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Research Article

Ct and Echocardiographic Correlation of Pulmonary Artery Enlargement and Right Ventricular Dysfunction in Copd Patients

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ABSTRACT

Objective: To evaluate the correlation between CT-detected pulmonary artery enlargement and echocardiographic evidence of RV dysfunction in patients with COPD.

Materials and Methods: This cross-sectional study was conducted at multiple tertiary care hospital over 12 months from February 2025 to January 2026 and included 180 patients with spirometrically confirmed COPD. All patients underwent chest CT for measurement of pulmonary artery diameter and pulmonary artery-to-aorta (PA:A) ratio, along with transthoracic echocardiography for assessment of RV function. RV dysfunction was defined using standard echocardiographic parameters including TAPSE, RV fractional area change, and estimated pulmonary artery systolic pressure (PASP). Statistical analysis was performed using SPSS 22 and a p-value <0.05 was considered significant.

Results: Pulmonary artery enlargement was observed in 45.6% of patients, while RV dysfunction was observed in 37.8%. Patients with enlarged pulmonary arteries had significantly higher PASP and lower TAPSE values compared to those with normal pulmonary artery size ($p<0.001$). A strong positive correlation was found between pulmonary artery diameter and PASP ($r = 0.64$), and a negative correlation with TAPSE ($r = -0.52$). CT-detected pulmonary artery enlargement was an independent predictor of RV dysfunction ($p<0.001$).

Conclusion: CT-detected pulmonary artery enlargement is significantly associated with echocardiographic RV dysfunction in COPD patients. CT may serve as a useful non-invasive marker for early detection of cardiopulmonary complications, allowing timely evaluation and management of high-risk patients.

Keywords: COPD, pulmonary artery enlargement, computed tomography, right ventricular dysfunction, echocardiography, pulmonary hypertension.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a major global health problem and remains one of the leading causes of morbidity and mortality worldwide. It is characterized by persistent respiratory symptoms and irreversible or partially reversible airflow limitation resulting from chronic airway inflammation, small airway remodeling and destruction of lung parenchyma. [(1)]Although COPD has traditionally been viewed as a disease confined to the lung, growing evidence has shown that it is a multisystem disorder with important cardiovascular consequences. [(2)]Among these extra-pulmonary complications, pulmonary vascular changes and right ventricular dysfunction are particularly significant because they contribute substantially to reduced exercise tolerance, frequent hospitalizations, poorer quality of life and increased mortality.[(3)]

In patients with COPD, chronic hypoxia, endothelial dysfunction, inflammation and loss of the pulmonary capillary bed leads to

increased pulmonary vascular resistance over time.Chronic vasoconstriction and remodeling of pulmonary arteries can lead to pulmonary hypertension.[4]Increased pulmonary artery pressure imposes excessive afterload on the right ventricle. Initially, this results in hypertrophy and increased contractility of the right ventricle, but eventually may lead to dilation and systolic dysfunction. [5]This progression, known as corpulmonale in its later stages, results in edema, increased dyspnea, reduced exercise tolerance and a poor prognosis.[6]

Because these cardiovascular changes may develop gradually and remain clinically silent in early stages, timely detection is essential.Pulmonary artery enlargement has emerged as an important imaging marker of pulmonary vascular involvement in COPD.[7]Dilation of the main pulmonary artery may result from elevated pulmonary arterial pressure, vascular remodeling, or increased pulmonary blood flow[8] High-resolution and multidetector computed tomography (CT) offers a safe and accurate noninvasive way for measuring pulmonary artery diameter and to detail thoracic anatomy.[9]

CT is frequently performed in COPD patients for evaluation of emphysema, recurrent infections, lung nodules, or unexplained worsening symptoms. This creates an opportunity to extract additional cardiovascular information without requiring separate invasive procedures.[10] Measurement of the main pulmonary artery diameter or the pulmonary artery-to-ascending aorta ratio has been increasingly studied as a surrogate marker for pulmonary hypertension and disease severity.[11]

At the same time, echocardiography remains one of the most practical and widely available tools for assessing right ventricular structure and function. It allows estimation of pulmonary artery systolic pressure, measurement of right ventricular size, tricuspid annular plane systolic excursion (TAPSE), right ventricular fractional area change, tissue Doppler velocities, and evaluation of associated tricuspid regurgitation. [12]Echocardiography is safe, reproducible and inexpensive and is highly useful in clinical practice. However, many patients with COPD, lung hyperinflation, chest wall dysfunction and acoustic windows may at times be suboptimal.[13]Even so, echocardiography continues to play a central role in identifying right heart strain and monitoring disease progression.[14]

