RESEARCH PAPER

High Resolution Computed Tomography Assessment of Pulmonary Artery Diameters in Chronic Obstructive Pulmonary Disease: A Cross-Sectional Comparative Study

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder frequently associated with pulmonary vascular remodeling and pulmonary hypertension, both of which contribute to increased morbidity and mortality.

To evaluate pulmonary artery diameters using computed tomography in patients with chronic obstructive pulmonary disease, compare the findings with non-COPD participants, and assess the influence of smoking on pulmonary artery dimensions.

Materials and Methods: A hospital-based cross-sectional study was conducted among 200 participants (100 females and 100 males) aged 18–70 years. Chest CT examinations were performed using a standardized multidetector CT protocol. The diameters of the main pulmonary artery (MPA), left pulmonary artery (LPA), and right pulmonary artery (RPA) were measured on axial CT images. Participants were categorized into COPD and non-COPD groups and further classified according to smoking status. Descriptive statistics were calculated, and independent Student's *t*-tests were used to compare pulmonary artery diameters between groups. A p-value <0.05 was considered statistically significant.

Results: The mean age of the study population was 42.20 ± 15.91 years. The mean diameters of the MPA, LPA, and RPA were 26.36 ± 2.45 mm, 16.11 ± 1.68 mm, and 17.15 ± 1.61 mm, respectively. COPD patients demonstrated significantly larger pulmonary artery diameters than non-COPD participants, including the MPA (27.46 ± 2.51 mm vs. 25.47 ± 2.02 mm; $p < 0.01$), LPA (16.72 ± 1.46 mm vs. 15.61 ± 1.69 mm; $p < 0.01$), and RPA (17.85 ± 1.55 mm vs. 16.58 ± 1.43 mm; $p < 0.02$). Smokers also exhibited significantly greater pulmonary artery diameters than non-smokers, with the MPA measuring 27.48 ± 2.35 mm versus 25.26 ± 2.03 mm ($p < 0.001$). Histogram analysis demonstrated that most COPD patients had MPA diameters between 25 and 29 mm, while a subset exceeded the 29 mm threshold for pulmonary artery dilatation.

Conclusion: Pulmonary artery diameters measured by computed tomography are significantly increased in patients with COPD and in smokers, indicating pulmonary vascular remodeling associated with chronic lung disease and tobacco exposure. CT-based assessment of pulmonary artery dimensions, particularly the main pulmonary artery, represents a simple, reproducible, and non-invasive imaging biomarker that may facilitate the early identification of pulmonary vascular involvement and aid in the screening of patients at risk for pulmonary hypertension.

Keywords: Chronic obstructive pulmonary disease; Computed tomography; Main pulmonary artery; Pulmonary artery diameter; Pulmonary vascular remodeling; Smoking; Pulmonary hypertension.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation, chronic airway inflammation, and irreversible structural changes within the lungs. It represents one of the leading causes of morbidity and mortality worldwide, affecting more than 390 million individuals and accounting for over 3 million deaths annually. COPD imposes a substantial healthcare burden owing to frequent exacerbations, reduced quality of life, and the development of systemic complications, particularly cardiovascular and pulmonary vascular disorders. Although traditionally regarded as an airway disease, increasing evidence suggests that COPD is a complex multisystem disorder involving significant pulmonary vascular remodeling that contributes to disease progression and adverse clinical outcomes. [1–3]

Pulmonary vascular abnormalities in COPD arise from multiple pathophysiological mechanisms, including chronic hypoxemia, endothelial

dysfunction, inflammation, oxidative stress, destruction of the pulmonary capillary bed, and cigarette smoke-induced vascular injury. These mechanisms promote vasoconstriction, medial hypertrophy, intimal fibrosis, and increased pulmonary vascular resistance, eventually leading to pulmonary artery enlargement and pulmonary hypertension (PH). Pulmonary hypertension is a well-recognized complication of COPD and is associated with impaired exercise capacity, right ventricular dysfunction, increased frequency of exacerbations, hospitalization, and reduced ssurvival. [4–6] Early detection of pulmonary vascular involvement is therefore essential for improving clinical management and prognostic assessment.

