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Pulmonary Hypertension and Cardiac Dysfunction Across GOLD Stages in Stable COPD: An Echocardiographic Cross-Sectional Study

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ABSTRACT

Background: Cardiovascular abnormalities may remain clinically silent in stable chronic obstructive pulmonary disease (COPD), yet substantially influence symptoms and prognosis. Data describing the full echocardiographic spectrum across spirometric GOLD grades are limited.

Objective: To determine the prevalence and pattern of cardiac dysfunction in clinically stable COPD and assess its relationship with airflow limitation and selected clinical factors.

Materials and Methods: This cross-sectional study included 385 adults aged 40 years or older with stable COPD. Participants underwent post-bronchodilator spirometry, clinical evaluation, electrocardiography, and two-dimensional transthoracic echocardiography. Major outcomes included echocardiographic pulmonary hypertension, right ventricular (RV) dilatation, reduced tricuspid annular plane systolic excursion (TAPSE), left ventricular (LV) systolic and diastolic dysfunction, and valvular abnormalities. Associations were examined using chi-square tests, Spearman correlation, and multivariable logistic regression.

Results: Mean age was 61.69 ± 8.03 years and 84.9% were male. Mild, moderate, severe, and very severe COPD accounted for 17.9%, 34.0%, 30.6%, and 17.4% of participants, respectively. Echocardiographic pulmonary hypertension was present in 79.2%, RV dilatation in 14.5%, reduced TAPSE in 22.1%, any LV diastolic dysfunction in 64.7%, and moderate/severe tricuspid regurgitation in 48.8%. At least one cardiac comorbidity was identified in 91.4%. Pulmonary hypertension increased from 44.9% in mild COPD to 100% in very severe COPD, while reduced TAPSE increased from 0% to 71.6% (both $p<0.001$). FEV1% predicted correlated inversely with pulmonary artery systolic pressure (rho=-0.72, $p<0.001$); FEV1/FVC correlated positively with TAPSE (rho=0.621, $p<0.001$). Each one-grade increase in GOLD severity independently increased the odds of any cardiac comorbidity (adjusted OR 5.60, 95% CI 2.62-11.97) and pulmonary hypertension (adjusted OR 4.54, 95% CI 2.78-7.42).

Conclusion: Cardiac involvement was highly prevalent in stable COPD and showed a strong severity-related gradient. Echocardiography may be particularly valuable in moderate-to-very-severe COPD and in patients with clinical features suggesting cardiocirculatory compromise.

Keywords: *Chronic obstructive pulmonary disease; pulmonary hypertension; echocardiography; right ventricular dysfunction; TAPSE; GOLD grade.*

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a heterogeneous respiratory disorder characterized by persistent respiratory symptoms and post-bronchodilator airflow limitation. Beyond the airways and alveoli, COPD has important systemic consequences and frequently coexists with cardiovascular disease.[1-4] These comorbidities can intensify breathlessness, reduce exercise tolerance, increase hospitalization, and complicate clinical assessment because symptoms of cardiac and pulmonary dysfunction often overlap.

Cardiac involvement in COPD includes pulmonary vascular disease, pulmonary hypertension, right ventricular remodeling or failure, left ventricular dysfunction, arrhythmia, and functional valvular abnormalities. Chronic hypoxemia, loss of pulmonary vascular bed, endothelial dysfunction, hyperinflation, altered intrathoracic pressure, systemic inflammation, and ventricular interdependence all contribute to this cardiopulmonary interaction.[5] Pulmonary hypertension and RV dysfunction are traditionally associated with advanced COPD, but echocardiographic studies indicate that subclinical biventricular abnormalities may appear earlier in the disease course.

Transthoracic echocardiography offers a practical, non-invasive method to estimate pulmonary artery pressure and evaluate chamber size, RV systolic performance, LV systolic and diastolic function, and valvular lesions. Standardized right-heart assessment, including tricuspid annular plane systolic excursion (TAPSE), improves the recognition of RV dysfunction.[6] An earlier Indian study demonstrated a linear rise in pulmonary hypertension with COPD severity, but its small sample size limited detailed stage-wise and multivariable analyses.[7]

The present study therefore evaluated a large cohort of clinically stable COPD patients across the full spirometric GOLD spectrum. The primary objectives were to characterize the prevalence and pattern of echocardiographic cardiac abnormalities, determine their relationship with airflow limitation, and assess whether age, smoking exposure, disease duration, and clinical symptoms were associated with cardiac comorbidity.

