population

In all eligible COPD patients, the estimated median annual change in post-bronchodilator FEV_1 was −38.3 (interquartile range (IQR) = −107.5–33.3] ml/year during 3 years of follow-up (**Supplementary Information 5**). We found that the annual FEV_1 change was −30.0 (IQR = −90.0–50.0) mL/year in COPD patients without structural abnormality and −41.7 (IQR = −113.8–23.3) mL/year in those with structural abnormalities. In COPD patients with structural abnormality, we found that annual FEV_1 change was −41.7 (IQR = −110.0–10.0) mL/year in BE/TDL-dominant structural abnormality and −40.0 (IQR = −109.2–28.3) mL/year in emphysema-dominant structural abnormality.

3.3. Lung function decline and clinical outcomes

In a multivariable analysis with a linear mixed model, FEV_1 decline was significantly accelerated in COPD patients with structural abnormalities (beta-coefficient = −70.6 mL/year, P-value = 0.039, Table 3). Significant clinical factors related to an accelerated annual decline in FEV_1 in COPD patients were older age, female sex, a higher CAT score, moderate-to-severe exacerbation history

Table 1
Baseline demographic characteristics of COPD patients with and without structural abnormality.

	Without structural abnormality (n = 85)	With structural abnormality (n = 152)	P-value
Age, year, mean (SD)	68.0 (7.2)	67.8 (7.1)	0.819
Age category, year, n (%)			
40-64	26 (30.6)	54 (35.5)	0.530
65-69	24 (28.2)	28 (18.4)	0.112
70-74	21 (24.7)	40 (26.3)	0.907
≥75	14 (16.5)	30 (19.7)	0.656
Male, n (%)	71 (83.5)	142 (93.4)	0.028
Body mass index, kg/m^2, mean (SD)	23.4 (3.6)	22.72 (3.5)	0.167
Smoking status, n (%)			
Never smoker	13 (15.3)	10 (6.6)	0.052
Ex-smoker	54 (63.5)	109 (71.7)	0.247
Current smoker	18 (21.2)	33 (21.7)	1.000
CCI, category, n (%)			
0	34 (40.0)	57 (37.5)	1.000
1–2 (mild)	28 (32.9)	47 (30.9)	0.197
≥3 (moderate to severe)	23 (27.1)	48 (31.6)	0.566
Previous history of asthma, n (%)	38 (45.2)	53 (35.6)	0.189
Symptoms and quality of life			
CAT score, mean (SD)	13.9 (7.8)	15.1 (7.7)	0.283
≥10, n (%)	58 (68.2)	110 (72.4)	0.601
SGRQ-C, mean (SD)	32.6 (21.1)	37.1 (20.6)	0.112
≥25, n (%)	48 (56.5)	105 (69.1)	0.071
6MWD, mean (SD)	386 (105)	388 (112)	0.892
Previous exacerbation history, n (%)			
Moderate-to-severe	10 (11.8)	35 (23.0)	0.051
Severe	5 (5.9)	15 (9.9)	0.415

Data are expressed as mean (±standard deviation) or number (percentage).
CAT, COPD assessment test; CCI, Charlson comorbidity index; COPD, chronic obstructive pulmonary disease; FEV_1, forced expiratory volume in 1 s; SD, standard deviation; SGRQ-C, St. George's respiratory questionnaire for COPD patients; 6MWD, 6 min walking distance.

Table 2
Baseline lung function of COPD patients with and without structural abnormality.

	Without structural abnormality (n = 85)	With structural abnormality (n = 152)	P-value
Baseline lung function			
Post-BDR FEV_1, L, mean (SD)	1.7 (0.6)	1.7 (0.5)	0.886
Post-BDR FEV_1, % of predicted value, mean (SD)	59.5 (17.3)	56.4 (16.6)	0.174
GOLD grade 1, n (%)	12 (14.1)	14 (9.2)	0.346
GOLD grade 2, n (%)	46 (54.1)	86 (56.6)	0.819
GOLD grade 3, n (%)	25 (29.4)	48 (31.6)	0.835
GOLD grade 4, n (%)	2 (2.4)	4 (2.6)	1.000
Post-BDR FVC, L, mean (SD)	3.0 (0.8)	3.2 (0.8)	0.048
Post-BDR FVC, % of predicted value, mean (SD)	76.6 (13.8)	78.3 (15.4)	0.382
<80%, n (%)	52 (61.2)	84 (55.3)	0.456
Post-BDR FEV_1/FVC, %, mean (SD)	54.5 (11.5)	50.3 (11.5)	0.007
DL_{CO}, % of predicted value, mean (SD)	69.7 (19.4)	64.3 (18.9)	0.058
DL_{CO}/VA, % of predicted value, mean (SD)	80.2 (20.6)	76.0 (22.8)	0.194
TLC, L, mean (SD)	5.3 (1.0)	5.7 (0.8)	0.005
TLC, % of predicted value, mean (SD)	96.2 (13.9)	97.0 (13.7)	0.744

