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Original Article

A Prospective Study of ECG and Echocardiographic Findings in Chronic Obstructive Pulmonary Disease Patients in a Tertiary Care Hospital

Dr. Gayathri B1, Dr. S.A. Krithika2, Dr. T. Vivekanandan3

¹Assistant Professor, Department of Pulmonary Medicine, Madha Medical College and Research Institute, Chennai, Tamil Nadu, India
²Associate Professor, Department of Pulmonary Medicine, Panimalar Medical College and Hospital, Chennai, Tamil Nadu, India
³Assistant Professor, Department of General Medicine, Madha Medical College and Research Institute, Chennai, Tamil Nadu, India

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Corresponding Author:

Dr. Gayathri B

Assistant Professor, Department of Pulmonary Medicine, Madha Medical College and Research Institute, Chennai, Tamil Nadu, India

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease is a major cause of global morbidity and mortality, frequently associated with cardiovascular complications that remain underdiagnosed in routine clinical practice. Electrocardiographic and echocardiographic abnormalities are common in COPD patients but their prevalence and correlation with disease severity require further characterization in Indian tertiary care settings.

Objectives: To determine the prevalence and pattern of electrocardiographic and echocardiographic abnormalities in patients with COPD and to assess their correlation with disease severity as per GOLD classification.

Materials and Methods: A prospective observational study was conducted at a tertiary care hospital in Chennai from March 2024 to August 2024. Ninety patients diagnosed with COPD according to GOLD criteria underwent clinical evaluation, spirometry, 12-lead electrocardiography, and transthoracic echocardiography. ECG parameters included P wave morphology, QRS axis, rhythm abnormalities, and right ventricular hypertrophy. Echocardiographic evaluation assessed pulmonary artery systolic pressure, right ventricular dimensions and function, and left ventricular function. Statistical analysis employed Chi-square test, one-way ANOVA, and correlation analysis with p<0.05 considered significant.

Results: Mean age was 62.4±9.8 years with male predominance (73.3%). ECG abnormalities were present in 74 patients (82.2%), with P pulmonale (40.0%), right axis deviation (32.2%), and right ventricular hypertrophy (28.9%) being most common. Pulmonary hypertension was detected in 59 patients (65.6%). Mean PASP showed progressive increase: Stage I 30.1±9.2 mmHg, Stage II 33.9±9.9 mmHg, Stage III 42.0±13.2 mmHg, and Stage IV 48.5±13.3 mmHg (F=11.704, p<0.001). Right ventricular dysfunction was present in 27 patients (30.0%) and left ventricular diastolic dysfunction in 44 patients (48.9%). Significant correlation existed between pulmonary hypertension and GOLD stage (χ²=12.828, p=0.005).

Conclusion: Electrocardiographic and echocardiographic abnormalities are highly prevalent in COPD patients and correlate significantly with disease severity. Routine cardiovascular evaluation should be incorporated in COPD management to enable early detection of complications and guide therapeutic interventions.

Keywords: Chronic Obstructive Pulmonary Disease, Electrocardiography, Echocardiography, Pulmonary Hypertension, Right Ventricular Dysfunction, GOLD Classification.

INTRODUCTION

Chronic Obstructive Pulmonary Disease represents a major global health challenge, currently ranking as the third leading cause of death worldwide with approximately 3.23 million deaths annually.[1] The Global Burden of Disease Study estimates COPD affects over 384 million individuals globally, with considerable geographic variation in prevalence

rates.[2] In India, the burden is substantial, with prevalence estimates ranging from 6% to 10% among adults, affecting approximately 30 million individuals.[3]

COPD is characterized by persistent respiratory symptoms and airflow limitation due to airway and alveolar abnormalities, typically caused by significant exposure to noxious particles or gases.[4] Beyond its primary pulmonary manifestations, COPD is increasingly recognized as a systemic disease with significant cardiovascular involvement.[5] Cardiovascular disease represents the leading cause of mortality in patients with mild to moderate COPD and remains a major contributor to morbidity across all disease stages.[6]

The pathophysiological mechanisms linking COPD to cardiovascular complications involve chronic hypoxemia, systemic inflammation, oxidative stress, and mechanical effects of hyperinflation.[7] Pulmonary hypertension develops in approximately 20% to 90% of COPD patients depending on disease severity, primarily through hypoxic pulmonary vasoconstriction, structural remodeling of pulmonary vessels, and destruction of the pulmonary vascular bed.[8] This progression leads to right ventricular dysfunction and cor pulmonale, significantly impacting prognosis.

