

Echocardiographic Assessment of Pulmonary Artery Pressure and Its Association with Disease Severity in Chronic Obstructive Pulmonary Disease: A Prospective Observational Study

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung disease with clinically important cardiovascular comorbidity, significant morbidity and mortality. PH is a complication of COPD and linked to increased symptom burden, decreased exercise capacity, right-ventricular dysfunction and worse outcomes. Right-heart catheterization is an invasive procedure and is not indicated for routine examination and evaluation, so transthoracic echocardiography is a safer, non-invasive method to estimate pulmonary artery systolic pressure (PASP) and estimate the likelihood of PH. The aim of this study was to assess the echocardiographic estimation of PASP and its association with the severity of COPD (spiro) as assessed by spirometry. **Material and Methods:** This was a prospective observational study carried out in Respiratory Medicine Department, Noida International Institute of Medical Sciences (NIIMS), Noida, Uttar Pradesh, India. Sixty patients with spirometry-diagnosed COPD who met the eligibility criteria were enrolled, with no data missing. Severity of the disease was determined by post-bronchodilator FEV1 percentage predicted. Transthoracic echocardiograms (TTE) were performed in all patients. In this study, echocardiographic PH was defined as an estimated systolic pulmonary artery pressure (sPAP) of ≥ 30 mmHg. Frequencies and percentages were used for summarising data. **Results:** Among 60 patients, 25 (41.67%) met the study's echocardiographic threshold for PH. Mild, moderate, and severe pressure elevation was observed in 14 (23.33%), 6 (10.00%), and 5 (8.33%) patients, respectively. The frequency of echocardiographic PH increased across spirometric categories, occurring in 23.08% of patients with mild COPD, 46.67% with moderate COPD, 61.54% with severe COPD, and 66.67% with very severe COPD. **Conclusion:** Echocardiographic evidence of PH was observed in 41.67% of the study population and showed a descriptive increase with worsening airflow limitation. Transthoracic echocardiography may assist the initial evaluation of appropriately selected patients with COPD; however, definitive haemodynamic diagnosis requires right-heart catheterization.

Keywords: Chronic obstructive pulmonary disease; pulmonary hypertension; pulmonary artery systolic pressure; echocardiography; GOLD classification; right-ventricular dysfunction.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms and persistent airflow obstruction arising from airway and/or alveolar abnormalities. It remains a major global cause of morbidity and mortality and is frequently accompanied by comorbidities that influence symptoms, healthcare use, and prognosis.^[1] Cardiovascular disease is particularly relevant in COPD, and pulmonary hypertension (PH) due to lung disease and/or hypoxia is classified within Group 3 PH.^[2,3]

COPD-associated PH results from interacting mechanisms that include chronic alveolar hypoxia, hypoxic pulmonary vasoconstriction, endothelial dysfunction, pulmonary vascular remodeling, inflammation, and loss of the pulmonary vascular bed in emphysema.^[3-6] PH in COPD is usually mild to moderate, whereas severe PH occurs in a smaller subgroup and may represent a distinct pulmonary vascular phenotype.^[4-7] Even non-severe elevations in

pulmonary pressure can contribute to exertional dyspnoea, impaired exercise capacity, right-ventricular loading, and poorer clinical outcomes.^[3-6]

Right-heart catheterization is the reference standard for haemodynamic diagnosis of PH. Transthoracic echocardiography is recommended as the initial non-invasive test when PH is suspected because it can estimate the probability of PH and assess right-heart structure and function; however, individual PASP estimates may be imprecise, particularly in

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chronic lung disease, and should not be considered equivalent to invasive confirmation.^[2,8,9]

Published estimates of PH prevalence in COPD vary widely because of differences in disease severity, referral setting, altitude, case selection, diagnostic thresholds, and whether echocardiography or right-heart catheterization was used. A 2022 systematic review and meta-analysis found a substantial pooled burden of PH in COPD, with marked between-study heterogeneity.^[10] Better characterization of echocardiographic pulmonary vascular involvement across spirometric stages may therefore aid clinical recognition and risk assessment, while acknowledging the limitations of non-invasive estimates. The present study evaluated echocardiographically estimated PASP in patients with COPD and examined its distribution across GOLD spirometric severity categories.

Aim: To evaluate pulmonary artery pressure using transthoracic echocardiography in patients with chronic obstructive pulmonary disease and determine its association with disease severity.

Objectives

Primary objectives

1. To estimate pulmonary artery systolic pressure using transthoracic echocardiography in patients with chronic obstructive pulmonary disease.
2. To evaluate the distribution of echocardiographic pulmonary hypertension across disease severity categories classified according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD).

Secondary objective: To determine the prevalence of echocardiographic pulmonary hypertension among patients with chronic obstructive pulmonary disease.