MATERIALS AND METHODS

Study design and setting

A hospital-based cross-sectional observational study was conducted in the Department of Respiratory Medicine, Kamla Nehru Chest Hospital, Dr. S. N. Medical College, Jodhpur, Rajasthan. Adults aged 40 years or older with COPD diagnosed according to GOLD criteria were enrolled during the approved study period. Only clinically stable patients without an acute exacerbation in the preceding four weeks were included.

Eligibility criteria

Patients were eligible when post-bronchodilator spirometry confirmed persistent airflow obstruction and they could complete echocardiographic evaluation. Major exclusions were a recent COPD exacerbation, known primary cardiac disease unrelated to COPD, primary pulmonary hypertension, advanced chronic kidney or liver disease, uncontrolled thyroid disease, malignancy with limited life expectancy, pregnancy or lactation, and inability to undergo spirometry or echocardiography.

Clinical and spirometric assessment

Demographic characteristics, smoking status, smoking duration, pack-years, duration of COPD, respiratory symptoms, exercise intolerance, exacerbation history, orthopnea or paroxysmal nocturnal dyspnea, peripheral edema, jugular venous pressure, and clinical signs of right-heart failure were recorded using a structured proforma. Resting oxygen saturation was measured, and electrocardiography was performed.

Post-bronchodilator forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and FEV1/FVC were measured by spirometry. Airflow limitation was classified into mild, moderate, severe, and very severe spirometric GOLD grades using FEV1 percentage predicted thresholds specified in the study protocol.[1]

Echocardiographic assessment

Two-dimensional transthoracic echocardiography was performed by a trained cardiologist using a standardized protocol. The examination included pulmonary artery systolic pressure (PASP), RV basal dimension, TAPSE, LV ejection fraction (LVEF), LV diastolic function, chamber dimensions, tricuspid and mitral regurgitation, and other structural abnormalities. For study analysis, echocardiographic pulmonary hypertension was defined as PASP >30 mmHg, RV dilatation as a basal RV dimension >41 mm, and reduced RV longitudinal systolic function as TAPSE <17 mm. LV systolic function was categorized by LVEF, while LV diastolic dysfunction was graded according to the reporting echocardiographic assessment.

Sample size and statistical analysis

Using an anticipated prevalence of cardiac abnormality of 52%, a 95% confidence level, and 5% absolute precision, the calculated sample size was 384; 385 patients were ultimately analyzed. Continuous variables were summarized as mean ± standard deviation and categorical variables as number and percentage. Chi-square tests examined associations between categorical variables. Spearman rank correlation assessed relationships between spirometric or clinical variables and echocardiographic measures. Multivariable logistic regression estimated independent predictors of any cardiac comorbidity and pulmonary hypertension, with adjusted odds ratios (ORs) and 95% confidence intervals (CIs). A two-sided p value <0.05 was considered statistically significant.

Ethical considerations

The study was approved by the Institutional Ethics Committee of Dr. S. N. Medical College, Jodhpur (reference SNMC/IEC/2026/3403-04). Written informed consent was obtained from all participants. Data were coded to preserve confidentiality, and no experimental intervention was performed.

RESULTS

Participant characteristics

A total of 385 clinically stable COPD patients were included. Mean age was 61.69 ± 8.03 years; 43.9% were aged 60-69 years and 84.9% were male. Current and former smokers represented 37.7% and 48.3% of the cohort, respectively. Nearly half had COPD for 6-10 years. Moderate and severe COPD constituted the largest spirometric groups. Baseline characteristics are summarized in Table 1.

Table 1. Baseline characteristics and spirometric distribution (n=385)

Characteristic	Value
Age, mean ± SD (years)	61.69 ± 8.03
Male sex	327 (84.9%)
Current smoker	145 (37.7%)
Former smoker	186 (48.3%)
Pack-years >20	195 (50.6%)
COPD duration >10 years	69 (17.9%)
Dyspnea	348 (90.4%)
Exercise intolerance/fatigue	293 (76.1%)
Resting SpO2 <90%	136 (35.3%)
GOLD mild	69 (17.9%)
GOLD moderate	131 (34.0%)
GOLD severe	118 (30.6%)
GOLD very severe	67 (17.4%)

Data are presented as n (%) unless otherwise stated. SpO2, peripheral oxygen saturation.

