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Research Article

ASSESSMENT OF AIRWAVE OSCILLOMETRY AND ITS CORRELATION WITH SPIROMETRY AND HRCT THORAX FINDINGS AMONG COPD PATIENTS

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ABSTRACT:

Background: Chronic obstructive pulmonary disease (COPD) is characterized by progressive airflow limitation and structural lung abnormalities. The present study aimed to correlate airwave oscillometry (AOS) parameters with spirometry and High-Resolution Computed Tomography (HRCT) Thorax findings among COPD patients.

Methodology: This cross-sectional comparative study conducted at the tertiary care centre from South India. This study was conducted on patients diagnosed with COPD. All participants underwent detailed clinical and radiological evaluation, including spirometry, AOS, and HRCT of the thorax. The primary endpoint was to evaluate correlations between spirometric and AOS parameters and HRCT-derived radiological markers.

Results: Thirty-one COPD patients (mean age 65.3 ± 10.7 years; 87.1% male) were included in the study. Progressive COPD severity was associated with significant declines in FEV_1, FVC, and FEF25–75 (all $p = 0.001$). AOS demonstrated rising resistance with severity (R5, $p = 0.001$; R20, $p = 0.003$), while R5–R20 and X5 worsened without significance. Post-bronchodilator FVC correlated negatively with R5–R20 ($\rho = -0.356$, $p = 0.05$) and X5 ($\rho = -0.51$, $p = 0.03$), and positively with X5insp ($\rho = 0.34$, $p = 0.05$). Notably, AOS indices (R5, R20) correlated negatively with tracheal index, whereas spirometric measures showed no significant association, suggesting superior sensitivity of AOS in detecting structural airway narrowing.

Conclusion: Spirometry captured progressive expiratory flow limitation with COPD and emphysema severity, while AOS provided complementary insights into small airway dysfunction and lung inhomogeneity. AOS measures, particularly resistance and reactance indices, may enhance the evaluation of COPD beyond conventional spirometry.

Keywords: Chronic Obstructive Pulmonary Disease, Spirometry, Airwave oscillometry, oscillometry, High-Resolution Computed Tomography

Introduction:

Chronic obstructive pulmonary disease (COPD) is a progressive inflammatory disorder of the airways characterized by irreversible airflow obstruction. It is now recognized as a systemic condition, with extrapulmonary effects driven by chronic inflammation, hypoxemia-related metabolic changes, and physical inactivity, collectively worsening disease severity, morbidity, and mortality (1,2). According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2025 defines COPD as a heterogeneous lung disease characterized by chronic symptoms dyspnea, cough, sputum, and/or exacerbations caused by airway (bronchitis, bronchiolitis) and/or alveolar (emphysema) abnormalities, resulting in persistent, often progressive airflow limitation (3). It is the 4th leading cause of death globally and is projected by WHO to become the 3rd by 2030 (4). The global prevalence of COPD in adults aged 30–79 is about 10.3% (95% CI: 8.2–12.8%), with data indicating stability over the past decade (6.1% in 2011 vs. 6.0% in 2021) (5,6).

COPD arises from a complex interplay of genetic, environmental, and early-life factors. Cigarette smoking is the primary risk factor, but only some smokers develop the disease. Other contributors include passive smoking, air pollution, occupational exposures, and genetic disorders such as alpha-1 antitrypsin deficiency (7). Acute COPD exacerbations are frequent, typically triggered by factors such as bacterial or viral infections or environmental pollutants (8).COPD develops through oxidative stress and a protease-antiprotease imbalance, causing alveolar destruction, impaired gas exchange, and loss of lung elasticity.

Reduced lung growth or suboptimal peak lung function from genetic or environmental influences further heightens long-term COPD risk (8–11).

Treatment for COPD can alleviate symptoms and reduce exacerbations, but recurrent acute episodes accelerate lung function decline, leading to advanced emphysema and, in some cases, the need for lung transplantation to prevent fatal outcomes. Early intervention is vital; however, it requires diagnostic tools that can identify subtle or early lung changes in at-risk individuals—an area where current standard diagnostics remain inadequate. (9). In addition, early recognition allows for timely intervention to preserve lung function and slow progression (10).

Spirometry is the gold standard for confirming COPD in suspected cases. Diagnosis is made when the post-bronchodilator FEV_1/FVC ratio is <0.7. While a reduced FEV_1 suggests airflow obstruction, it may also be affected by lung volumes, elastic recoil, respiratory muscle strength, and patient effort (12). In healthy individuals, most airway resistance arises in the 4–8th airway generations. Therefore, FEV1 primarily reflects obstruction in the larger airways, and substantial small airways disease must be present before FEV1 shows abnormalities.

Evaluating the mid-portion of expiratory flow can provide additional insight into small airway involvement. The Forced Expiratory Flow between 25% and 75% of the FVC (FEF25–75) is commonly used for this purpose (13). However, FEF25–75 depends on FVC, so variations in FVC can alter the flow-volume segment assessed, and its reproducibility is poor unless adjusted for lung volume (14). Moreover, FEF25–75 often remains normal when the FEV1/FVC ratio is > 75% and correlates poorly with other indicators of small airways disease (15–17).

Leading guidelines, including those from the GOLD and the American Thoracic Society/European Respiratory Society (ATS/ERS), recommend using specific percent predicted FEV_1 ($ppFEV_1$) values or z-score thresholds to determine the severity and classification of airflow limitation. (7,18). Although $ppFEV_1$ is commonly used to assess COPD severity, it has limitations due to reliance on reference equations that may not represent the general population and often include race-based adjustments, risking misclassification. In cases with concomitant restriction, FEV_1 may overestimate severity. The FEV_1/FVC ratio offers a more reliable and clinically relevant measure, correlating better with key outcomes (19). Comprehensive evaluation of COPD requires both functional and structural assessment. Although spirometry is the GOLD-recommended standard for diagnosing fixed airflow obstruction, it has several shortcomings, including reliance on patient effort, limited sensitivity for early or small airway disease, lack of information on lung volumes or gas exchange, and reduced utility in acutely ill or uncooperative patients (7). To overcome these limitations, alternative tools are valuable. High-resolution CT (HRCT) offers detailed visualization of airway and parenchymal involvement and helps quantify structural damage (20), whereas oscillometry provides a rapid, non-invasive assessment of respiratory mechanics during tidal breathing, making it suitable for patients unable to perform forced maneuvers. However, its clinical utility is constrained by the absence of standardized global reference values and inter-device variability, which limit cross-study comparability. Despite these challenges, oscillometry demonstrates potential in characterizing disease phenotypes and warrants further investigation (21–23).