Computed tomography (CT) has become an indispensable imaging modality for the comprehensive evaluation of COPD. Beyond the assessment of emphysema, airway wall thickening, and air trapping, multidetector CT enables accurate measurement of pulmonary artery dimensions and provides valuable information regarding pulmonary vascular morphology. Enlargement of the main pulmonary artery (MPA) and its branches has emerged as an important imaging biomarker of pulmonary vascular remodeling and has demonstrated strong associations with pulmonary hypertension, COPD severity, acute exacerbations, and mortality. Several studies have shown that CT-derived pulmonary artery measurements correlate with invasive hemodynamic parameters and may facilitate non-invasive identification of patients at risk for pulmonary hypertension.[7–10]

Among the CT-derived parameters, the diameter of the main pulmonary artery and the pulmonary artery-to-aorta (PA:A) ratio have been extensively investigated. Wells et al. demonstrated that a PA:A ratio greater than one independently predicts severe COPD exacerbations and hospitalization, highlighting its prognostic significance.[11] Similarly, Shen et al. reported significantly larger pulmonary artery diameters in patients with COPD compared with healthy controls and suggested that CT-based measurements could serve as useful indicators of pulmonary vascular involvement.[12] Recent studies have further emphasized that pulmonary artery enlargement reflects chronic pulmonary vascular remodeling and is associated with reduced pulmonary function, impaired gas exchange, and increased cardiovascular risk.[13–15]

Cigarette smoking remains the principal risk factor for COPD and plays a central role in pulmonary vascular injury. Chronic exposure to tobacco smoke induces oxidative stress, inflammatory cytokine release, endothelial dysfunction, and smooth muscle proliferation, resulting in structural remodeling of the pulmonary arteries even before clinically significant airflow obstruction develops. Imaging studies have demonstrated that smokers exhibit increased pulmonary artery diameters and higher PA:A ratios than non-smokers, suggesting that smoking independently contributes to pulmonary vascular abnormalities irrespective of COPD severity.[16–18] Consequently, evaluation of pulmonary artery dimensions in smokers may facilitate early identification of individuals at increased risk for pulmonary vascular disease.

Despite growing evidence supporting CT assessment of pulmonary artery enlargement, limited data are available regarding simultaneous evaluation of the main, right, and left pulmonary artery diameters among COPD patients in the Indian population. Furthermore, the influence of smoking on these individual pulmonary artery measurements has not been extensively investigated in regional clinical settings. Establishing population-specific reference values and understanding the relationship between pulmonary artery dimensions, COPD, and smoking may improve the diagnostic utility of CT in routine clinical practice.

Therefore, the present study aimed to evaluate pulmonary artery diameters using multidetector computed tomography in patients with COPD and compare these measurements with those of non-COPD participants. In addition, the study investigated the influence of smoking on pulmonary artery dimensions and examined the extent of pulmonary artery enlargement among COPD patients. The findings may contribute to the growing evidence supporting CT-derived pulmonary artery measurements as simple, reproducible, and non-invasive imaging biomarkers for pulmonary vascular remodeling and early identification of patients at risk of developing pulmonary hypertension.

Methods

Study Design

A cross-sectional observational study was conducted to evaluate pulmonary artery diameters on HRCT in patients with chronic obstructive pulmonary disease (COPD) and compare the measurements with those of non-COPD participants. The study also assessed the influence of smoking status on pulmonary artery dimensions.

Study Setting

The study was carried out in the Department of Radiodiagnosis at Primary Tertiary Hospital, India. All chest CT examinations were performed using a multidetector computed tomography (MDCT) scanner following a standardized imaging protocol. Data collection was conducted over the study period from [Month Year] to [Month Year].

Study Population

The study included 200 adult participants who underwent chest CT examination during the study period. Participants were categorized into two groups:

- COPD group: 89 participants diagnosed with COPD based on clinical findings according to

the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines.

- Non-COPD group: 111 participants without clinical or evidence of COPD who underwent CT examination for other non-pulmonary vascular indications.

Inclusion Criteria

Participants fulfilling the following criteria were included:

- Adults aged 18–70 years.
- Patients undergoing chest CT examination during the study period.
- Individuals without COPD for the comparison group.
- CT images of adequate quality for pulmonary artery measurements.
- Participants providing written informed consent.

Exclusion Criteria

- Congenital heart disease.
- Known pulmonary embolism.
- Interstitial lung disease.
- Previous pulmonary artery surgery.
- Mediastinal masses causing vascular compression.
- Severe motion artifacts or incomplete CT examinations.
- Inadequate clinical records.