MATERIALS AND METHODS

Study design and setting: This prospective observational study was conducted in the Department of Respiratory Medicine, Noida International Institute of Medical Sciences (NIIMS), Noida, Uttar Pradesh, India, over a period of 18 months. The study evaluated pulmonary artery systolic pressure using transthoracic echocardiography and described its distribution across disease severity categories in patients with COPD.

Study population: A total of 60 consecutive patients with spirometry-confirmed COPD attending the outpatient department or admitted to the Department of Respiratory Medicine during the study period were enrolled after satisfying the predefined eligibility criteria.

Inclusion criteria

- Age ≥ 40 years.
- Diagnosis of COPD based on clinical features and spirometric evidence of persistent airflow limitation.
- Stable COPD patients willing to participate.

- Written informed consent.

Exclusion criteria

- Congenital or acquired valvular heart disease.
- Significant left-ventricular systolic dysfunction.
- Interstitial lung disease.
- Chronic thromboembolic pulmonary hypertension.
- Pulmonary hypertension due to causes other than COPD.
- Connective-tissue disorders associated with pulmonary hypertension.
- Poor echocardiographic window.
- Refusal to participate.

Clinical evaluation: All participants were thoroughly evaluated with detailed clinical data being collected such as smoking history, duration of symptoms, occupational exposures and related co-morbidities. A general physical and systemic examination was carried out, paying particular attention to the cardiovascular and respiratory system.

Spirometric assessment: PFT was conducted by following the standard recommendations. FEV₁, FVC and FEV₁/FVC ratio were measured. The severity of the disease was graded using the GOLD spirometric grading which refers to percent of FEV₁ after bronchodilator.

Echocardiographic assessment: Transthoracic 2D Echo was performed in all patients. The peak velocity of the tricuspid regurgitant jet was measured and pulmonary artery systolic pressure was estimated by using the modified Bernoulli equation and the right atrial pressure according to the study protocol. Echocardiographic PH was operationally defined as sPAP ≥ 30 mmHg used for this study. This threshold is not the same as the current haemodynamic definition for PH (which requires right-heart catheterization). Where possible, right-ventricular size and function, right atrial dimensions, tricuspid regurgitation and associated cardiac abnormalities were examined.

Outcome measures: Primary outcome: PASP (estimated by echocardiography).

Secondary outcomes: Prevalence of echocardiographic PH per study threshold and the distribution of echocardiographic PH across the severity categories of COPD spirometry.

Statistical analysis: Data were entered into Microsoft Excel and analysed using the Statistical Package for the Social Sciences (SPSS). Categorical variables were summarized as frequencies and percentages. Because no inferential statistical test or effect estimate was available in the source data, the relationship between COPD severity and echocardiographic PH is presented descriptively.

RESULTS

Distribution according to COPD severity: A total of 60 patients with spirometry-confirmed COPD were included. Patients were categorized according to GOLD spirometric severity based on post-bronchodilator findings.

Table 1: Distribution of patients according to COPD severity

GOLD stage	Number	Percentage (%)
Mild (FEV ₁ $\geq 80\%$ predicted)	26	43.33
Moderate (FEV ₁ 50-79% predicted)	15	25.00
Severe (FEV ₁ 30-49% predicted)	13	21.67
Very severe (FEV ₁ $<30\%$ predicted)	6	10.00
Total	60	100

Echocardiographic findings: Estimated pulmonary artery pressure was below the study threshold in 35 patients

(58.33%), whereas 25 patients (41.67%) met the study's echocardiographic definition of PH.

Table 2: Echocardiographic findings

Finding	Number	Percentage (%)
Below study PH threshold	35	58.33
Echocardiographic PH	25	41.67
Total	60	100

Severity of pulmonary pressure elevation: Among the 25 patients meeting the study threshold, mild, moderate, and

severe pulmonary pressure elevation was observed in 14, 6, and 5 patients, respectively.

Table 3: Severity of pulmonary pressure elevation

Severity	Number	Percentage of total sample (%)
Mild	14	23.33
Moderate	6	10.00
Severe	5	8.33
Total meeting PH threshold	25	41.67

Echocardiographic PH according to COPD severity: Echocardiographic PH was observed in 6 of 26 patients (23.08%) with mild COPD, 7 of 15 patients (46.67%) with moderate COPD, 8 of 13 patients (61.54%) with severe

COPD, and 4 of 6 patients (66.67%) with very severe COPD. The proportion increased descriptively across worsening spirometric categories; no inferential test of trend was available.

Table 4: Echocardiographic PH according to COPD severity

COPD severity	Patients with PH	Percentage (%)
Mild	6/26	23.08
Moderate	7/15	46.67
Severe	8/13	61.54
Very severe	4/6	66.67

DISCUSSION

The present prospective observational study found that 25 of 60 patients (41.67%) met the study's echocardiographic threshold for PH. The proportion increased descriptively from 23.08% in mild COPD to 66.67% in very severe COPD. These findings indicate a substantial burden of echocardiographic pulmonary vascular involvement in this tertiary-care cohort, but they should be interpreted as a descriptive pattern because no formal test of association or trend was available.