Forced oscillation (FOT) and impulse oscillometry (IOS) are the two main techniques, with FOT applying sinusoidal pressure oscillations and IOS delivering square wave pressure pulses across multiple frequencies. Both methods are patient-friendly and yield detailed assessments of lung impedance (Zrs), from which resistance (Rrs) and reactance (Xrs) are

derived (24–26). Oscillometry generates sound waves between 3 and 35 Hz; lower frequencies assess small airways while higher frequencies assess large airways. This enables evaluation of the entire airway tree (26,27). In COPD, increased airway resistance (e.g., with bronchoconstriction) raises Rrs, while increased compliance (as in emphysema) lowers Xrs, making it more negative (28). Key indices include R5 (total airway resistance), R20 (large airway resistance), R5-R20 (small airway resistance), X5 (reactance at 5 Hz), and AX (area under the Xrs curve).

Advances in FOT have produced small, portable devices such as airwave oscillometry (AOS), which utilizes a vibrating mesh to generate a multifrequency sinusoidal pseudorandom noise (PRN) signal. The portability of AOS allows for both home- and clinic-based lung function testing, offering practical advantages over traditional FOT devices (29,30). Notably, AOS not only achieves more successful measurements than spirometry, especially in populations unable to perform forced maneuvers, but it also demonstrates greater sensitivity in detecting bronchodilator reversibility (29). Given the limitations of spirometry in fully characterizing COPD, this study aims to correlate AOS with spirometry and HRCT findings to evaluate whether oscillometry can offer complementary or earlier insights into small airway dysfunction and structural lung damage, ultimately enhancing diagnostic accuracy and disease assessment in COPD patients.

Methodology

This study was designed as a cross-sectional comparative study and conducted in the Department of Respiratory Medicine, Mahatma Gandhi Medical College and Research Institute (MGMCRI), Pondicherry, between 2022 and 2025. Approval was obtained from the Institutional Ethics Committee (IEC: MGMCRI/Res/01/2024/09/IHEC/03) prior to commencement, ensuring adherence to ethical standards for research involving human participants. This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and adhered to the standards of Good Clinical Practice (GCP) guidelines. The written informed consent was obtained from all participants before enrollment.

Study Population The study included patients aged above 40 years with a pre-existing diagnosis of COPD or those presenting with symptoms suggestive of COPD, confirmed by spirometry as per the GOLD 2024 guidelines. Patients with acute exacerbation of COPD, microbiologically confirmed pulmonary tuberculosis, active lower respiratory tract infection (viral or bacterial), hemoptysis, or pneumothorax, as well as pregnant women, were excluded from the study.

Study Procedure

All participants underwent a standardized evaluation protocol. AOS was performed using the Tremoflo C-100 system (Thorasys, Canada) to assess small airway function. Parameters recorded included resistance indices—R5 (resistance at 5 Hz), R20 (resistance at 20 Hz), and R5–R20 (marker of small airway involvement)—and reactance indices such as X5 (at 5 Hz; inspiratory, expiratory, and post-bronchodilator) and AX (area under the reactance curve). Spirometry was conducted with MIR WinspiroPro (MIR, Italy) to measure forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC ratio, and FEF25–75, with both pre- and post-bronchodilator values obtained, and disease severity staged according to GOLD criteria. HRCT of the thorax was selectively performed using the GE 660 Optima 128-slice CT scanner in patients with persistent symptoms despite optimal therapy, disproportionate symptoms relative to spirometry findings, FEV_1 <45% predicted with hyperinflation or gas trapping, or eligibility for lung cancer screening as per ACCP/USPSTF guidelines. HRCT findings assessed included emphysema, mosaic

attenuation, bullae, bronchiectasis, fibrosis, and features of pulmonary hypertension, with emphysema severity categorized by mean lung density as mild (−850 to −900 HU), moderate (−900 to −950 HU), or severe (<−950 HU).

Study Outcomes

The primary outcomes were correlations between AOS parameters (resistance and reactance indices), spirometry findings (FEV_1, FEV_1/FVC, FEF25–75), and HRCT thorax features (emphysema, air-trapping, and structural abnormalities).

Secondary outcomes included the strength of these correlations, expressed as weak, moderate, strong, or very strong, as well as group differences in pulmonary function and imaging findings based on clinical variables such as age, gender, smoking history, and COPD severity. Statistical significance was determined at $p < 0.05$.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 23 (IBM Corp., Armonk, NY, USA)**.** Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on their distribution. Group comparisons were performed using the Chi-square test or Fisher's exact test for categorical variables, and the Independent Student's t-test or Mann–Whitney U test for continuous variables, as appropriate. Correlations between AOS, spirometry, and HRCT findings were assessed using Pearson's or Spearman's correlation coefficients, with correlation strength categorized as weak (≤0.35), moderate (0.36–0.67), strong (0.68–0.89), or very strong (≥0.90). A p-value of <0.05 was considered statistically significant.

RESULTS Among the 31 patients, the mean age was 65.3 ± 10.7 years, with the majority being male (87.1%). Mean height and weight were 160 ± 6.7 cm and 62.4 ± 5.4 kg, respectively, yielding a mean BMI of 23.5 ± 4.0 kg/m². Regarding smoking status, 41.9% were never smokers, 6.5% were current smokers, and 51.6% were reformed smokers. Dyspnea assessment using the mMRC scale showed 29.0% with grade I, 54.8% with grade II, and 16.1% with grade III. Distribution across COPD groups was 25.8% in Group A, 41.9% in Group B, 6.5% in Group C, and 32.3% in Group D. Among the 14 patients with bronchiectasis, 41.2% were never smokers and 41.2% were reformed smokers (**Table 1**).