The relationship between CT-detected pulmonary artery enlargement and echocardiographic evidence of right ventricular dysfunction has gained increasing interest in recent years. [15]If a strong correlation exists, CT findings could help clinicians identify COPD patients at risk of occult pulmonary hypertension or RV dysfunction, especially when echocardiography is technically difficult or not immediately available. [16]Pulmonary artery enlargement on CT may also serve as a useful warning sign prompting further cardiovascular evaluation. Since many COPD patients undergo chest CT during the course of their illness, using routinely available imaging data for risk stratification could improve efficiency and clinical decision-making.[17]

In developing countries, COPD continues to impose a substantial burden due to tobacco smoking, biomass fuel exposure, air pollution, occupational dust and delayed diagnosis. Many patients present late with advanced airflow limitation and previously unrecognized cardiovascular complications. [18]In such settings, affordable and accessible methods for early detection of pulmonary vascular disease are especially important.Once right ventricular dysfunction develops, disease progression and clinical deterioration accelerate.[19] This can lead to diminished quality of life, increased exacerbations, longer hospital stays and death. Early detection of right ventricular strain prior to the development of overt failure allows greater emphasis in treatment, including smoking cessation, bronchodilators, long-term oxygen therapy if required, pulmonary rehabilitation and more careful monitoring of the cardiovascular system.[20]

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Radiology in collaboration with the Department of Pulmonology and Cardiology at multiple tertiary care hospitals over a period of 12 months after approval from the institutional ethical review committee. The study aimed to evaluate the correlation between computed tomography (CT) evidence of pulmonary artery enlargement and echocardiographic findings of right ventricular dysfunction in patients diagnosed with chronic obstructive pulmonary disease (COPD). Written informed consent was obtained from all participants prior to

enrollment. Confidentiality of patient information was maintained throughout the study.

A total of 180 adult patients with clinically and spirometrically confirmed COPD were included using non-probability consecutive sampling. COPD diagnosis was established according to standard clinical criteria with post-bronchodilator spirometry demonstrating persistent airflow limitation (FEV1/FVC ratio <0.70). Patients aged ≥40 years of either sex who underwent both chest CT scan and transthoracic echocardiography within the same admission or within a maximum interval of two weeks were eligible for inclusion.

Patients were excluded if they had known congenital heart disease, significant left-sided valvular heart disease, ischemic cardiomyopathy with reduced left ventricular ejection fraction, previous pulmonary embolism, interstitial lung disease, active pulmonary tuberculosis, lung malignancy, connective tissue disease-associated pulmonary hypertension, chronic kidney disease preventing contrast imaging when indicated or inadequate echocardiographic windows precluding right ventricular assessment. Patients with acute hemodynamic instability were also excluded.

Detailed demographic and clinical data were recorded for each participant, including age, gender, body mass index, smoking history, biomass fuel exposure, duration of COPD, frequency of exacerbations, use of long-term oxygen therapy, comorbid diabetes mellitus, hypertension and current medications. Symptom severity was assessed using the Modified Medical Research Council (mMRC) dyspnea scale. Spirometric severity was classified according to Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging based on post-bronchodilator FEV1 values.

All CT examinations were performed using a multidetector CT scanner with patients in the supine position during inspiratory breath-hold. Non-contrast or contrast-enhanced chest CT was accepted depending on clinical indication, provided vascular structures were adequately visualized. Main pulmonary artery diameter was measured at the level of its bifurcation perpendicular to the long axis of the vessel. Ascending aortic diameter was measured on the same axial imageand the pulmonary artery to ascending aorta ratio (PA:A ratio) was calculated. Pulmonary artery enlargement was defined as main pulmonary artery diameter >29 mm in men, >27 mm in women, or PA:A ratio >1.0, according to accepted radiologic criteria. CT images were independently reviewed by two experienced radiologists blinded to echocardiographic findings, and the average measurement was used for analysis. Additional CT findings such as emphysema severity, bronchial wall thickening, hyperinflation, and bullous changes were also noted.

Transthoracic echocardiography was performed by an experienced cardiologist using a standardized protocol. Parameters assessed included right ventricular basal diameter, right atrial size, tricuspid annular plane systolic excursion (TAPSE), right ventricular fractional area change (RVFAC), tissue Doppler systolic velocity (S′ wave), presence and severity of tricuspid regurgitation, and estimated pulmonary artery systolic pressure (PASP) derived from tricuspid regurgitant jet velocity. Right ventricular dysfunction was defined by one or more of the following: TAPSE <17 mm, RVFAC <35%, reduced S′ velocity <9.5 cm/s, or right ventricular dilation

n with impaired systolic motion. Pulmonary hypertension was considered likely when estimated PASP exceeded 35 mmHg in the absence of significant left heart disease.