Overall echocardiographic abnormalities

Echocardiographic evidence of pulmonary hypertension was present in 305 patients (79.2%). PASP was 31-50 mmHg in 61.6% and 51-70 mmHg in 17.7%. RV dilatation was identified in 14.5% and reduced TAPSE in 22.1%. LVEF remained at least 50% in 92.7%; however, LV diastolic dysfunction was common, affecting 64.7% of participants. Tricuspid regurgitation was the predominant valvular abnormality, with moderate or severe regurgitation in 48.8%. Overall, 352 patients (91.4%) had at least one cardiac comorbidity (Table 2).

Table 2. Overall echocardiographic findings

Finding	n (%)
Pulmonary hypertension (PASP >30 mmHg)	305 (79.2)
PASP 31-50 mmHg	237 (61.6)
PASP 51-70 mmHg	68 (17.7)
RV dilatation (>41 mm)	56 (14.5)
Reduced TAPSE (<17 mm)	85 (22.1)
LVEF ≥50%	357 (92.7)
LVEF 40-49%	27 (7.0)
LVEF <40%	1 (0.3)

Finding	n (%)
Any LV diastolic dysfunction	249 (64.7)
Moderate/severe tricuspid regurgitation	188 (48.8)
Any cardiac comorbidity	352 (91.4)

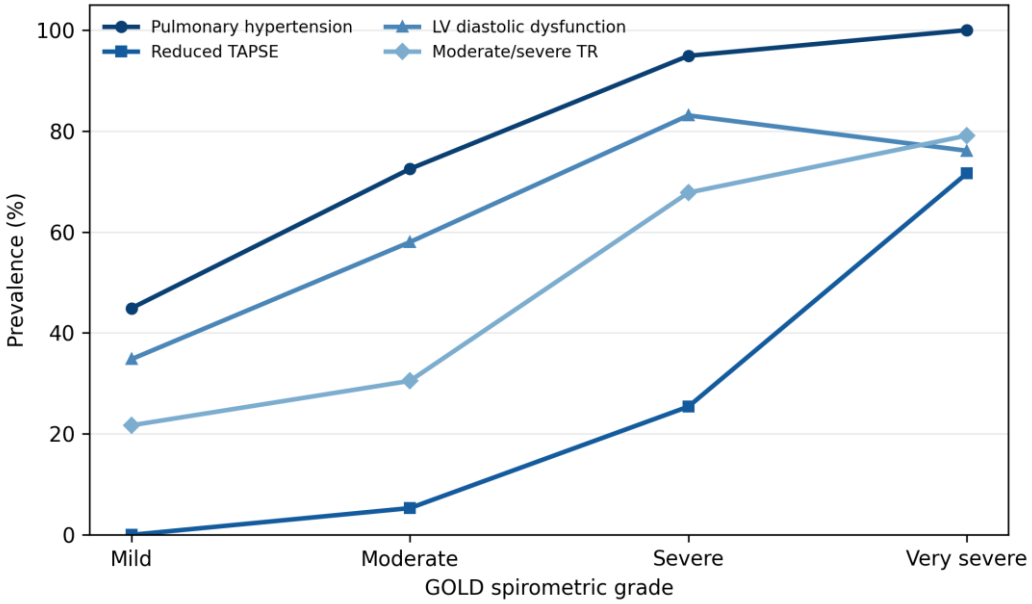
PASP, pulmonary artery systolic pressure; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; LVEF, left ventricular ejection fraction; LV, left ventricle.