Table 1: Demographic and clinical characteristics

Variable	**Proportions [Mean ± S or n (%)]**
Age	65.29±10.7
Gender	
Female	4 (12.9)
Male	27 (87.1)
Height (cm)	160+6.7
Weight (Kg)	62.4+5.4

BMI (Kg/m^2)	23.5+4.0
Smoking status	
Never smoker	13 (41.94)
Current smoker	2 (6.45)
Reformed smoker	16 (51.61)
Dyspnea	
MMRC-I	9 (29.0)
MMRC-II	17 (54.8)
MMRC-III	5 (16.1)
COPD groups,	
A	8 (25.8)
B	13 (41.9)
C	2 (6.5)
D	10 (32.3)
Bronchiectasis (n = 14),	
Never Smokers	6 (41.2)
Reformed Smokers	6 (41.2)

Pulmonary Function Indices Assessed by Spirometry and Oscillometry Across COPD Severity

FEV_1, FVC, and FEF25–75 decreased markedly from moderate to very severe COPD (all p = 0.001), while FEV_1/FVC showed a borderline decline (p = 0.051), reflecting progressive airflow limitation and small airway dysfunction. Oscillometry parameters R5 and R20 increased significantly with severity (p = 0.001 and 0.003), whereas R5–R20 and X5 Post showed trends toward worsening without reaching significance (**Table 2**). Spearman correlation analysis revealed that post-bronchodilator FVC correlated negatively with Post R5–R20 (ρ = −0.356, p = 0.05) and Post X5 (ρ = −0.51, p = 0.03), and positively with Post X5insp (ρ = 0.34, p = 0.05), indicating that higher peripheral airway resistance and reduced reactance are associated with lower lung volumes. FEV_1 and FEV_1/FVC showed weak or non-significant correlations with oscillometry, while FEF25–75 correlated moderately with Post R5–R20 (ρ = 0.32, p = 0.07) and Post X5insp (ρ = 0.27, p = 0.14), suggesting oscillometry's utility in detecting small airway obstruction. Overall, oscillometric measures reflecting reactance (X5, Ax) and frequency-dependent resistance (R5–R20) complement spirometry by providing insight into peripheral airway function, particularly in early or subtle disease (**Supplementary table 1**) (**Figure 1 and 2**).

Table 2: Spirometry and oscillometry parameters with COPD severity

	Moderate (N=8)	**Severe (N=16)**	**Very severe (N=7)**	**P value**
FEV1	57.05±2.58	40.29±4.78	25.43±4.45	0.001*
FVC	74.05±5.48	59.16±9.25	37.46±5.13	0.001*
FEV1/FVC	60.78±3.23	54.07±7.7	51.43±8.59	0.051
FEF 25-75	34.65±4.43	22.7±3.28	13.13±3.14	0.001*
R 5	4.91±0.84	5.41±0.921	7.14±1.36	0.001*
R 20	1.95±0.79	2.39±0.74	3.45±0.94	0.003*
R5-R20	2.95±0.57	3.01±0.73	3.68±0.80	0.136
X5 Post	-4.26 ± 1.73	-4.14± 2.07	-6.69 ± 3.22	0.059
X5 INSP	-3.19 ± 1.07	-3.74 ± 0.83	-4.48 ± 2.70	0.279
X5 exp	-5.03 ± 2.26	-5.37 ± 2.04	-7.68 ± 3.85	0.381
Post AX	46.95± 23.64	49.07±16.24	73.41±42.36	0.118

*Significant

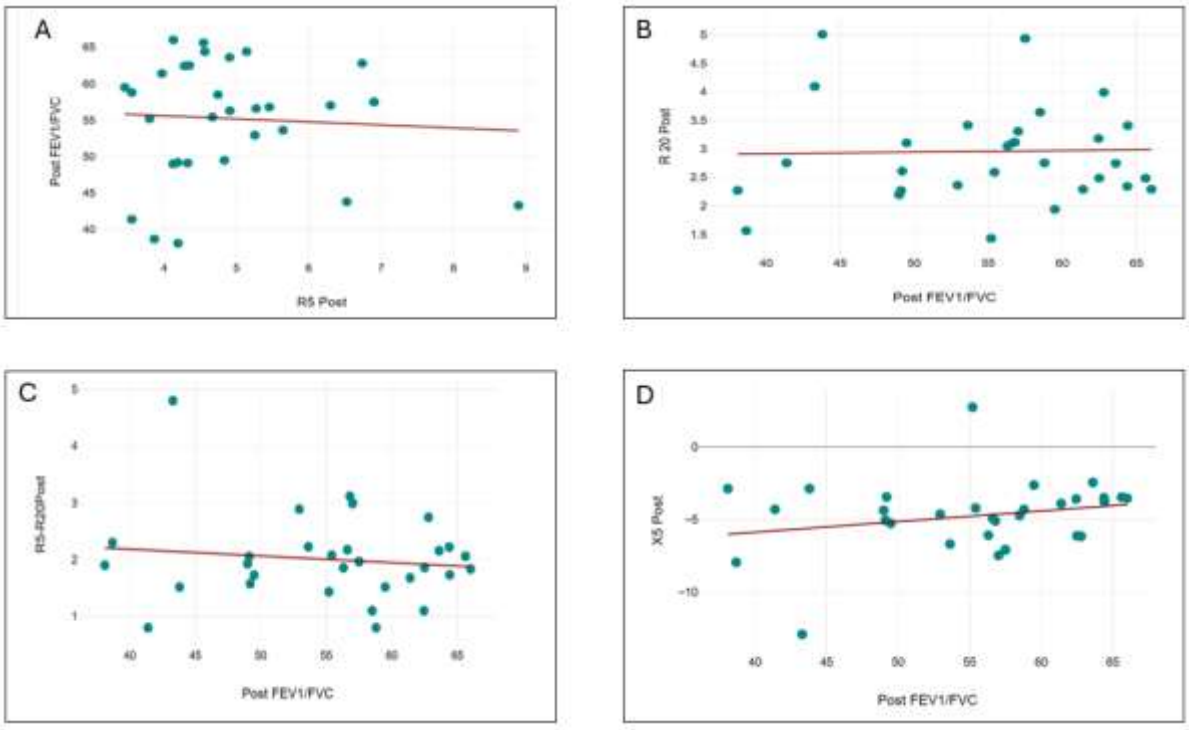

Figure 1: Scatter plots showing the relationship between post-bronchodilator FEV_1/FVC (%) and oscillometric parameters. (A) Post FEV_1/FVC vs.R5, (B) Post FEV_1/FVC vs. R20, (C) Post FEV_1/FVC vs. R5-R20 and (D) Post FEV_1/FVC vs. X5. The red line represents the linear regression fit.