The observed prevalence is broadly compatible with contemporary evidence. A systematic review and meta-analysis of COPD populations reported a pooled PH prevalence of approximately 39%, while documenting marked heterogeneity across studies.^[10] Such variability is expected because estimates depend on the severity and clinical setting of COPD, altitude, comorbidity exclusions, and, importantly, the diagnostic method and threshold used.^[3-6,10] Echocardiographic studies often report higher prevalence than catheter-based cohorts because echocardiography estimates pressure non-invasively and is vulnerable to measurement error and selection bias.^[2,8,9]

The increasing proportion of PH across worsening spirometric stages is biologically plausible. Chronic hypoxaemia promotes hypoxic pulmonary vasoconstriction and, over time, vascular remodeling; emphysema reduces the pulmonary capillary bed, while endothelial dysfunction and inflammation further increase pulmonary vascular resistance.^[3-6] Classic longitudinal and catheterization-based studies also showed that pulmonary pressure may increase during the natural history of severe COPD and that severe PH is uncommon but clinically important.^[7,11,12] Nevertheless,

the relationship between FEV₁ and pulmonary vascular disease is not uniform: contemporary reviews recognize that a severe pulmonary vascular phenotype may occur despite only moderate spirometric impairment.^[3-6]

Current haemodynamic definitions of PH are based on precise right-heart catheterisation, with a mean pulmonary arterial pressure (mPAP) > 20 mmHg defining PH; echocardiography, however, is used to estimate rather than confirm the diagnosis of PH. The operational threshold of sPAP ≥ 30 mmHg in this study is based on the original protocol and is not directly interchangeable with the current invasive definition of PH. The importance of this distinction is especially great in COPD, where hyperinflation, inadequate acoustic windows and suboptimal tricuspid regurgitation signals may diminish the accuracy of PASP estimation.^[8,9]

Although these are some of its limitations, TTE is clinically useful and should be used as a first step when PH is suspected. It can provide the evaluation of tricuspid-regurgitation velocity, right-ventricular size and function, right-atrial enlargement and, when needed, other cardiac causes of dyspnoea, and this may be used in conjunction with other symptoms, oxygenation, pulmonary-function tests, imaging, biomarkers and clinical context, and can also be used in association with right-heart catheterization, the latter being generally reserved for cases where the haemodynamic findings would alter management or be required for transplant evaluation or enrolment in specialist pathways.

The clinical significance of the recognition of PH in COPD is related to increased symptom burden, a reduced exercise tolerance, right-heart dysfunction, increased resource use, and worse survival rates. PH should be suspected when dyspnoea or functional limitation is more severe than airflow obstruction,

when the degree of hypoxaemia is very severe, or when the right-heart strain is present. In unselected COPD associated PH (COPD PH) the primary focus should be optimal treatment of the underlying lung disease and correction of hypoxaemia as indicated, rather than routine use of PH-targeted drugs.^[1-6]

The limitations of this study are that it had a prospective study design, COPD was spirometrically confirmed, the patients were systematically assessed by echocardiography, and the patients had a wide spectrum of airflow limitation. The study data also originated from tertiary care setting in India.

There are a number of restrictions that are of particular importance. The study sample was small and was restricted to one centre which was not representative and has restricted generalizability. The diagnosis of PH was made by echocardiographic sPAP threshold, instead of contemporary haemodynamic criteria, and right-heart catheterization was not used. An independent relationship between the severity of COPD and PH cannot be asserted due to a lack of inferential statistical analysis, confidence interval or multivariable adjustment. Other potential confounding factors were not analysed (age, sex, smoking burden, oxygen saturation, extent of emphysema, sleep-disordered breathing, thromboembolic disease, diffusion capacity and treatment status). Lastly, the cross sectional design did not allow for the evaluation of progression and clinical outcomes.

To sum up, the proportion of echocardiographic PH based on the study threshold was about two-fifths of this COPD cohort and gradually increased with increasing spirometric categories. The results provide clinical evidence for the presence of pulmonary vascular involvement in advanced COPD and highlight that echocardiography is not a diagnostic tool for haemodynamic confirmation, but a screening and probability-assessment technique.

CONCLUSION

Of 60 patients, 41.67% fulfilled the PH criteria established by this study using the echocardiographic parameters, the percentage of whom increased in a descriptive manner with increasing categories of spirometric severity. In appropriate patients with COPD, particularly those with symptoms or hypoxaemia that do not correspond to the severity of airflow limitation, transthoracic echos may help detect pulmonary vascular and right-heart involvement. The results should be taken with some caution as the echocardiogram does not give a definite diagnosis of haemodynamics and no inferential association was tested in this study. Larger multicentre investigations are needed which employ current echocardiographic probability criteria, use proper statistical analysis, and involve right-heart catheterisation if clinically appropriate.

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Conflicts of interest

There are no conflicts of interest.

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