Table 3. Major echocardiographic abnormalities across GOLD spirometric grades

Abnormality	Mild (n=69)	Moderate (n=131)	Severe (n=118)	Very severe (n=67)	p value
Pulmonary hypertension	31 (44.9%)	95 (72.5%)	112 (94.9%)	67 (100.0%)	<0.001
RV dilatation	0 (0.0%)	2 (1.5%)	24 (20.3%)	30 (44.8%)	<0.001
Reduced TAPSE	0 (0.0%)	7 (5.3%)	30 (25.4%)	48 (71.6%)	<0.001
LV diastolic dysfunction	24 (34.8%)	76 (58.0%)	98 (83.1%)	51 (76.1%)	<0.001
Moderate/severe TR	15 (21.7%)	40 (30.5%)	80 (67.8%)	53 (79.1%)	<0.001
Any cardiac comorbidity	48 (69.6%)	119 (90.8%)	118 (100.0%)	67 (100.0%)	<0.001

Values are n (% within GOLD grade). TR, tricuspid regurgitation.

Figure 1. Severity-related pattern of selected echocardiographic abnormalities



Pulmonary hypertension, reduced TAPSE, and moderate/severe tricuspid regurgitation rose sharply with worsening airflow limitation. LV diastolic dysfunction was most frequent in severe COPD and remained common in very severe disease.

Associations and correlation analysis

GOLD grade was significantly associated with every major echocardiographic outcome (all p<0.001). Pulmonary hypertension increased from 44.9% in mild COPD to 100% in very severe COPD. RV dilatation rose from 0% to 44.8%, reduced TAPSE from 0% to 71.6%, and moderate/severe tricuspid regurgitation from 21.7% to 79.1%. LV diastolic dysfunction increased from 34.8% in mild disease to 83.1% in severe COPD and remained frequent in very severe disease (76.1%).

A higher smoking burden, longer COPD duration, dyspnea, orthopnea or paroxysmal nocturnal dyspnea, and peripheral edema were associated with a greater prevalence of cardiac comorbidity. Cardiac abnormalities were present in 95.9% of patients with >20 pack-years (p=0.003), 98.6% of those with COPD duration >10 years (p=0.036), and all patients reporting orthopnea/paroxysmal nocturnal dyspnea or peripheral edema.

Spearman analysis demonstrated a strong inverse relationship between FEV1% predicted and PASP (rho=-0.72, p<0.001). Better preserved FVC and FEV1/FVC were associated with higher TAPSE, whereas pack-years and COPD duration correlated positively with PASP (Table 4).

Table 4. Correlation of pulmonary/clinical variables with echocardiographic measures

Variable 1	Variable 2	Spearman rho	p value	Direction
FEV1 % predicted	PASP (mmHg)	-0.720	<0.001	Negative
FVC % predicted	TAPSE (mm)	0.590	<0.001	Positive
FEV1/FVC ratio	TAPSE (mm)	0.621	<0.001	Positive
Pack-years	PASP (mmHg)	0.312	<0.001	Positive
COPD duration (years)	PASP (mmHg)	0.508	<0.001	Positive

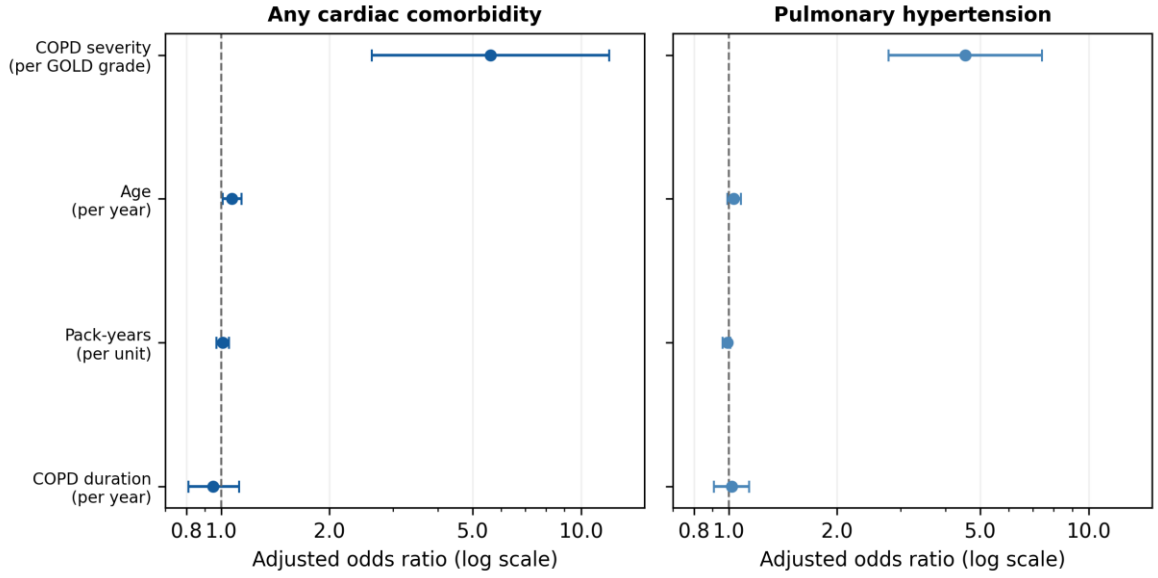
FEV1, forced expiratory volume in one second; FVC, forced vital capacity; PASP, pulmonary artery systolic pressure; TAPSE, tricuspid annular plane systolic excursion.