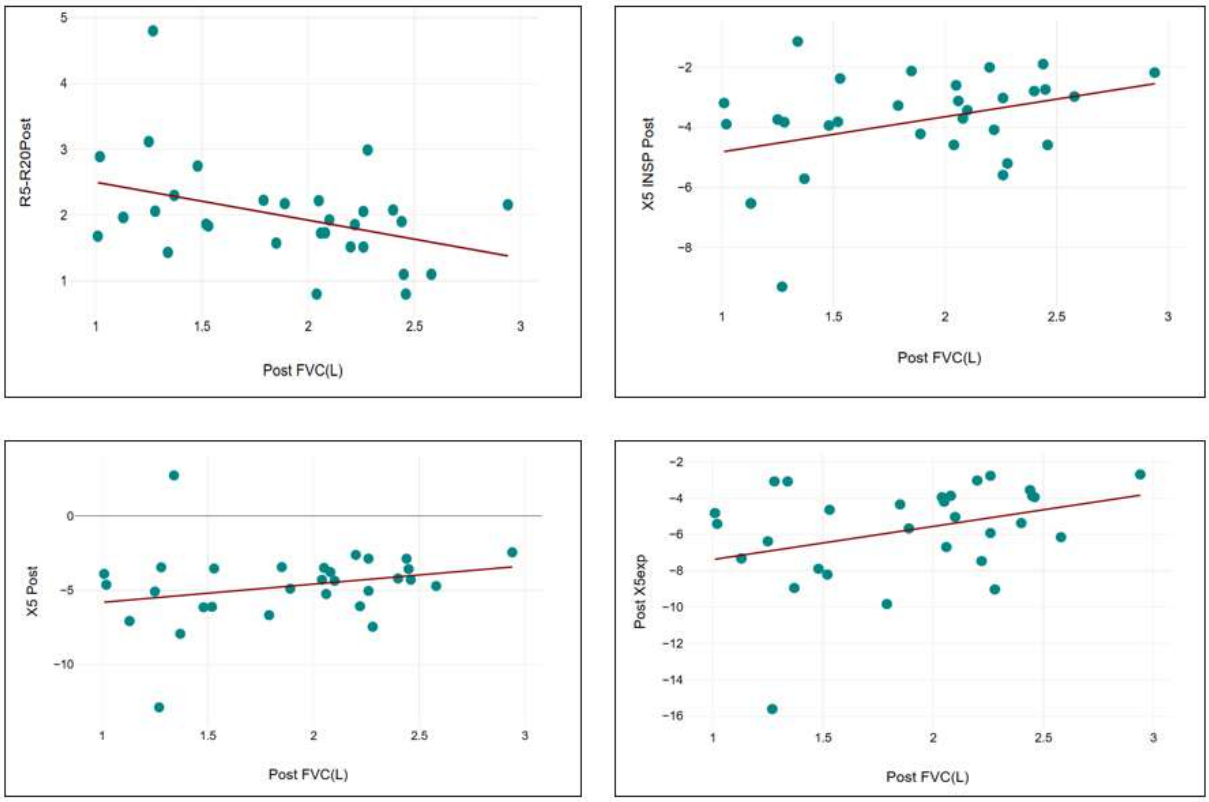

Figure 2: Post-bronchodilator spirometry and oscillometry parameters.

The study evaluated associations between spirometric and oscillometric parameters across common chest CT abnormalities in COPD patients. Patients with bullae exhibited the lowest post-bronchodilator FEV_1/FVC (48.6 ± 4.9) and FEV_1% predicted (38.5 ± 0.69), indicating marked obstructive impairment. Those with pulmonary artery hypertension (PAH) had the lowest FEV_1% (36.5 ± 0.6) and FEF25–75% (19.8 ± 5.4), reflecting small airway involvement. Fibrotic changes were associated with relatively preserved FEV_1/FVC (61.7 ± 4.4) and FVC (1.9 ± 0.53 L, FVC% 61.9 ± 8.1), consistent with a restrictive pattern. Bronchiectasis showed moderate impairments (FEV_1/FVC 55.4 ± 7.1; FEV_1% 43.6 ± 9.8) with elevated R5 (5.1 ± 1.2) and R20 (3.4 ± 0.6), suggestive of peripheral airway resistance. Patients with bronchial wall thickening demonstrated the highest Post Ax (70.5 ± 13.5) and Fres (24.3 ± 1.9), indicating increased lung inhomogeneity and impedance, while mosaic attenuation was associated with moderate airflow limitation (FEV_1/FVC 59.2 ± 5.1; FEV_1% 41.2 ± 11.8) and elevated oscillometric indices (Fres 49.3 ± 12.3; Ax 49.5 ± 21.3). These results highlight that distinct CT patterns correspond to specific physiological impairments,

particularly small airway dysfunction in structural abnormalities such as bullae, bronchiectasis, and PAH **(Table 3).**

Table 3: Association with HRCT parameters

Spirometry parameters	Bronchial wall thickening (n=4)	Mosaic attenuation (n=10)	Bronchiectasis (n=14)	>2 lobes (n=7)	Fibrosis (n=6)	PAH (n=3)	Bullae (n=3)
Post FEV1/FVC	57.7±0.92	59.2±5.1	55.45±7.1	58.7±2.8	61.7±4.4	59.5±4.1	48.6±4.9
Post FEV1 (L)	1.0±0.35	1.01±.38	1.0±0.26	1.0±0.34	1.2±0.27	1.0±0.08	1.02±0.99
Post FEV1 (%)	44.3±15.3	41.2±11.8	43.6±9.8	44.5±12.3	49.5±5.2	36.5±0.6	38.5±0.69
Post FVC (L)	1.8±0.6	1.7±0.56	1.98±0.48	1.8±0.58	1.9±.53	1.7±0.26	2.1±0.27
Post FVC (%)	61.2±20.3	52.6±13.2	1014±56.3	1.8±0.58	61.9±8.1	48.5±4.1	61.3±6.5
Post FEF 25-75 (L)	0.52±0.18	0.54±0.24	0.64±0.1	0.54±0.1	0.67±0.12	0.52±0.007	0.53±0.03
Post FEF 25-75(%)	26.7±13.5	2381±1.1	24.3±7.4	26.3±9.9	28.9±5.7	19.8±5.4	23.7±1.17
Post R5	4.9±1.1	5.1±1.1	5.1±1.2	5.6±1.4	4.9±1.2	4.8±0.65	5.4±1.3
Post R20	2.8±0.87	3.2±1.0	3.4±0.6	3.6±0.97	3.01±0.74	2.7±0.43	3.5±1.3
Post R5-R20	2.01±0.74	2.0±0.45	1.7±0.65	1.9±0.79	1.9±0.68	2.01±0.22	1.9±0.37
X5 post	-6.5±2.5	-4.7±1.8	-4.5±0.6	-5.6±1.6	-4.5±1.2	-5.0±0.86	-4.8±1.9
X5 INSP	-5.6±1.6	-.3.7±1.6	-4.0±1.3	-4.6±1.5	-4.3±1.2	-4.3±0.28	-3.9±1.4
X5 exp	-5.7±2.7	-5.6±2.5	-5.6±1.7	-6.5±2.0	-2.8±0.67	-6.9±1.7	-6.9±3.5
Post Ax	70.5±13.5	49.5±21.3	55.5±18.3	67.8±20.3	-5.4±1.7	53.1±1.3	44.01±16.2
Post FRES	24.3±1.9	49.3±12.3	33±13.8	49±3.2	41±12.3	26.9±11.7	29.7±4.8

