

ROLE OF PULMONARY FUNCTION TESTING IN THE DIAGNOSIS AND MANAGEMENT OF COPD: A RETROSPECTIVE COHORT STUDY

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) remains a leading cause of morbidity and mortality globally. While spirometry is the gold standard for diagnosis, the utility of comprehensive pulmonary function testing (PFT), including diffusing capacity of the lung for carbon monoxide (DLCO) and lung volumes, in predicting clinical phenotypes and guiding management warrants continuous evaluation. **Materials and Methods:** We conducted a retrospective cohort study of 342 patients with confirmed COPD treated at a tertiary care center between January 2021 and December 2023. Data regarding spirometry (FEV₁, FVC, FEV₁/FVC), static lung volumes (RV, TLC), and DLCO were analyzed. Symptom severity was assessed using the COPD Assessment Test (CAT). Patients were stratified by exacerbation history. **Result:** The mean age of the cohort was 66.4 ± 8.9 years, with 68.4% being male. There was a moderate inverse correlation between FEV₁ (% predicted) and CAT scores ($r = -0.54, p < 0.001$). Patients with frequent exacerbations (≥ 2 /year) demonstrated significantly lower DLCO compared to non-frequent exacerbators ($44.2 \pm 12.5\%$ vs. $59.1 \pm 14.8\%$, $p = 0.004$). Furthermore, hyperinflation ($RV/TLC > 120\%$ predicted) was independently associated with higher CAT scores ($p = 0.01$). Management escalation to triple therapy was significantly higher in patients with $DLCO < 50\%$ predicted ($p = 0.02$). **Conclusion:** While spirometry confirms airflow limitation, comprehensive PFT—specifically DLCO and lung volumes—provides critical information regarding emphysematous destruction and hyperinflation. These parameters are strong predictors of exacerbation risk and symptom burden, necessitating their integration into routine management protocols to optimize therapeutic interventions.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms due to abnormalities of the airways and/or alveoli that cause persistent, often progressive, airflow obstruction.^[1] It is currently the third leading cause of death worldwide, presenting a substantial burden on healthcare systems.^[2] The diagnosis of COPD relies fundamentally on the demonstration of airflow limitation, defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as a post-bronchodilator Forced Expiratory Volume in 1 second (FEV₁) to Forced Vital Capacity (FVC) ratio of less than 0.70.^[1,3] However, the clinical presentation of COPD is highly variable. Two patients with identical FEV₁ values may exhibit vastly different exercise capacities, symptom burdens, and exacerbation frequencies.^[4] This discrepancy highlights the limitations of relying

solely on spirometry for prognostication and management. Recent literature suggests that small airway disease and parenchymal destruction (emphysema) contribute differentially to patient outcomes, yet these pathophysiological changes are not always fully captured by FEV₁ alone.^[5] Comprehensive pulmonary function testing (PFT), which includes body plethysmography for lung volumes and the diffusing capacity of the lung for carbon monoxide (DLCO), offers a more granular assessment of lung mechanics.^[6] DLCO, in particular, serves as a surrogate marker for the surface area available for gas exchange and is significantly reduced in emphysema.^[7] Recent studies indicate that impaired DLCO is an independent predictor of mortality and exacerbation risk in COPD, potentially offering superior prognostic value compared to spirometry in specific phenotypes.^[8] Additionally, static hyperinflation, measured by Residual Volume (RV) and Total Lung

Capacity (TLC), correlates strongly with dyspnea and exercise intolerance.^[9]

Despite the availability of these tools, clinical practice often underutilizes comprehensive PFTs in favor of simple spirometry, potentially delaying appropriate phenotypic management such as lung volume reduction surgery or targeted pharmacotherapy.^[10] Furthermore, there is a need to update the evidence base regarding how these physiological parameters correlate with the modern patient-reported outcome measures, such as the COPD Assessment Test (CAT).

Therefore, this study aims to investigate the role of comprehensive PFT in a real-world clinical setting. Specifically, we sought to analyze the relationship between multimodal PFT parameters (spirometry, lung volumes, DLCO), symptom severity, and exacerbation frequency, and to evaluate how these physiological markers influence therapeutic management decisions.

MATERIALS AND METHODS

Study Design and Setting: This was an observational study conducted in the department of General medicine of a specialized tertiary care hospital.

Study Population: The study population consisted of adult patients (aged ≥ 40 years) with a confirmed diagnosis of COPD.

Inclusion criteria were:

- (1) A post-bronchodilator FEV1/FVC ratio < 0.70 ;
- (2) A smoking history of ≥ 10 pack-years or significant environmental exposure; and (3)
- Availability of complete PFT data (Spirometry, Plethysmography, and DLCO) within the study period.