Table 5. Multivariable predictors of cardiac outcomes

Outcome	Predictor	Adjusted OR	95% CI	p value
Any cardiac comorbidity	COPD severity (per GOLD grade)	5.60	2.62-11.97	<0.001
Any cardiac comorbidity	Age (per year)	1.07	1.01-1.14	0.020
Any cardiac comorbidity	Pack-years	1.01	0.97-1.05	0.647
Any cardiac comorbidity	COPD duration (years)	0.95	0.81-1.12	0.525
Pulmonary hypertension	COPD severity (per GOLD grade)	4.54	2.78-7.42	<0.001
Pulmonary hypertension	Age (per year)	1.03	0.99-1.08	0.095
Pulmonary hypertension	Pack-years	0.99	0.96-1.01	0.267
Pulmonary hypertension	COPD duration (years)	1.02	0.91-1.14	0.770

OR, odds ratio; CI, confidence interval. COPD severity was entered as an ordinal one-grade increase.

Figure 2. Adjusted odds ratios for any cardiac comorbidity and pulmonary hypertension



The vertical line denotes an adjusted OR of 1. COPD severity was the dominant independent predictor in both regression models; age remained significant only for overall cardiac comorbidity.

DISCUSSION

This large cross-sectional study demonstrates that echocardiographic cardiac abnormalities are extremely common among clinically stable COPD patients and are strongly related to the severity of airflow limitation. More than nine of every ten participants had at least one cardiac comorbidity, four of five had echocardiographic pulmonary hypertension, nearly two thirds had LV diastolic dysfunction, and almost half had moderate or severe tricuspid regurgitation. The stage-wise gradient was particularly striking for pulmonary hypertension, RV dilatation, reduced TAPSE, and functional tricuspid regurgitation. After adjustment for age, smoking burden, and disease duration, spirometric GOLD grade remained the strongest independent predictor of both overall cardiac comorbidity and pulmonary hypertension.

The prevalence of echocardiographic pulmonary hypertension in this cohort (79.2%) was higher than the 42.5% reported in the original study by Gupta et al.[7] and the approximately 60% described in a stable community-based cohort by Fayngersh et al.[9] A recent systematic review likewise found substantial heterogeneity in reported pulmonary hypertension prevalence among COPD populations.[8] The higher estimate in our study is plausibly related to the large proportion of moderate-to-very-severe disease, the study definition based on PASP >30 mmHg, long-standing tobacco exposure, and the high frequency of resting oxygen saturation below 90%. Importantly, the prevalence rose from 44.9% in mild COPD to universal involvement in the very severe group, reinforcing the close relationship between pulmonary vascular loading and loss of lung function.

Freixa et al.[10] documented cardiac abnormalities in 64% of patients evaluated after a first severe COPD hospitalization, including frequent right-heart disease. Our study extends that observation to a stable outpatient population and shows a clear severity-response relationship. The strong inverse correlation between FEV1% predicted and PASP (rho=-0.72) also supports prior work showing that poorer spirometry, reduced exercise capacity, and desaturation identify patients at higher risk for pulmonary hypertension.[17] These findings are biologically consistent with hypoxic vasoconstriction, vascular remodeling, destruction of the pulmonary capillary bed, and increased RV afterload as COPD advances.