Sample Size

A total of 200 participants were included using a consecutive sampling technique. The sample comprised 100 males and 100 females, ensuring balanced gender representation. The sample size was considered adequate to detect statistically significant differences in pulmonary artery diameters between study groups.

CT Imaging Protocol

Chest CT examinations were performed using a multidetector CT scanner under standardized acquisition parameters. Typical acquisition parameters included:

- Tube voltage: 120 kVp
- Tube current: Automated tube current modulation
- Slice thickness: 1–1.25 mm
- Reconstruction interval: 1 mm
- Pitch: 0.9–1.2
- Matrix size: 512 × 512
- Patient position: Supine
- Breath-hold: Full inspiration

Pulmonary Artery Measurements

Pulmonary artery measurements were performed on axial CT images using the institution's Picture Archiving and Communication System (PACS). Measurements were obtained by experienced radiologists blinded to participants' clinical information. The following vascular parameters were measured:

- **Main Pulmonary Artery (MPA):** Maximum transverse diameter measured at the level of the pulmonary artery bifurcation.
- **Right Pulmonary Artery (RPA):** Maximum internal diameter measured immediately distal to the bifurcation.
- **Left Pulmonary Artery (LPA):** Maximum internal diameter measured proximal to the first major branch.

Data Collection

The following variables were collected:

Demographic Variables

- Age
- Gender

Clinical Variables

- COPD status
- Smoking status

Radiological Variables

- Main pulmonary artery diameter (mm)
- Left pulmonary artery diameter (mm)
- Right pulmonary artery diameter (mm)

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Descriptive statistics for demographic and pulmonary artery measurements. Independent Student's t-test for comparison of mean pulmonary artery diameters between COPD and non-COPD groups. Independent Student's t-test for comparison between smokers and non-smokers. Frequency distribution and histogram analysis to evaluate the extent and distribution of pulmonary artery enlargement. A p-value <0.05 was considered statistically significant.

Result

The study population comprised 200 participants, with an equal distribution of 100 females (50.0%) and 100 males (50.0%). The mean age of the female participants was 42.78 ± 15.23 years, while the mean age of the male participants was 41.62 ± 16.61 years. The average ages of the two groups were comparable, indicating a balanced age distribution between female and male participants. This demographic balance minimizes the likelihood of age- and sex-related bias in the assessment of pulmonary artery diameters and subsequent analyses.

Table 1: Gender distribution of COPD and non-COPD participants.

Group	Female	Male
COPD	47	42
Non-COPD	53	58
Total	100	100

The table 1 presents the distribution of study participants according to gender and COPD status. Of the 200 participants, 100 were females, and 100 were males. The COPD and non-COPD groups were compared to evaluate whether gender distribution differed between the two groups. The frequencies and percentages of females and males in each group are presented to assess the representativeness of the study population and the potential influence of sex on pulmonary artery diameter measurements.

Table 2. Distribution of study participants according to age group and gender

Age Group	Female Count (% of Total)	Male Count (% of Total)	Combined Group Total
18–35 years	35 (17.5%)	46 (23.0%)	81 (40.5%)
36–50 years	26 (13.0%)	20 (10.0%)	46 (23.0%)
51–70 years	39 (19.5%)	34 (17.0%)	73 (36.5%)
Total	100 (50.0%)	100 (50.0%)	200 (100.0%)

Table 2 presents the demographic distribution of the study population by age group and gender. A total of 200 participants were included, comprising an equal number of females (n = 100, 50.0%) and males (n = 100, 50.0%). The largest proportion of participants belonged to the 18–35 years age group, accounting for 81 participants (40.5%), including 35 females (17.5%) and 46 males (23.0%). The 51–70 years age group constituted 73 participants (36.5%), with 39 females (19.5%) and 34 males (17.0%). The 36–50 years age group had the smallest representation, comprising 46 participants (23.0%), including 26 females (13.0%) and 20 males (10.0%). Overall, the study achieved an equal gender distribution while representing participants across all three age categories, with the highest representation in the younger adult age group.