The correlation between tracheal index and post-bronchodilator pulmonary function was assessed. Spirometric indices, including FEV_1, FEV_1/FVC, FVC, and FEF25–75, showed weak, non-significant associations with tracheal index (all $p > 0.05$). In contrast, oscillometry parameters demonstrated significant negative correlations, with post R5 ($\rho = -0.36$, $p = 0.04$) and post R20 ($\rho = -0.445$, $p = 0.02$), indicating that increased central and peripheral airway resistance is associated with reduced tracheal index. Other oscillometric measures (R5–R20, X5, AX, Fres) showed no significant correlations. These findings suggest that resistance-focused oscillometry parameters, particularly R5 and R20, may better reflect anatomical changes such as tracheal narrowing, whereas conventional spirometry is less sensitive to such structural alterations **(Table 4).** Post-bronchodilator spirometry and oscillometry were compared across increasing emphysema severity. FEV_1/FVC ratio declined significantly from 61.3 ± 3.6 in mild to 43.2 ± 6.8 in very severe emphysema ($p = 0.04$), with FEV_1% predicted (56.4 ± 3.7% to 24.0 ± 8.0%, $p = 0.03$) and FVC% predicted ($p = 0.03$) also showing significant reductions, reflecting progressive airflow limitation and reduced expiratory flow. FEF25–75 declined with severity but did not reach significance ($p = 0.53$). Among oscillometry parameters, Post AX increased significantly with severity ($p = 0.03$), indicating worsening small airway inhomogeneity, while Post R5, R5–R20, X5, and Fres worsened non-significantly. Overall, spirometry captured significant declines in expiratory function with emphysema progression, whereas oscillometry highlighted increasing small airway dysfunction and lung inhomogeneity (**Supplementary Table 2**).

Table 4: Correlation of Spirometry and oscillometry parameters with tracheal index

Spirometry and oscillometry parameters	**Tracheal index**	
	Spearman's rho	**P value**
Post FEV1/FVC	-0.19	0.28
Post FEV1(L)	-0.08	0.64
Post FEV1(%)	-0.04	0.98
Post FVC(L)	0.06	0.72
Post FVC(%)	0.169	0.36
Post FEF 25-75 (L)	-0.03	0.87
Post FEF 25-75(%)	0.04	0.82
Post R5	**-0.36**	**0.04**
Post R20	**-0.445**	**0.02**
Post R5-R20	-0.06	0.72
X5 post	0.05	0.78
X5 INSP	0.023	0.72
X5 exp	-0.18	0.52
Post Ax	0.057	0.39
Post FRES	0.07	0.17

Spirometry and oscillometry parameters were compared across three smoking categories: reformed smokers (n = 16), current smokers (n = 2), and non-smokers (n = 13). No statistically significant differences were observed in post-bronchodilator FEV_1/FVC, FEV_1, FVC, or FEF25–75 among the groups (all $p > 0.05$). Reformed smokers demonstrated the highest bronchodilator responsiveness in FEV_1 (7.53 ± 1.63%) and FEF25–75 (7.16 ± 15.84%), whereas current smokers showed minimal or negative responses, suggesting reduced airway reversibility in active smokers, though these trends were not statistically significant. Oscillometry parameters, including R5, R20, R5–R20, X5, AX, and Fres, showed no significant differences across smoking groups ($p > 0.05$). Reformed smokers exhibited slightly higher total and peripheral airway resistance (R5, R5–R20) and more negative reactance (X5), indicating increased small airway stiffness and inhomogeneity, while current smokers had the highest tracheal index, though none of these differences reached statistical significance. Overall, both spirometry and oscillometry demonstrated similar lung function across smoking categories, with trends suggesting better bronchodilator responsiveness in those who had quit smoking (**Table 5**).

Table 5: Spirometry, oscillometry and tracheal index parameters stratified by smoking status

Parameter	Reformed (n=16)	Current (n=2)	No Smoking (n=13)	P value
Pre FEV1/FVC (%)	54.24 ± 6.93	53.74 ± 10.80	56.89 ± 8.25	0.419
Post FEV1/FVC (%)	54.25 ± 9.14	52.90 ± 16.26	55.83 ± 6.90	0.945
Post FEV1 (Litres)	29.94 ± 15.37	48.50 ± 12.02	42.72 ± 11.53	0.055
Change in FEV1 (%)	7.53 ± 1.63	2.50 ± 1.26	5.98 ± 1.14	0.697
Post FVC (Litres)	1.71 ± 0.55	2.26 ± 0.29	1.89 ± 0.54	0.390
Post FEF25–75 (L/sec)	0.46 ± 0.17	0.64 ± 0.21	0.54 ± 0.21	0.211
Change in FEF25–75 (%)	7.16 ± 15.84	-1.00 ± 15.56	6.09 ± 14.38	0.842
Post R5 (kPa/L/s)	5.45 ± 1.60	4.06 ± 0.71	4.85 ± 0.89	0.351
Post bronchodilators change in R5 (%)	-7.25 ± 11.11	-19.00± 12.1	-7.05 ± 13.56	0.545
Post R20	3.23 ± 1.14	2.55 ± 0.29	2.89 ± 0.77	0.732
Post R5-R20	2.23 ± 0.96	1.51 ± 1.00	1.95 ± 0.61	0.577
Post X5 (kPa/L/s)	-5.63 ± 2.87	-3.89 ± 0.56	-4.41 ± 2.34	0.582
Post Ax (cmH_2O/L)	61.43 ± 37.14	39.97 ± 0.37	50.03 ± 21.31	0.642
Post Fres (Hz)	30.81 ± 4.73 (n=8)	32.41 ± 3.00 (n=2)	31.82 ± 4.83 (n=11)	0.853
Tracheal index	1.10 ± 0.17	1.28 ± 0.01	1.14 ± 0.19	0.441

DISCUSSION:

This study assessed the role of AOS in COPD and its correlation with spirometry and HRCT thorax findings. Despite these advantages, the clinical adoption of oscillometry is limited by the absence of universal reference values and inter-device variability. However, oscillometry demonstrates potential in assessing small airway dysfunction and differentiating COPD phenotypes (21–26).