Data are expressed as mean (±standard deviation) or number (percentage).
COPD, chronic obstructive pulmonary disease; DL_{CO}, diffusing capacity for carbon monoxide; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; GOLD, Global Initiative for Chronic Obstructive Lung Disease; ICS, inhaled corticosteroid; LABA, long-acting beta-agonist; LAMA, long-acting muscarinic antagonist; Post-BDR, Post-bronchodilator; SD, standard deviation; TLC, total lung capacity; VA, alveolar volume.

within the previous 1 year, and a lower baseline FVC. During the 3-year follow-up period, there was no significant difference in the 3-year total number of moderate-to-severe exacerbations, the annual rate of moderate-to-severe exacerbations, and mortality events between COPD patients with and without structural abnormalities (**Supplementary Information 6**). Our multivariable regression analyses found no difference in the risk of moderate-to-severe exacerbation or mortality between COPD patients with and without structural abnormalities (**Supplementary Information 7 and 8**).

3.4. Lung function change according to structural abnormality

COPD patients with BE/TDL-dominant structural abnormality showed an accelerated annual FEV_1 decline compared to those without structural abnormality (beta-coefficient = −103.7 mL/year, P-value = 0.043, Table 4). In addition, the annual FEV_1 decline rate was worse in those with emphysema-dominant structural abnormalities than in COPD patients without structural abnormalities (beta-coefficient = −84.1 mL/year, P-value = 0.018, Table 5). There was no significant difference in the 3-year total number of moderate-to-severe exacerbations, the annual rate of moderate-to-severe exacerbations, and mortality events between COPD patients without structural abnormalities and those with BE/TDL-dominant or emphysema-dominant structural abnormalities (**Supplementary Information 9**). Our multivariable regression analyses found that the risk of moderate-to-severe exacerbation or mortality was not significantly higher in COPD patients with BE/TDL-dominant or emphysema-dominant structural abnormalities than those without structural abnormalities (**Supplementary Information 10 and 11**).

Among COPD patients with structural abnormalities, there was no significant difference in the annual FEV_1 decline between BE/TDL-dominant structural abnormalities and emphysema-dominant structural abnormalities (**Supplementary Information 12**). Significant clinical factors related to an accelerated annual decline in FEV_1 in COPD patients with structural abnormalities were older age, female sex, lower BMI, higher CAT score, and higher baseline FVC. During the 3-year follow-up period, there was no significant difference in the 3-year total number of moderate-to-severe exacerbations, the annual rate of moderate-to-severe exacerbations, and mortality events between BE/TDL-dominant and emphysema-dominant structural abnormalities (**Supplementary Information 13**).

Table 3
Adjusted effect of clinical factors contributing to the annual FEV_1 change in COPD patients.

	Annual FEV_1 change, mL/year		P-value
	Beta-coefficient	Standard error	
Age	−16.73	2.29	<0.001
Sex, female	−458.15	70.29	<0.001
Current smoker	−23.637	72.47	0.744
CCI ≥3	124.1	39.08	0.001
CAT score	−6.45	2.10	0.002
Moderate-to-severe exacerbation history within previous 1 year	−238.95	41.50	<0.001
Baseline FVC, % of predicted value	17.15	1.12	<0.001
Structural abnormality on chest CT	−70.59	34.08	0.039

The results of multivariable linear mixed effect model were summarized as slope estimate %/year (standard error).
BMI and baseline FEV_1 were not included for multivariable analysis because of multicollinearity with structural abnormality.
CAT, COPD assessment test; CCI, Charlson comorbidity index; COPD, chronic obstructive pulmonary disease; CT, computed tomography; FVC, forced vital capacity.

Table 4
Effect of BE/TDL-dominant structural abnormality contributing to the annual FEV_1 change compared to no structural abnormality.

	Annual FEV_1 change, mL/year		P-value
	Beta-coefficient	Standard error	
Age	−21.6	3.26	<0.001
Sex, female	−496.74	78.54	<0.001
BMI	13.53	7.00	0.054
Current smoker	−91.00	84.97	0.285
CAT score	5.21	2.97	0.080
Moderate-to-severe exacerbation history within previous 1 year	−179.06	67.20	0.008
Baseline FVC, % of predicted value	15.85	1.71	<0.001
DL_{CO}, % of predicted value	10.17	1.40	<0.001
BE/TDL-structural abnormality (reference: no structural abnormality)	−103.65	50.91	0.043

The results of multivariable linear mixed effect model were summarized as slope estimate %/year (standard error).
Baseline FEV_1 were not included for multivariable analysis because of multicollinearity with structural abnormality.
BE/TDL, bronchiectasis or tuberculosis destroyed lung; BMI, body mass index; CAT, COPD assessment test; CCI, Charlson comorbidity index; COPD, chronic obstructive pulmonary disease; DL_{CO}, diffusing capacity for carbon monoxide; FVC, forced vital capacity.