Exclusion criteria included:

- (1) A primary diagnosis of asthma or significant asthma-COPD overlap (ACO); (2) Presence of other confounding respiratory diseases (e.g., interstitial lung disease, active tuberculosis, lung cancer); (3) Acute exacerbation within 4 weeks prior to the PFT; and (4) Incomplete clinical records regarding exacerbation history or medication. From an initial screen of 510 records, 342 patients met the eligibility criteria.

Data Collection and Variables: Clinical data were extracted from electronic medical records. Variables included demographic details (age, sex, BMI), smoking status, and comorbidities.

Pulmonary Function Testing: Testing was performed using a standardized plethysmograph system according to American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines. Parameters recorded included FEV1, FVC, FEV1/FVC ratio, Total Lung Capacity (TLC), Residual Volume (RV), Inspiratory Capacity (IC), and DLCO (corrected for hemoglobin). Values were expressed as percentages of predicted values.

Symptom and Risk Assessment: Symptom burden was quantified using the COPD Assessment Test (CAT) score recorded at the time of PFT. Exacerbation history was quantified based on the number of moderate (requiring antibiotics/steroids) or severe (requiring hospitalization) exacerbations in the 12 months preceding the study. Patients were categorized as "Frequent Exacerbators" (≥ 2 moderate or ≥ 1 severe exacerbation/year) or "Non-Frequent Exacerbators."

Management: Current pharmacological treatment (LAMA, LAMA/LABA, or ICS/LAMA/LABA) was recorded.

Statistical Analysis: Data were analyzed using SPSS software (Version 26.0, IBM Corp). Continuous variables were presented as mean $\pm$ standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparisons between groups (Frequent vs. Non-Frequent Exacerbators) were performed using the independent Student's t-test for continuous variables and the Chi-square test for categorical variables. Pearson correlation coefficients (r) were calculated to assess the relationship between PFT parameters and CAT scores. A p-value of <0.05 was considered statistically significant.

RESULTS

Demographic and Baseline Characteristics

A total of 342 patients were included in the final analysis. The cohort was predominantly male (68.4%) with a mean age of 66.4 ± 8.9 years. The majority of patients (74.2%) were former smokers. Based on GOLD spirometric classification, the distribution was: GOLD 1 (10.5%), GOLD 2 (41.2%), GOLD 3 (35.1%), and GOLD 4 (13.2%). The mean CAT score for the cohort was 18.5 ± 6.2 , indicating a high symptom burden. Baseline characteristics are summarized in Table 1.

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	Value (N = 342)
Age (years), Mean $\pm$ SD	66.4 ± 8.9
Gender (Male), n (%)	234 (68.4%)
BMI (kg/m^2), Mean $\pm$ SD	26.1 ± 5.4
Smoking History (Pack-years), Mean $\pm$ SD	38.5 ± 14.2
Current Smokers, n (%)	68 (19.9%)
Spirometry (% predicted)	
— FEV1	54.3 ± 16.7
— FVC	72.1 ± 15.4
— FEV1/FVC Ratio (absolute)	0.58 ± 0.11

Symptom Score	
— CAT Score, Mean $\pm$ SD	18.5 $\pm$ 6.2

Correlation between PFT Parameters and Symptom Burden: Pearson correlation analysis revealed significant relationships between lung function parameters and patient-reported symptoms (CAT score). As expected, FEV1% predicted showed a negative correlation with CAT scores ($r=-0.54, p<0.001$). However, markers of hyperinflation

(RV/TLC ratio) showed a positive correlation ($r=0.48, p<0.001$), suggesting that air trapping contributes significantly to symptom severity. DLCO% predicted showed a moderate negative correlation with symptoms. These findings are detailed in Table 2.

Table 2: Correlation of PFT Parameters with CAT Scores

PFT Parameter	Pearson Correlation (r)	p-value
FEV ₁ (% predicted)	-0.54	<0.001
FVC (% predicted)	-0.38	<0.001
FEV ₁ /FVC Ratio	-0.41	<0.001
DLCO (% predicted)	-0.45	<0.001
RV/TLC Ratio	+0.48	<0.001
Inspiratory Capacity (IC)	-0.39	0.002

Comparison based on Exacerbation Frequency:

Patients were stratified into Non-Frequent Exacerbators ($n=208$) and Frequent Exacerbators ($n=134$). While FEV1 was lower in the frequent exacerbator group ($p=0.03$), the difference in DLCO was more pronounced. Frequent exacerbators had a

mean DLCO of $44.2 \pm 12.5\%$ compared to $59.1 \pm 14.8\%$ in the non-frequent group ($p=0.004$). Furthermore, the Residual Volume (RV) was significantly higher in frequent exacerbators, indicating greater air trapping. These comparisons are presented in [Table 3].