In this study, 31 COPD patients were evaluated with a mean age of 65.3 ± 10.7 years, with 68% aged over 60, predominantly male (87.1%), and of normal BMI (23.5 ± 4.0 kg/m²). These findings align with a large Spanish cohort by Morena et al. (n = 73,901), which reported a mean age of 73 years and male predominance (76.8%) (31). Similarly, Wang et al. in the USA observed COPD with 77.8% of cases occurring in males, underscoring the gender disparity and symptom burden across age groups (32).

Tobacco use is the principal risk factor for COPD, with disease risk influenced by smoking duration, intensity, age of initiation, cumulative exposure, and abstinence and promoting disease progression (33). Several tools are available to assess breathlessness, with the Modified Medical Research Council (MMRC) dyspnea scale being one of the most widely applied (34). In the present study, most patients (54.8%) reported moderate dyspnea (Grade II) which were similar in the earlier study by Dawood et al., on COPD patients (35).

In this study, 51.6% of participants were reformed smokers, 41.9% were smokers, and 6.5% were current smokers. Bronchiectasis was observed in both smokers and non-smokers, with 41.2% of affected patients being never-smokers and an equal proportion being reformed smokers. However, Yang et al. reported 23,609 new cases (0.3%), showing higher risk in ex-smokers (aHR = 1.07) and current smokers (aHR = 1.06), greatest with ≥10 pack-years (aHR = 1.12) showing smoking is an independent risk factor, though bronchiectasis also occurs in never-smokers (36).

In the present study, spirometric indices (FEV_1, FVC, FEF25–75) declined progressively with increasing COPD severity, consistent with the expected pattern of airflow limitation. Similar findings were reported by Raj et al., who demonstrated a significant reduction in spirometric parameters across GOLD stages, confirming spirometry's role in grading COPD severity (37).

Oscillometric parameters in our study showed significant abnormalities with disease progression. Increased R5 and R20 reflected higher total and central airway resistance, while more negative X5 and

elevated AX indicated worsening small airway dysfunction. Gunawardana et al. demonstrated that AOS had a much higher feasibility rate (98%) compared to spirometry (68%), especially in younger children who struggled with forced maneuvers. They also reported significant correlations of oscillometric indices such as R5 and X5 with spirometric measures including FEV_1, FEF75, and FEV_1/FVC, and found that bronchodilator responses detected by spirometry corresponded with marked changes in oscillometric parameters (38).

This study reported a stronger association between FVC and oscillometric parameters (R5–R20, X5) than with FEV_1. This suggests that FVC may better reflect small airway dysfunction. Similar Grieg et al. reported that all AOS measurements were more sensitive than FEV_1 in detecting bronchodilator responsiveness among patients with moderate–severe COPD. Particularly, R5–R19 and AX showed stronger sensitivity compared to FEV_1, though R5–R19 exhibited greater variability due to being derived from two separate resistance values. These findings suggest that AOS can serve as a valuable adjunct to spirometry, especially for detecting small-airway dysfunction that may not be evident with conventional measures (39). These observations align with the work of Kolsum et al., who found oscillometry to be more sensitive than spirometry in detecting small airway impairment, particularly in early disease (40). A study by Chan et al. showed excellent reliability (ICC $\geq$ 0.80), demonstrating that oscillometry is a reproducible tool. These findings support the role of AOS in monitoring patients on biologic therapy, offering a sensitive alternative or complement to spirometry (41).

On CT correlation, we found emphysema to be associated with reduced FEV_1/FVC and FEV_1%, whereas bronchiectasis and bronchial wall thickening corresponded with higher resistance and reactance values. These findings are in line with those of Yamamoto et al. 2020, reported that together, HRCT and oscillometry provide complementary information, with reactance offering a non-invasive functional correlate to structural changes on HRCT. Vital capacity and FVC showed negative correlations with both oscillometric indices and HRCT fibrosis scores, indicating that worsening structural changes on HRCT were paralleled by greater functional impairment on oscillometry, consistent with restrictive ventilatory deficiency (42).

The smoking status did not yield statistically significant differences in our study, trends suggested impaired bronchodilator reversibility in current

smokers and increased reactance abnormalities in reformed smokers. A comparative study demonstrated that among children exposed to passive smoking, oscillometry was more sensitive in identifying airway changes. Specifically, higher values of reactance area, central airway resistance, and obstruction indices (Fres, X5, AX) were observed in the passive smoking group. These findings suggest that oscillometry may detect early or subtle airway impairment not captured by standard spirometry (43). Pisi et al. demonstrated that oscillometry is sensitive in detecting small airway dysfunction, with R5–R20 inversely correlating with FEV_3/FEV_6 and identifying aiway dysfunction even in heavy smokers with normal spirometry. A notable proportion of smokers (≥30 pack-years) showed elevated R5–R20, indicating early airway involvement (44). In parallel, Verma et al. further confirmed oscillometry's utility, reporting increased R5 and R20 and reduced X5 in smokers, including those with normal spirometry ($p < 0.001$) (45). Overall, our findings demonstrate that oscillometry complements spirometry and highlights its potential as a valuable adjunct in both clinical practice and research settings. However, certain limitations must be acknowledged. The study was conducted at a single center with relatively small sample size. Future multicentre studies with larger cohorts are warranted to validate these findings and strengthen the evidence for routine incorporation of AOS alongside spirometry.

Conclusion

Oscillometry demonstrated greater sensitivity than spirometry in detecting airway resistance and structural narrowing in COPD. While spirometric indices like FEV_1 and FEV_1/FVC showed expected trends of decline with disease severity and emphysema progression, oscillometry parameters such as R5–R20 and AX captured additional insights into peripheral airway involvement and lung inhomogeneity. The significant correlations between AOS parameters and both functional and anatomical indices highlight its value as a complementary tool for assessing disease severity and airway remodelling.