Table 3: Descriptive statistics of age and pulmonary artery diameter measurements in the study population

Variable	Mean	SD	Minimum	Maximum
Age (years)	42.20	15.91	18.00	70.00
Main Pulmonary Artery (mm)	26.36	2.45	20.80	33.20
Left Pulmonary Artery (mm)	16.11	1.68	11.90	21.10
Right Pulmonary Artery (mm)	17.15	1.61	12.50	22.10

Table 3 summarizes the descriptive statistics for participants' age and pulmonary artery diameter measurements. The mean age of the study population was 42.20 ± 15.91 years, with an age range of 18 to 70 years. The main pulmonary artery (MPA) had a mean diameter of 26.36 ± 2.45 mm, with measurements ranging from 20.80 to 33.20 mm. The left pulmonary artery (LPA) demonstrated a mean diameter of 16.11 ± 1.68 mm, with values ranging from 11.90 to 21.10 mm. The right pulmonary artery (RPA) had a mean diameter of 17.15 ± 1.61 mm, with a range of 12.50 to 22.10 mm. Among the pulmonary artery measurements, the MPA exhibited the largest average diameter, followed by the RPA and LPA. These descriptive statistics provide an overview of the distribution and variability of the measured anatomical parameters within the study cohort.

Table 4. Comparison of pulmonary artery diameters between COPD and non-COPD groups.

Pulmonary Artery Segment	COPD Group (Mean ± SD)	Non-COPD Group (Mean ± SD)	t	p-value
Main Pulmonary Artery	27.46 ± 2.51	25.47 ± 2.02	6.07	<0.01
Left Pulmonary Artery	16.72 ± 1.46	15.61 ± 1.69	4.97	<0.01
Right Pulmonary Artery	17.85 ± 1.55	16.58 ± 1.43	5.94	<0.02

Table 4 compares the mean pulmonary artery diameters between participants with chronic obstructive pulmonary disease (COPD) and those without COPD. The main pulmonary artery (MPA) diameter was significantly greater in the COPD group (27.46 ± 2.51 mm) than in the non-COPD group (25.47 ± 2.02 mm) ($t = 6.07$, $p < 0.01$). Similarly, the left pulmonary artery (LPA) diameter was significantly larger in the COPD group (16.72 ± 1.46 mm) compared with the non-COPD group (15.61 ± 1.69 mm) ($t = 4.97$, $p < 0.01$). The right pulmonary artery (RPA) also demonstrated a significantly greater mean diameter in participants with COPD (17.85 ± 1.55 mm) than in those without COPD (16.58 ± 1.43 mm) ($t = 5.94$, $p < 0.02$). Overall, all three pulmonary artery segments exhibited significantly larger diameters in the COPD group, indicating an association between COPD and pulmonary artery enlargement, with the greatest

absolute difference observed in the main pulmonary artery.

Table 5: Comparison of pulmonary artery diameters between smokers and non-smokers.

Pulmonary Artery Segment	Smoker Group (Mean ± SD)	Non-Smoker Group (Mean ± SD)	t	p-value
Main Pulmonary Artery	27.48 ± 2.35	25.26 ± 2.03	7.13	< 0.001
Left Pulmonary Artery	16.66 ± 1.65	15.56 ± 1.54	4.87	<0.001
Right Pulmonary Artery	17.59 ± 1.51	16.71 ± 1.59	4.01	<0.001

Table 5 presents the comparison of pulmonary artery diameters between smokers and non-smokers. The main pulmonary artery (MPA) diameter was significantly higher in smokers (27.48 ± 2.35 mm) than in non-smokers (25.26 ± 2.03 mm) ($t = 7.13$, $p < 0.001$). Similarly, the left pulmonary artery (LPA) showed a significantly greater mean diameter among smokers (16.66 ± 1.65 mm) compared with non-smokers (15.56 ± 1.54 mm) ($t = 4.87$, $p < 0.001$). The right pulmonary artery (RPA) diameter was also significantly larger in smokers (17.59 ± 1.51 mm) than in non-smokers (16.71 ± 1.59 mm) ($t = 4.01$, $p < 0.001$). These findings indicate that smoking is associated with significantly increased diameters of the main, left, and right pulmonary arteries, with the greatest difference observed in the main pulmonary artery. The consistently low p-values demonstrate that these differences are statistically highly significant.

Table 6: Summary of pulmonary artery diameter measurements in COPD and non-COPD participants, including the influence of smoking status.