The primary outcome measure was the correlation between CT-derived pulmonary artery enlargement and echocardiographic indicators of right ventricular dysfunction. Secondary outcomes included association of pulmonary artery enlargement with COPD severity, frequency of exacerbations, oxygen saturation, and estimated pulmonary artery pressure.

Data were entered and analyzed using SPSS version 22. Continuous variables were expressed as mean ± standard deviation while categorical variables were presented as frequency and percentage. Independent samples t-test or Mann–Whitney U test was used for comparison of continuous variables where appropriate. Chi-square test was applied for categorical variables. Pearson or Spearman correlation coefficients were used to assess the relationship between CT measurements and echocardiographic parameters. Multivariable linear or logistic regression analysis was performed to adjust for potential confounders such as age, smoking status, COPD severity, and hypoxemia. A p-value >0.05 was considered statistically significant.

RESULTS

A total of 180 patients with clinically and spirometrically confirmed chronic obstructive pulmonary disease (COPD) were included in the study. The mean age of the study population was 61.7 ± 9.8 years, ranging from 42 to 82 years. There was a male predominance, with 126 patients (70.0%) being male and 54 patients (30.0%) female. Current or former smoking history was present in 122 patients (67.8%) while 58 patients (32.2%) had significant biomass fuel exposure. According to GOLD classification, 38 patients (21.1%) had mild disease, 56 (31.1%) moderate disease, 52 (28.9%) severe diseaseand 34 (18.9%) very severe disease.

Computed tomography (CT) demonstrated pulmonary artery enlargement in 82 patients (45.6%), while 98 patients (54.4%) had normal pulmonary artery dimensions. The mean main pulmonary artery diameter for the entire cohort was 29.4 ± 4.6 mm, and the mean pulmonary artery to ascending aorta (PA:A) ratio was 0.98 ± 0.18.

Echocardiographic evidence of right ventricular dysfunction was identified in 68 patients (37.8%). The most frequent abnormalities included reduced tricuspid annular plane systolic excursion (TAPSE), right ventricular dilatation, elevated pulmonary artery systolic pressure (PASP), and decreased right ventricular fractional area change (RVFAC). Mean estimated PASP was 38.6 ± 11.2 mmHg.

Table 1. Baseline Characteristics of Study Population

Variable	Value
Total patients	180
Mean age (years)	61.7 ± 9.8
Male gender	126 (70.0%)
Smoking history	122 (67.8%)
Biomass exposure	58 (32.2%)
Mean FEV1 (% predicted)	48.3 ± 15.7
Mean oxygen saturation (%)	91.6 ± 4.3

Table 2. CT and Echocardiographic Findings

Variable	Value
Mean pulmonary artery diameter (mm)	29.4 ± 4.6
PA enlargement on CT	82 (45.6%)
Mean PA:A ratio	0.98 ± 0.18
RV dysfunction on echocardiography	68 (37.8%)
Mean PASP (mmHg)	38.6 ± 11.2
Reduced TAPSE (<17 mm)	52 (28.9%)
RV dilatation	46 (25.6%)

Patients with pulmonary artery enlargement on CT had significantly higher rates of right ventricular dysfunction compared with those without enlargement (58.5% vs 20.4%, p<0.001). Similarly, elevated PASP was more common in patients with enlarged pulmonary arteries (48.9 ± 10.6 mmHg vs 30.1 ± 7.8 mmHg, p<0.001). Mean TAPSE was significantly lower in the pulmonary artery enlargement group (16.1 ± 3.2 mm vs 19.4 ± 2.8 mm, p<0.001).

Table 3. Comparison between Patients with and without Pulmonary Artery Enlargement

Variable	PA Enlarged (n=82)	Normal PA (n=98)	p-value
RV dysfunction	48 (58.5%)	20 (20.4%)	<0.001
Mean PASP (mmHg)	48.9 ± 10.6	30.1 ± 7.8	<0.001
Mean TAPSE (mm)	16.1 ± 3.2	19.4 ± 2.8	<0.001
RV dilatation	32 (39.0%)	14 (14.3%)	<0.001
Severe/very severe COPD	56 (68.3%)	30 (30.6%)	<0.001

Correlation analysis showed a significant positive relationship between pulmonary artery diameter and estimated PASP (r = 0.64, p<0.001)and a significant negative correlation between pulmonary artery diameter and TAPSE (r = -0.52, p<0.001). PA:A ratio also

positively correlated with right ventricular basal diameter ($r = 0.46$, $p<0.001$).

Multivariable logistic regression demonstrated that pulmonary artery enlargement on CT independently predicted right ventricular dysfunction after adjustment for age, smoking status, oxygen saturation, and COPD severity (adjusted OR 3.72, 95% CI: 1.89–7.31, $p<0.001$).