Table 5
Effect of emphysema-dominant structural abnormality contributing to the annual FEV_1 change compared to no structural abnormality.

	Annual FEV_1 change, mL/year		P-value
	Beta-coefficient	Standard error	
Age	−15.53	2.45	<0.001
Sex, female	−445.92	60.75	<0.001
Current smoker	0.334	41.54	0.994
CAT score	−11.44	2.24	<0.001
Moderate-to-severe exacerbation history within previous 1 year	−258.74	43.30	<0.001
Baseline FVC, % of predicted value	18.34	1.18	<0.001
Emphysema-dominant structural abnormality (reference: no structural abnormality)	−84.13	35.53	0.018

The results of multivariable linear mixed effect model were summarized as slope estimate %/year (standard error).
BMI and baseline FEV_1 were not included for multivariable analysis because of multicollinearity with structural abnormality.
CAT, COPD assessment test; COPD, chronic obstructive pulmonary disease; FVC, forced vital capacity.

4. Discussion

Our study found that structural abnormalities were significantly associated with lung function changes, but did not affect clinical prognosis in patients with COPD. Patients with structural abnormalities had a faster annual FEV_1 decline rate during the 3-year observation period, even after adjusting for clinically important variables. Patients with BE/TDL-dominant or emphysema-dominant structural abnormalities showed an accelerated FEV_1 decline compared to those without structural abnormalities. We found no significant difference in annual FEV_1 changes between the BE/TDL-dominant and emphysema-dominant structural abnormalities. The morphological features of chest CT need to be considered as a clinical subtype related to longitudinal lung function changes in COPD patients.

Radiologic subtyping of structural abnormalities is related to the pathophysiology of structural changes in COPD. Visually defined radiological findings in COPD patients can be divided into two subtypes: airway-predominant disease and emphysema-predominant disease [6,16]. Pathological mechanisms of chronic bronchitis include infiltration of mononuclear cells and influx of neutrophils in the airway, which produce airway inflammation and cause mucus hypersecretion [17]. In emphysema, the lung parenchyma is destroyed by apoptosis of endothelial or epithelial cells and a dysregulated protease-antiprotease balance [17,18]. Subtyping using chest CT can provide information on clinical features and may help predict treatment responses or clinical outcomes [6,19]. In our study, COPD patients with BE/TDL or emphysema showed a more rapid FEV_1 decline than those without structural abnormalities. Considering these structural abnormalities, including emphysema, bronchiectasis, and TDL, are increasingly detected in clinical practice with the increasing use of chest CT for lung cancer screening, doctors may find and treat more COPD patients at an early stage.

Bronchiectasis can lead to chronic and indolent airway inflammatory progression via a vicious cycle [20]. Although it has not yet been elucidated whether bronchiectasis causes COPD, it aggravates airflow limitation and the clinical course in COPD patients [21]. Neutrophil elastase activity may be related to an accelerated decrease in FEV_1 in patients with bronchiectasis [22]. A recent report showed that brensocatib that inhibits neutrophil serine protease activity significantly reduces exacerbation risk and is potentially beneficial in preserving lung function [23]. In addition, clinical factors, such as *Pseudomonas aeruginosa* colonization, exacerbation events, and systemic inflammation, can further accelerate lung function decline in patients with bronchiectasis [24]. Considering the faster rate of lung function decline, further studies on appropriate therapeutic strategies to preserve lung function in COPD patients with bronchiectasis are required.

TDL is radiologically diagnosed as a destructive lung parenchymal sequela due to pulmonary tuberculosis. Many studies have

reported lung function impairment after treatment for pulmonary tuberculosis [25]. TDL causes chronic respiratory symptoms, accelerated lung function decline, and more exacerbations [26,27]. Although the mechanism of airflow limitation by TDL is poorly understood, multiple factors, including host immune response, genetic predisposition, comorbidities, and environmental exposure, may affect lung function decline in patients with TDL [25]. Many studies have reported a higher risk of COPD development in patients with TDL [28–30]. Our study showed that TDL was found in 18% of patients and was related to accelerated FEV_1 decline in COPD patients. Therefore, COPD patients with TDL need to be considered a high-risk group for rapid lung function decline.