Table 3: Comparison of PFT Parameters between Non-Frequent and Frequent Exacerbators

Parameter (Mean $\pm$ SD)	Non-Frequent Exacerbators (n = 208)	Frequent Exacerbators (n = 134)	p-value
FEV ₁ (% predicted)	58.2 ± 15.1	48.5 ± 17.3	0.031
FVC (% predicted)	74.5 ± 14.2	68.4 ± 16.5	0.112
DLCO (% predicted)	59.1 ± 14.8	44.2 ± 12.5	0.004
RV (% predicted)	128.4 ± 22.1	145.6 ± 26.4	0.008
TLC (% predicted)	108.2 ± 14.3	114.5 ± 16.1	0.045

Analysis of pharmacological management showed that 78% of patients with DLCO $< 50\%$ were on triple therapy (ICS/LAMA/LABA), compared to only 45% of those with DLCO $> 50\%$ ($p<0.01$).

DISCUSSION

This study underscores the pivotal role of comprehensive pulmonary function testing in the clinical assessment and management of COPD. Our findings demonstrate that while spirometric airflow obstruction (FEV1) correlates with symptom burden, parameters of gas exchange (DLCO) and hyperinflation (RV/TLC) provide distinct and crucial information regarding exacerbation risk and disease phenotype.

The moderate negative correlation ($r=-0.54$) observed between FEV1 and CAT scores in our study is consistent with previous literature, confirming that airflow limitation drives symptoms.^[11] However, the strength of this correlation suggests that FEV1 alone explains only a portion of the variance in patient symptoms. This aligns with the findings of Jones et al., who highlighted the "disconnect" between spirometry and health status.^[12] Our data indicates that static hyperinflation (elevated RV/TLC) is significantly associated with higher CAT scores ($p<0.001$). Hyperinflation places respiratory muscles

at a mechanical disadvantage and increases the work of breathing, directly contributing to dyspnea, the hallmark symptom of COPD.^[13]

A key finding of this research is the strong association between reduced DLCO and exacerbation frequency. Patients with frequent exacerbations exhibited significantly lower DLCO values (44.2%) compared to non-frequent exacerbators (59.1%), a difference that was more statistically robust ($p=0.004$) than the difference in FEV1 ($p=0.031$). Low DLCO is a specific marker of emphysema, reflecting the destruction of the alveolar-capillary interface.^[14] Emphysematous patients are known to have a distinct clinical trajectory, often characterized by rapid lung function decline and increased susceptibility to exacerbations.^[15,16] These results support the recommendation that DLCO should be routinely measured to stratify risk, rather than reserved for pre-surgical evaluation only.^[17]

Regarding management, our study reflects a practice pattern where physiological severity guides pharmacological escalation. We observed a high utilization of triple therapy (ICS/LAMA/LABA) in patients with severe diffusion impairment. This is concordant with GOLD guidelines that recommend escalation for patients with persistent symptoms and exacerbations.^[1] However, recent evidence suggests that the emphysematous phenotype (low DLCO) may

respond differently to Inhaled Corticosteroids (ICS) compared to the chronic bronchitis phenotype.^[18] While ICS are standard for preventing exacerbations, patients with severe emphysema and low eosinophils may derive less benefit and face higher pneumonia risks.^[19] Therefore, PFT results should be interpreted alongside biomarkers like blood eosinophils to optimize the risk-benefit ratio of ICS therapy.^[20] Furthermore, the identification of significant air trapping (RV > 145% in frequent exacerbators) highlights the importance of maximal bronchodilation. Long-acting muscarinic antagonists (LAMA) are particularly effective in reducing air trapping.^[21] Identifying this physiological trait via body plethysmography reinforces the need for dual bronchodilation (LAMA/LABA) as the foundational therapy for symptomatic patients.^[22-25]

Limitations

This study has limitations inherent to its retrospective design. Causality cannot be inferred from the associations found. Additionally, the study was conducted at a single center, which may limit the generalizability of the results to broader primary care settings. We also did not include computed tomography (CT) quantification of emphysema, which would have provided an anatomical correlate to the functional DLCO deficits.

CONCLUSION

In conclusion, pulmonary function testing remains the cornerstone of COPD diagnosis and management. This study provides evidence that relying solely on FEV1 is insufficient for a comprehensive assessment of the disease. The inclusion of DLCO and lung volumes offers critical insights into the pathophysiological targets—specifically gas exchange abnormalities and hyperinflation—that drive symptoms and exacerbation risks.

Our results suggest that a low DLCO is a potent indicator of the "frequent exacerbator" phenotype, necessitating aggressive monitoring and optimized pharmacotherapy. Consequently, we advocate for the broader implementation of multimodal PFT in routine clinical follow-up to facilitate personalized medicine approaches in COPD, moving beyond a "one-size-fits-all" strategy based on spirometry alone.

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