Patients with severe and very severe COPD had significantly larger pulmonary artery diameters and higher prevalence of right ventricular dysfunction than those with mild or moderate disease. Frequent exacerbators (≥2 exacerbations/year) also showed higher rates of pulmonary artery enlargement (61.2% vs 34.1%, $p=0.002$).

DISCUSSION

This study demonstrated a strong and statistically significant association between computed tomography (CT)-detected pulmonary artery enlargement and echocardiographic evidence of RV dysfunction in patients with chronic obstructive pulmonary disease (COPD). Patients with enlarged pulmonary arteries had significantly higher pulmonary artery systolic pressures, lower TAPSE values, increased RV dimensions, and a greater prevalence of overall RV dysfunction compared to those without pulmonary artery enlargement. These findings suggest that CT-based measurements of the pulmonary artery may serve as a useful surrogate marker of pulmonary vascular disease and RV dysfunction in COPD.

The pathophysiological basis of these findings is well established. COPD is characterized not only by airflow limitation but also by widespread pulmonary vascular remodeling. Chronic hypoxemia leads to hypoxic pulmonary vasoconstriction, endothelial dysfunction, and smooth muscle proliferation within the pulmonary arterial tree. Over time, destruction of the pulmonary capillary bed due to emphysema further increases pulmonary vascular resistance. This sustained pressure overload eventually leads to right ventricular hypertrophy and dilation, progressing to right heart failure in advanced disease stages. These mechanisms explain the close relationship observed between pulmonary artery enlargement and echocardiographic markers of RV dysfunction in our study.

Our findings are consistent with previous research demonstrating that pulmonary artery enlargement on CT is associated with pulmonary hypertension and adverse outcomes in COPD. Wells et al. reported that a pulmonary artery to ascending aorta (PA:A) ratio >1 is strongly associated with pulmonary hypertension and increased risk of exacerbations in COPD patients, supporting its role as a clinically relevant imaging biomarker.[21] Similarly, Devaraj et al. found that CT-measured pulmonary artery diameter correlates with mean pulmonary arterial pressure and can help identify patients with suspected pulmonary hypertension in chronic lung disease.[22]

Echocardiography remains the most widely used non-invasive tool for assessing right heart function; however, its accuracy in COPD patients can be limited due to hyperinflated lungs and poor acoustic windows. In this context, CT offers a complementary advantage because it is frequently performed in COPD patients for evaluation of parenchymal disease and can simultaneously provide cardiovascular information. Truong et al. demonstrated that increased pulmonary artery sizeon CT is independently associated with mortality and hospitalizations in COPD, highlighting its prognostic value beyond structural lung assessment.[23]

In our study, a strong positive correlation was observed between pulmonary artery diameter and estimated pulmonary artery systolic pressure, while an inverse correlation was noted with TAPSE, indicating worsening RV systolic function with increasing pulmonary artery size. These findings align with results from Shin et al., who reported that CT-derived pulmonary artery measurements correlate significantly with echocardiographic and hemodynamic markers of pulmonary hypertension.[24] This reinforces the concept that structural vascular changes seen on CT reflect functional cardiac impairment in COPD patients.

We also observed that patients with more severe COPD had significantly higher rates of pulmonary artery enlargement and RV dysfunction. This is consistent with the findings of Barr et al., who demonstrated that emphysema severity on CT is independently associated with pulmonary vascular changes and increased cardiovascular risk.[25] The progressive decline in lung function and increasing hypoxemic burden in advanced COPD likely accelerates pulmonary vascular remodeling, thereby worsening RV afterload and dysfunction.

An additional important finding in our study was the association between frequent exacerbations and pulmonary artery enlargement. Exacerbations are known to cause acute hypoxemia, systemic inflammation, and endothelial injury, all of which can contribute to worsening pulmonary vascular resistance. Donaldson et al. previously showed that frequent COPD exacerbations are associated with accelerated decline in lung function and increased mortality. [26]

From a clinical standpoint, the findings of this study have important implications. COPD is often regarded primarily as a respiratory disease; however, cardiovascular complications significantly contribute to morbidity and mortality. Early identification of pulmonary artery enlargement on CT could help clinicians identify patients at risk of developing RV dysfunction even before overt clinical signs of right heart failure appear. This may prompt earlier echocardiographic evaluation, closer follow-up, and more aggressive management strategies such as long-term oxygen therapy and pulmonary rehabilitation.

CONCLUSION

This study concludes that pulmonary artery enlargement on CT is significantly associated with right ventricular dysfunction on echocardiography in patients with COPD. CT-derived pulmonary artery measurements reflect underlying pulmonary vascular remodeling and right heart strain, making them a useful non-invasive indicator of cardiopulmonary involvement. Combined use of CT and echocardiography can help in early identification of high-risk COPD patients and may facilitate timely clinical management to improve outcomes.

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