Pulmonary Artery	COPD vs. Non-COPD Findings	Smoking Status Findings	Interpretation
Main Pulmonary Artery (MPA)	Significantly larger in COPD patients (27.46 ± 2.51 mm vs. 25.47 ± 2.02 mm; $t = 6.07$, $p < 0.01$)	Significantly larger in smokers (27.48 ± 2.35 mm vs. 25.26 ± 2.03 mm; $t = 7.13$, $p < 0.001$)	MPA diameter is significantly increased in COPD patients and smokers, suggesting pulmonary vascular remodeling associated with chronic lung disease and smoking exposure.
Left Pulmonary Artery (LPA)	Significantly larger in COPD patients (16.72 ± 1.46 mm vs. 15.61 ± 1.69 mm; $t = 4.97$, $p < 0.01$)	Significantly larger in smokers (16.66 ± 1.65 mm vs. 15.56 ± 1.54 mm; $t = 4.87$, $p < 0.001$)	LPA enlargement is associated with COPD and smoking, indicating structural changes in the pulmonary arterial circulation.
Right Pulmonary Artery (RPA)	Significantly larger in COPD patients (17.85 ± 1.55 mm vs. 16.58 ± 1.43 mm; $t = 5.94$, $p < 0.02$)	Significantly larger in smokers (17.59 ± 1.51 mm vs. 16.71 ± 1.59 mm; $t = 4.01$, $p < 0.001$)	RPA diameter is also significantly increased in COPD patients and smokers, supporting the presence of pulmonary artery dilatation in COPD.

Table 6 examined the presence and extent of pulmonary artery enlargement in patients with COPD. Across all pulmonary artery segments, participants with COPD demonstrated significantly greater arterial diameters than those without COPD. The main pulmonary artery showed the largest difference between the two groups, followed by the right and left pulmonary arteries. Similar findings were observed when participants were stratified by smoking status, with smokers exhibiting significantly larger pulmonary artery diameters than non-smokers. These results suggest that COPD is associated with significant pulmonary artery enlargement, and smoking may further contribute to pulmonary vascular remodeling. The consistent statistical significance across all comparisons supports the hypothesis that pulmonary artery diameter measured on CT imaging may serve as a useful imaging biomarker for identifying vascular changes associated with COPD.

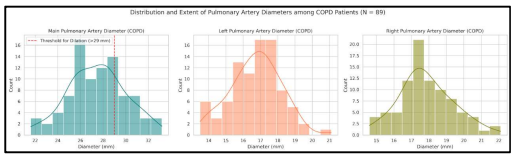

Figure 1: Distribution and extent of main, left, and right pulmonary artery diameters among patients with chronic obstructive pulmonary disease (COPD) (N = 89). Histograms with kernel density estimation (KDE) curves illustrate the distribution of pulmonary artery diameters. The red dashed vertical line in the main pulmonary artery (MPA) histogram represents the commonly accepted threshold for pulmonary artery dilatation (>29 mm).

The above Figure 1 illustrates the distribution of pulmonary artery diameters among the 89 participants diagnosed with COPD. The main pulmonary artery (MPA) diameter demonstrates an approximately normal distribution, with most measurements clustered between 25 and 29 mm. A subset of patients exhibited MPA diameters exceeding the 29 mm threshold, indicating pulmonary artery dilatation and suggesting the presence of pulmonary vascular remodeling or possible pulmonary hypertension in these individuals.

The left pulmonary artery (LPA) diameter also follows an approximately normal distribution, with the majority of values ranging from 16 to 18 mm and relatively few observations at the extremes. The distribution indicates limited variability and no marked outliers.

Similarly, the right pulmonary artery (RPA) diameter exhibits a near-normal distribution, with most measurements concentrated between 17 and 19 mm. The RPA demonstrates a slightly wider range than the LPA but remains symmetrically distributed without evidence of significant skewness.

Overall, the histograms demonstrate that pulmonary artery diameters in COPD patients are generally normally distributed, with enlargement being most evident in the main pulmonary artery. The presence of several MPA measurements above the established dilatation threshold supports the occurrence of pulmonary arterial enlargement in a subset of COPD patients. These findings are consistent with the quantitative analysis showing significantly larger pulmonary artery diameters among COPD patients compared with non-COPD participants and reinforce the potential value of CT-based pulmonary artery measurements as imaging biomarkers of pulmonary vascular involvement in COPD.