Reference:

1. Chan SMH, Selemidis S, Bozinovski S, Vlahos R. Pathobiological mechanisms underlying metabolic syndrome (MetS) in chronic obstructive pulmonary disease (COPD): clinical significance and therapeutic strategies. Pharmacology & Therapeutics. 2019 June 1;198:160–88.
2. Decramer M, Janssens W, Miravitlles M. Chronic obstructive pulmonary disease. Lancet. 2012;379(9823):1341–51.
3. 2025 GOLD Report. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the prevention, diagnosis and management of chronic obstructive pulmonary disease: 2025 report. Fontana, WI: GOLD; 2024.

4. Vogelmeier CF, Criner GJ, Martinez FJ, Anzueto A, Barnes PJ, Bourbeau J, et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease 2017 Report: GOLD Executive Summary. Eur Respir J. 2017 Mar;49(3):1700214.
5. Liu Y, Carlson SA, Watson KB, Xu F, Greenlund KJ. Trends in the Prevalence of Chronic Obstructive Pulmonary Disease Among Adults Aged ≥18 Years — United States, 2011–2021. MMWR Morb Mortal Wkly Rep. 2023 Nov 17;72(46):1250–6.
6. De Miguel-Díez J, Fernández-Villar A, Doña Díaz E, Padilla Bernáldez M, Trillo-Calvo E, Molina París J, et al. Chronic Obstructive Lung Disease: Treatment Guidelines and Recommendations for Referral and Multidisciplinary Continuity of Care. J Clin Med. 2024 Jan 5;13(2):303.
7. Agustí A, Celli BR, Criner GJ, Halpin D, Anzueto A, Barnes P, et al. Global Initiative for Chronic Obstructive Lung Disease 2023 Report: GOLD Executive Summary. Am J Respir Crit Care Med. 207(7):819–37.
8. Agarwal AK, Raja A, Brown BD. Chronic Obstructive Pulmonary Disease. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025.
9. Jung T, Vij N. Early Diagnosis and Real-Time Monitoring of Regional Lung Function Changes to Prevent Chronic Obstructive Pulmonary Disease Progression to Severe Emphysema. J Clin Med. 2021 Dec 12;10(24):5811.
10. Devine JF. Chronic Obstructive Pulmonary Disease: An Overview. Am Health Drug Benefits. 2008 Sept;1(7):34–42.
11. Rodrigues S de O, Cunha CMC da, Soares GMV, Silva PL, Silva AR, Gonçalves-de-Albuquerque CF. Mechanisms, Pathophysiology and Currently Proposed Treatments of Chronic Obstructive Pulmonary Disease. Pharmaceuticals. 2021 Oct;14(10):979.
12. Pride NB. TESTS OF FORCED EXPIRATION AND INSPIRATION. Clinics in Chest Medicine. 2001 Dec 1;22(4):599–622.
13. McFadden ER, Linden DA. A reduction in maximum mid-expiratory flow rate: A spirographic manifestation of small airway disease. The American Journal of Medicine. 1972 June 1;52(6):725–37.
14. Boggs PB, Bhat KD, Vekovius WA, Debo MS. Volume-adjusted maximal mid-expiratory flow (Iso-volume FEF25-75%): definition of "Significant" responsiveness in healthy, normal subjects. Ann Allergy. 1982 Mar;48(3):137–8.
15. Gelb AF, Williams AJ, Zamel N. Spirometry: FEV, vs FEF25-75 Percent. CHEST. 1983 Oct 1;84(4):473–4.
16. Sutherland ER, Martin RJ, Bowler RP, Zhang Y, Rex MD, Kraft M. Physiologic correlates of distal lung inflammation in asthma. Journal of Allergy and Clinical Immunology. 2004 June 1;113(6):1046–50.
17. Sorkness RL, Bleecker ER, Busse WW, Calhoun WJ, Castro M, Chung KF, et al. Lung function in adults with stable but severe asthma: air trapping and incomplete reversal of obstruction with bronchodilation. Journal of Applied Physiology. 2008 Feb;104(2):394–403.
18. Stanojevic S, Kaminsky DA, Miller MR, Thompson B, Aliverti A, Barjaktarevic I, et al. ERS/ATS technical standard on interpretive strategies for routine lung function tests. European Respiratory Journal. 2022 July 13;60(1).
19. Bhatt SP, Nakhmani A, Fortis S, Strand MJ, Silverman EK, Sciurba FC, et al. FEV1/FVC Severity Stages for Chronic Obstructive Pulmonary Disease. Am J Respir Crit Care Med. 2023;208(6):676–84.
20. Bhaskar R, Singh S, Singh P. Characteristics of COPD phenotypes classified according to the findings of HRCT and spirometric indices and its correlation to clinical characteristics. Afr Health Sci. 2018 Mar;18(1):90–101.
21. Brashier B, Salvi S. Measuring lung function using sound waves: role of the forced oscillation technique and impulse oscillometry system. Breathe. 2015 Mar 11;11(1):57–65.
22. Descatha A, Fromageot C, Ameille J, Lejaille M, Falaize L, Louis A, et al. Is Forced Oscillation Technique Useful in the Diagnosis of Occupational Asthma? Journal of Occupational and Environmental Medicine. 2005 Aug;47(8):847.