The relationship between emphysema and rapid decline in lung function has been reported in well-designed studies [5,13,31,32]. Dysregulated host immune responses, including protease and antiprotease imbalance, inhibition of histone deacetylase, excessive airway inflammation, and oxidative stress, can contribute to the development and progression of emphysema [12]. Inflammatory and mechanical factors involved in the pathogenesis of emphysema can worsen lung function in patients [33]. Lung function decline is related to emphysema severity in patients with COPD [31]. Recently, the concept of pre-COPD has been proposed, which includes patients with emphysema and normal lung function because airflow limitation progresses in this population [34]. In our study, even after adjusting for smoking status and exacerbation history, emphysema was significantly correlated with accelerated FEV_1 decline. This finding suggests that host factors are related to an accelerated lung function decline in emphysema. Therefore, it is necessary to investigate genetic factors related to various host responses to airway inflammation or parenchymal destruction caused by noxious exposure or infection.

In our study, structural abnormalities did not impact on prognosis. There seems several reasons for the negative results. First, while structural abnormalities contribute to the deterioration of chronic airway inflammation or the progression of COPD, their direct association with exacerbation or mortality may not be clinically evident. Indeed, our findings align with recent studies that demonstrated no correlation between BE or TDL and the severity of acute exacerbations in COPD [35]. Furthermore, emphysema also showed no association with moderate to severe or severe exacerbation of COPD in multivariable analysis [36]. Second, in our study, there was a small number of exacerbation and mortality events, which might have resulted in insufficient statistical power. Third, our study had a relatively short observation period of 3 years, which would be insufficient duration for evaluating mortality. Fourth, exacerbations may be influenced more by factors beyond structural abnormalities, such as previous exacerbation history, symptom severity, and treatment.

Our study had several limitations. First, it was a retrospective study with a small number of patients. Our results need to be validated in a larger cohort of patients with COPD. Second, bronchiectasis and emphysema have heterogeneous features; however, our study did not quantify or qualify their extent, severity, and pattern. We classified the included patients according to the presence or absence of bronchiectasis and emphysema, as described in the radiologist's report. Currently, image data are being collected for further research. Third, lung function decline rate is affected by various factors other than structural abnormalities, such as health behavior, air pollution, and second-hand smoking. Even if the FEV_1 change is adjusted based on available clinical information, our results should be carefully interpreted.

In conclusion, the lung function decline rate was faster in COPD patients with BE/TDL-dominant or emphysema-dominant structural abnormalities than in those without. An individualized approach for preserving lung function in COPD patients with structural abnormalities should be investigated further.

Ethics approval and consent to participate

The Institutional Review Board at each hospital (Seoul Metropolitan Government-Seoul National University Boramae Medical Center IRB No. 06-2012-36) approved the study protocol. Written informed consent was obtained for all participants at enrolment. This study was conducted in accordance with the principles of the Declaration of Helsinki.

Consent for publication

Not applicable.

Availability of data and materials

The data that support the findings of this study are available from the KOrea COpd Subgroup Study (KOCOSS) team but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are however available from the authors upon reasonable request and with permission of the KOCOSS team.

Funding

This work was supported by the Research Program funded Korea National Institute of Health (Fund CODE 2016ER670100, 2016ER670101,2016ER670102, 2018ER67100,2018ER67101,2018ER67102, 2021ER120500, and 2021ER120501).

CRediT authorship contribution statement

Hyun Woo Lee: Writing – original draft, Visualization, Software, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Jung-Kyu Lee:** Writing – review & editing, Resources, Data curation. **Youlim Kim:** Writing

– review & editing, Resources, Data curation. **An-Soo Jang:** Writing – review & editing, Resources, Data curation. **Yong il Hwang:** Writing – review & editing, Resources, Data curation. **Jae Ha Lee:** Writing – review & editing, Resources, Data curation. **Ki-Suck Jung:** Writing – review & editing, Resources, Funding acquisition, Data curation. **Kwang Ha Yoo:** Writing – review & editing, Resources, Funding acquisition, Data curation. **Hyoung Kyu Yoon:** Writing – review & editing, Resources, Data curation. **Deog Kyeom Kim:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:Kwang Ha Yoo reports financial support was provided by Korea National Institute of Health. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

List of Abbreviations

BE/TDL	bronchiectasis or tdl
BMI	body mass index
CAT	COPD assessment test
COPD	chronic obstructive pulmonary disease
CT	computed tomography
DLCO	diffusing capacity for carbon monoxide
FEV_1	forced expiratory volume in 1 s
FVC	forced vital capacity
IQR	interquartile range
SGRQ-C	st george's respiratory questionnaire for COPD patients
STROBE	strengthening the reporting of observational studies in epidemiology
TDL	tuberculosis-destroyed lung; VA, alveolar volume

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e27683.

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