Discussion

The present study evaluated pulmonary artery diameters on HRCT in patients with chronic obstructive pulmonary disease (COPD) and compared these measurements with those of non-COPD participants. In addition, the influence of smoking on pulmonary artery dimensions was investigated. The principal findings demonstrated that the main pulmonary artery (MPA), left pulmonary artery (LPA), and right pulmonary artery (RPA) diameters were significantly larger in patients with COPD than in participants without COPD. Furthermore, smokers exhibited significantly greater pulmonary artery diameters than non-smokers. These observations suggest that pulmonary artery enlargement is closely associated with COPD and chronic tobacco exposure and support the utility of CT-derived pulmonary artery measurements as non-invasive imaging biomarkers of pulmonary vascular remodeling.

The demographic characteristics of the present study showed a balanced distribution of males and females, with both groups demonstrating comparable mean ages. Such demographic homogeneity reduces the influence of age- and sex-related confounding on vascular measurements and strengthens the internal validity of the comparisons. Previous population-based imaging studies have reported that pulmonary artery dimensions vary modestly with age and body habitus but are predominantly influenced by pulmonary vascular pathology rather than demographic characteristics alone.

The overall mean MPA diameter observed in the present study was 26.36 ± 2.45 mm, while COPD patients demonstrated a significantly larger mean diameter (27.46 ± 2.51 mm) compared with non-COPD participants (25.47 ± 2.02 mm). These findings agree with previous CT studies demonstrating enlargement of the pulmonary artery in COPD. Shen et al. reported that MPA, RPA, and LPA diameters were significantly greater in COPD patients than healthy controls and suggested that an MPA diameter of approximately 27.5 mm could aid in identifying COPD-related pulmonary vascular abnormalities.

The observed increase in pulmonary artery diameter is biologically plausible. COPD is increasingly recognized not only as an airway disease but also as a disorder involving substantial pulmonary vascular remodeling. Chronic hypoxia, endothelial dysfunction, destruction of the pulmonary capillary bed, inflammation, and increased pulmonary vascular resistance collectively contribute to progressive enlargement of the central pulmonary arteries. Wells and Dransfield emphasized that pulmonary artery enlargement represents an imaging manifestation of pulmonary vascular disease and is strongly associated with pulmonary hypertension, disease severity, and frequent COPD exacerbations.

Among the pulmonary artery segments, the main pulmonary artery exhibited the greatest absolute difference between COPD and non-COPD participants. This finding is consistent with earlier investigations showing that the MPA is the most

sensitive central vessel reflecting elevated pulmonary arterial pressure. Because the MPA receives the entire right ventricular stroke volume, vascular remodeling and increased pulmonary vascular resistance are often expressed initially as enlargement of this vessel before changes become apparent in peripheral branches.

The present study also demonstrated statistically significant enlargement of both the left and right pulmonary arteries in COPD patients. Although previous research has primarily emphasized MPA diameter and the pulmonary artery-to-aorta ratio (PA:A), several studies have reported that branch pulmonary arteries enlarge concurrently with increasing pulmonary vascular pressure. Shen et al. observed significantly larger right and left pulmonary artery diameters among COPD patients, particularly in individuals with coexisting pulmonary hypertension, supporting the findings of the current study.

Smoking emerged as another important determinant of pulmonary artery enlargement. Smokers exhibited significantly greater diameters of the MPA, LPA, and RPA than non-smokers, with the most pronounced difference observed in the MPA. Cigarette smoke induces oxidative stress, chronic inflammation, endothelial injury, and smooth muscle proliferation, resulting in pulmonary vascular remodeling even before advanced emphysematous destruction develops. Consequently, pulmonary artery enlargement may represent an early vascular manifestation of smoking-related lung injury. Recent CT-based imaging studies involving lung cancer screening populations similarly demonstrated increased MPA diameter and elevated PA:A ratios among chronic smokers, emphasizing the independent contribution of tobacco exposure to pulmonary vascular abnormalities.

An important observation in the present investigation was that several COPD patients exhibited MPA diameters exceeding the commonly accepted threshold of 29 mm, as illustrated by the histogram analysis. Although most measurements were clustered between 25 and 29 mm, a subset of patients demonstrated definite pulmonary artery dilatation. Previous reviews have shown that an enlarged pulmonary artery measured on CT is strongly associated with pulmonary hypertension across several chronic cardiopulmonary diseases. While pulmonary artery diameter alone is insufficient for definitive diagnosis, values exceeding established thresholds warrant further evaluation with echocardiography or right heart catheterization when clinically indicated.