23. Dandurand RJ, Lavoie JP, Lands LC, Hantos Z, Group the OHS. Comparison of oscillometry devices using active mechanical test loads. ERJ Open Research. 2019 Dec 23;5(4).
24. DuBois AB, Brody AW, Lewis DH, Burgess BF. Oscillation Mechanics of Lungs and Chest in Man. Journal of Applied Physiology. 1956 May;8(6):587–94.
25. Oostveen E, MacLeod D, Lorino H, Farré R, Hantos Z, Desager K, et al. The forced oscillation technique in clinical practice: methodology, recommendations and future developments. European Respiratory Journal. 2003 Dec 1;22(6):1026–41.
26. Komarow HD, Myles IA, Uzzaman A, Metcalfe DD. Impulse oscillometry in the evaluation of diseases of the airways in children. Ann Allergy Asthma Immunol. 2011 Mar;106(3):191–9.
27. Starczewska-Dymek L, Bożek A, Dymek T. Application of the Forced Oscillation Technique in Diagnosing and Monitoring of Asthma in Preschool Children. Advances in Respiratory Medicine. 2019 Mar;87(1):26–35.
28. Crim C, Celli B, Edwards LD, Wouters E, Coxson HO, Tal-Singer R, et al. Respiratory system impedance with impulse oscillometry in healthy and COPD subjects: ECLIPSE baseline results. Respir Med. 2011 July;105(7):1069–78.
29. Gunawardana S, Tuazon M, Wheatley L, Cook J, Harris C, Greenough A. Airwave oscillometry and spirometry in children with asthma or wheeze. J Asthma. :1–13.
30. Wong A, Hardaker K, Field P, Huvanandana J, King GG, Reddel H, et al. Home-based Forced Oscillation Technique Day-to-Day Variability in Pediatric Asthma. Am J Respir Crit Care Med. 2019 May;199(9):1156–60.
31. Morena D, Izquierdo JL, Rodríguez J, Cuesta J, Benavent M, Perralejo A, et al. The Clinical Profile of Patients with COPD Is Conditioned by Age. J Clin Med. 2023 Dec 9;12(24):7595.
32. Wang Z, Li Y, Lin J, Huang J, Zhang Q, Wang F, et al. Prevalence, risk factors, and mortality of COPD in young people in the USA: results from a population-based retrospective cohort. BMJ Open Respir Res. 2023 July 14;10(1):e001550.
33. Rey-Brandariz J, Pérez-Ríos M, Ahluwalia JS, Beheshtian K, Fernández-Villar A, Represas-Represas C, et al. Tobacco Patterns and Risk of Chronic Obstructive Pulmonary Disease: Results From a Cross-Sectional Study. Archivos de Bronconeumología. 2023 Nov 1;59(11):717–24.
34. Sunjaya A, Poulos L, Reddel H, Jenkins C. Qualitative validation of the modified Medical Research Council (mMRC) dyspnoea scale as a patient-reported measure of breathlessness severity. Respiratory Medicine. 2022 Nov 1;203:106984.
35. Dawood HN, Kadhim AL, Ismail HJ. Usefulness of Modified Medical Research Council (MMRC) Dyspnea Scale for Assessment of Dyspnea Severity in Clinical Practice in COPD patients. 2021;44(06).
36. Yang B, Han K, Kim B, Kang HK, Kim JS, Kim EG, et al. Association between Smoking Status and Incident Non-Cystic Fibrosis Bronchiectasis in Young Adults: A Nationwide Population-Based Study. J Pers Med. 2022 Apr 26;12(5):691.
37. Raj D, Sharma GK, Lodha R, Kabra SK. Correlation between impulse oscillometry and spirometry parameters in Indian patients with cystic fibrosis. Chron Respir Dis. 2014 Aug 1;11(3):139–49.
38. Gunawardana S, Tuazon M, Wheatley L, Cook J, Harris C, Greenough A. Airwave oscillometry and spirometry in children with asthma or wheeze. J Asthma. :1–13.
39. Greig R, Kuo CR, Chan R, Lipworth B. Reversibility of airwave oscillometry in COPD. Respiratory Medicine [Internet]. 2025 Jan 1 [cited 2025 Sept 14];236. Available from: https://www.resmedjournal.com/article/S0954-6111(24)00390-1/fulltext
40. Kolsum U, Borrill Z, Roy K, Starkey C, Vestbo J, Houghton C, et al. Impulse oscillometry in COPD: identification of measurements related to airway obstruction, airway conductance and lung volumes. Respir Med. 2009 Jan;103(1):136–43.
41. Chan R, Lipworth B. Medium-term repeatability for airwave oscillometry in patients with severe asthma taking

benralizumab. J Allergy Clin Immunol Glob. 2023 Aug;2(3):100119.
42. Yamamoto Y, Miki K, Tsujino K, Kuge T, Okabe F, Kawasaki T, et al. Oscillometry and Computed Tomography Findings in Patients with Idiopathic Pulmonary Fibrosis. ERJ Open Research. 2020 Oct 8;6:00391–2020.
43. Schivinski CIS, de Assumpção MS, de Figueiredo FCXS, Wamosy RMG, Ferreira LG, Ribeiro JD. Impulse oscillometry, spirometry, and passive smoking in healthy children and adolescents. Pulmonol. 2017 Nov 1;23(6):311–6.
44. Pisi R, Aiello M, Frizzelli A, Calzetta L, Marchi L, Bertorelli G, et al. Detection of Small Airway Dysfunction in Asymptomatic Smokers with Preserved Spirometry: The Value of the Impulse Oscillometry System. Int J Chron Obstruct Pulmon Dis. 2021 Sept 14;16:2585–90.
45. Verma DS, Jain DP, Agrawal DP, Maheshwari DR, Khandelwal DA, Agrawal DA, et al. Utility Of Impulse Oscillometery In Early Detecting Of Small Airway Obstruction In Smokers. European Journal of Cardiovascular Medicine. 2024 Dec 3;14:421–4.

Supplementary table 1: Correlation of spirometry and oscillometry parameters

Variables	Correlation	Post R5	Post R20	PostR5-R20	Post X5insp	Post X5exp	Post Ax	Post Fres	Post X5
Post FEV1/FVC	r_s	0.03	0.08	-0.05	0.18	0.20	-0.21	-0.28	0.76
	p value	0.85	0.65	0.76	0.33	0.26	0.25	0.20	0.21
Post FEV1 (Litres)	r_s	-0.08	0.07	-0.15	0.17	0.13	0.48	0.49	0.05
	p value	0.65	0.68	0.38	0.36	0.47	0.08	0.14	0.33
Post FVC (L)	r_s	-0.21	0.04	**-0.356**	**0.34**	**-0.31**	0.14	**0.33**	**-0.51**
	p value	0.24	0.98	**0.05**	**0.05**	**0.04**	0.53	0.08	0.03
Post FEF25-75 (L)	r_s	-0.12	0.006	0.32	0.27	-0.256	-0.08	0.28	0.80
	p value	0.53	0.97	0.07	0.14	0.16	0.97	0.11	0.11
FEVI Post Percentage perdicted	r_s	-0.018	0.136	-0.16	0.376	0.14	-0.215	0.13	0.33
	P value	0.81	0.466	0.39	0.26	0.28	0.24	0.37	0.23
FVC post percentage predicted	r_s	-0.13	0.04	-0.30	0.28	0.23	-0.135	0.03	0.25
	p value	0.47	0.79	0.37	0.12	0.34	0.46	0.98	0.16
FEF 25-75 post percentage predicted	r_s	-0.08	0.01	-0.15	0.33	0.23	-0.20	0.13	0.28
	p value	0.65	0.94	0.41	0.06	0.21	0.27	0.79	0.17

r_s: **Spearman's rho**