RV dilatation and reduced TAPSE were less frequent than pulmonary hypertension but showed the steepest stage-related increase. Reduced TAPSE was absent in mild COPD, present in only 5.3% of moderate disease, and rose to 71.6% in very severe COPD. Nasir et al.[12] similarly showed that abnormal conventional RV functional indices are common in COPD and carry prognostic significance. Sonaglioni et al.[11] reported impaired biventricular mechanics even without severe airflow obstruction, suggesting that myocardial dysfunction may precede overt chamber dilatation or reduced conventional systolic indices. Our results therefore support a continuum: pulmonary pressure elevation may appear early, whereas measurable RV systolic dysfunction becomes more prominent with advanced disease.

The high burden of LV diastolic dysfunction is another clinically important finding. Although 92.7% of participants had preserved LVEF, 64.7% had some degree of diastolic dysfunction. This pattern is consistent with reports that LV filling abnormalities may occur in COPD despite preserved systolic function. Mechanisms include reduced preload, hyperinflation-related cardiac compression, systemic inflammation, myocardial ischemia, hypoxemia, and ventricular interdependence. Xia et al.[13] demonstrated that RV pressure overload can adversely affect LV mechanics, while de Oliveira Caram et al.[14] found an association between LV diastolic dysfunction and COPD severity. In our cohort, LV diastolic dysfunction peaked in severe disease and remained highly prevalent in very severe disease, suggesting that left-heart involvement is not simply an incidental age-related finding.

Tricuspid regurgitation was the predominant valvular abnormality and moderate/severe regurgitation rose from 21.7% in mild disease to 79.1% in very severe COPD. In this setting, tricuspid regurgitation is most likely functional, reflecting elevated pulmonary pressure, RV remodeling, and annular dilatation rather than primary valvular pathology. Cuttica et al.[18] linked pulmonary arterial enlargement with adverse right-heart structural changes even in mild-to-moderate COPD. The parallel increase in PASP, RV dilatation, reduced TAPSE, and tricuspid regurgitation in our data provides a coherent echocardiographic picture of progressive right-sided pressure and volume stress.

Smoking exposure and disease duration were associated with cardiac abnormalities on bivariate analysis. Gao et al.[16] similarly observed that heavier smoking was related to lower FEV1 and more frequent pulmonary hypertension. However, pack-years and COPD duration were no longer independently significant in the adjusted models, whereas GOLD grade retained a large effect size. This suggests that part of the cardiovascular impact of cumulative exposure is mediated through the severity of established airflow limitation. Age independently predicted overall cardiac comorbidity but not pulmonary hypertension, implying that age-related myocardial and diastolic changes may contribute more broadly to the cardiac phenotype than to pulmonary vascular disease alone.

The overall prevalence of cardiac comorbidity was higher than in several earlier studies, including Indian and European series.[10,22,23] Differences in population selection, echocardiographic definitions, disease severity, and the inclusion of LV diastolic dysfunction and functional valvular lesions can substantially alter prevalence estimates. Nevertheless, the consistent message across studies is that clinically unrecognized cardiac disease is frequent in COPD. Macchia et al.[20] showed that unrecognized ventricular dysfunction carries prognostic implications, and Armentaro et al.[21] reported adverse cardiac events even in mild COPD patients with RV dysfunction. These observations strengthen the case for a lower threshold for cardiac assessment when symptoms appear disproportionate to spirometric impairment or when patients have hypoxemia, edema, orthopnea, recurrent exacerbations, or long-standing disease.

From a practical perspective, the data support targeted echocardiographic screening rather than reliance on LVEF alone. A normal ejection fraction did not exclude clinically relevant cardiac involvement, because pulmonary hypertension, RV dysfunction, diastolic dysfunction, and tricuspid regurgitation were far more common than overt LV systolic failure. Echocardiography may therefore add meaningful information to risk stratification in moderate-to-very-severe COPD and can help distinguish a cardiac contribution to persistent dyspnea or exercise limitation.

CONCLUSION

Cardiac involvement was highly prevalent among clinically stable COPD patients and was closely linked to worsening airflow limitation. Echocardiographic pulmonary hypertension, RV remodeling, reduced TAPSE, LV diastolic dysfunction, and functional tricuspid regurgitation were common, while overt LV systolic dysfunction was uncommon. GOLD severity independently predicted both overall cardiac comorbidity and pulmonary hypertension. These findings support incorporating echocardiography into the assessment of patients with moderate-to-very-severe COPD and of those with hypoxemia or symptoms suggestive of cardiac compromise.

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