The histogram distributions observed in this study demonstrated approximately normal distributions for the MPA, LPA, and RPA among COPD patients, indicating consistent measurement characteristics with relatively limited variability. The normal distribution of vessel diameters suggests that CT-derived pulmonary artery measurements provide reproducible quantitative parameters suitable for clinical assessment and research. Previous studies have likewise reported excellent interobserver and intraobserver reproducibility of pulmonary artery measurements obtained from multidetector CT, supporting their reliability in routine clinical practice.

The present findings have important clinical implications. Pulmonary hypertension is a frequent but often under-recognized complication of COPD and is associated with increased hospitalization, exercise limitation, right ventricular dysfunction, and mortality. Because echocardiography may be technically challenging in hyperinflated lungs and right heart catheterization is invasive, CT-based pulmonary artery measurements provide an attractive, non-invasive alternative for identifying patients requiring additional cardiovascular evaluation. Several investigators have suggested incorporating pulmonary artery measurements into routine chest CT interpretation for COPD patients, particularly when pulmonary hypertension is suspected clinically.

The results of the present study therefore reinforce the growing concept that COPD should be considered a pulmonary vascular disease in addition to an obstructive airway disorder. Pulmonary artery enlargement likely reflects cumulative vascular remodeling induced by chronic inflammation, smoking, hypoxia, and endothelial dysfunction. Consequently, quantitative CT assessment of pulmonary artery dimensions may contribute to early risk stratification, disease monitoring, and identification of patients at increased risk of pulmonary hypertension and adverse clinical outcomes.

Despite these encouraging findings, certain limitations should be acknowledged. This was a single-center cross-sectional study; therefore, causal relationships between COPD severity and pulmonary artery enlargement cannot be established. Pulmonary artery pressure was not confirmed using right heart catheterization, the reference standard for pulmonary hypertension. The study also did not evaluate pulmonary artery-to-aorta ratios, pulmonary function indices, or COPD severity classifications, which may provide additional prognostic information. Future multicenter prospective studies incorporating spirometry, echocardiography, right heart catheterization, quantitative emphysema assessment, and artificial intelligence-assisted CT analysis are warranted to establish standardized pulmonary artery reference values and improve prediction of pulmonary hypertension in COPD.

Overall, the present study demonstrates that CT-derived measurements of the main, left, and right pulmonary arteries are significantly greater in

COPD patients and smokers than in their respective comparison groups. These findings support the role of multidetector CT as a practical, reproducible, and non-invasive imaging modality for evaluating pulmonary vascular remodeling in COPD and suggest that pulmonary artery diameter may serve as a valuable imaging biomarker for the early identification of pulmonary hypertension and disease progression.

This single-centre, cross-sectional study cannot establish causality between COPD and pulmonary artery enlargement. Pulmonary hypertension was not confirmed using right heart catheterization or echocardiography, and COPD severity was not stratified. Additionally, smoking exposure was not quantified by pack-years, which may have influenced pulmonary artery measurements and limited the generalizability of the findings.

Conclusion

This study demonstrated that HRCT based pulmonary artery measurements are significantly associated with chronic obstructive pulmonary disease (COPD) and smoking status. The diameters of the main pulmonary artery (MPA), left pulmonary artery (LPA), and right pulmonary artery (RPA) were significantly larger in patients with COPD than in non-COPD participants. Similarly, smokers exhibited significantly greater pulmonary artery diameters than non-smokers, indicating that chronic tobacco exposure contributes to pulmonary vascular remodeling. Among the measured vessels, the main pulmonary artery showed the greatest degree of enlargement, with a subset of COPD patients demonstrating diameters exceeding the clinically accepted threshold of 29 mm, suggestive of pulmonary artery dilatation and potential pulmonary hypertension.

The findings highlight the potential of routine chest CT to serve not only as a tool for evaluating lung parenchymal abnormalities but also as a valuable, non-invasive modality for assessing pulmonary vascular changes in COPD. Quantitative pulmonary artery measurements may facilitate the early identification of patients at increased risk of pulmonary hypertension, allowing timely referral for further cardiopulmonary evaluation and appropriate clinical management. Overall, the study supports the incorporation of pulmonary artery diameter assessment into routine CT interpretation for COPD patients and emphasizes its role as a practical imaging biomarker for pulmonary vascular involvement. Future multicenter longitudinal studies integrating pulmonary function tests, echocardiography, right heart catheterization, and advanced imaging techniques are recommended to validate these findings and establish standardized CT reference values for pulmonary artery assessment in COPD.

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