Electrocardiography serves as an accessible, non-invasive tool providing valuable diagnostic and prognostic information in COPD patients. Characteristic ECG changes include P pulmonale, right axis deviation, right ventricular hypertrophy, right bundle branch block, and low voltage QRS complexes.[9,10] However, reported prevalence of ECG abnormalities varies widely from 23% to 69%, reflecting differences in patient populations, disease severity, and diagnostic criteria.[11,12]

Transthoracic echocardiography has emerged as the primary non-invasive modality for screening pulmonary hypertension and assessing right ventricular function in COPD patients.[13] Echocardiography allows estimation of pulmonary artery systolic pressure, assessment of right ventricular size and function, and detection of concurrent left heart abnormalities.[14] Despite its clinical utility, echocardiographic evaluation is not routinely performed in COPD management in many settings, leading to delayed diagnosis of cardiovascular complications.

While international studies have documented cardiovascular abnormalities in COPD, data from Indian tertiary care settings remain limited. Furthermore, the relationship between COPD severity classified by GOLD criteria and prevalence of specific cardiovascular abnormalities requires further elucidation. This study was undertaken to determine the prevalence and patterns of electrocardiographic and echocardiographic abnormalities in COPD patients and assess their correlation with disease severity.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Pulmonary Medicine at Madha Medical College and Research Institute, a tertiary care teaching hospital in Chennai, Tamil Nadu, India, over six months from March 2024 to August 2024. The study protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants.

Patients aged 40 years and above, diagnosed with COPD based on GOLD criteria (post-bronchodilator FEV₁/FVC <0.70), clinically stable for at least 4 weeks, and willing to provide consent were included. Patients with pre-existing cardiac diseases, other chronic respiratory diseases, acute exacerbation at evaluation, poor echocardiographic window, or unwilling to participate were excluded.

Sample size was calculated based on previous studies reporting 50% prevalence of ECG abnormalities in COPD patients, with 95% confidence level and 10% allowable error, yielding minimum sample size of 96 patients. Ninety patients who met inclusion criteria and completed the study protocol were enrolled.

All enrolled patients underwent comprehensive clinical evaluation including detailed history, smoking history quantified in pack-years, and physical examination. Post-bronchodilator spirometry was performed using calibrated computerized spirometer according to ATS/ERS guidelines. Patients were classified per GOLD severity: Stage I (Mild) $FEV_1 \ge 80\%$ predicted, Stage II (Moderate) $50\% \le FEV_1 < 80\%$, Stage III (Severe) $30\% \le FEV_1 < 50\%$, Stage IV (Very Severe) $FEV_1 < 30\%$ predicted.

Standard 12-lead ECG was performed with patients supine after 10 minutes rest, recorded at 25 mm/second paper speed and 10 mm/mV standardization. Two independent cardiologists blinded to clinical data interpreted ECGs. Parameters assessed included P wave morphology (P pulmonale defined as amplitude ≥2.5 mm in leads II, III, or aVF), QRS axis (right axis deviation if >+90°), right ventricular hypertrophy using standard criteria, right bundle branch block, ST-T wave changes, and arrhythmias.

Transthoracic echocardiography was performed by experienced echocardiographer using standard ultrasound machine with 2.5-5 MHz phased array transducer. Pulmonary artery systolic pressure was estimated using modified Bernoulli equation from peak tricuspid regurgitation velocity: $PASP = 4V^2 + RAP$. Pulmonary hypertension was defined as PASP

≥35 mmHg, classified as mild (35-49 mmHg), moderate (50-69 mmHg), or severe (≥70 mmHg). Right ventricular internal diameter, right atrial area, tricuspid annular plane systolic excursion, left ventricular ejection fraction, and diastolic function were assessed.

Data were entered in Microsoft Excel and analyzed using SPSS version 26.0 and R version 4.3.0. Continuous variables were expressed as mean \pm standard deviation and compared using independent t-test or one-way ANOVA. Categorical variables were presented as frequencies and percentages and compared using Chi-square test. Pearson correlation coefficient assessed linear relationships. All statistical tests were verified using R programming. P-value <0.05 was considered statistically significant.

RESULTS

The study enrolled 90 COPD patients with mean age 62.4±9.8 years (range 42-82 years). Male patients constituted 73.3% (66 patients) of the cohort. The majority belonged to age group 60-69 years (38.9%), followed by 50-59 years (31.1%). Current or former smokers comprised 76.7% with mean smoking history 24.6±12.3 pack-years.

Disease severity distribution showed GOLD Stage II (Moderate) most prevalent at 38.9%, followed by Stage III (Severe) 32.2%, Stage I (Mild) 18.9%, and Stage IV (Very Severe) 10.0%. Mean FEV₁ percentage predicted was $52.3\pm18.7\%$ and mean FEV₁/FVC ratio 0.58 ± 0.12 .

Table 1: Demographic and Clinical Characteristics of Study Population (N=90)

Variable	Category	Frequency (%) / Mean ± SD
Age (years)	Mean \pm SD	62.4 ± 9.8
	40-49	8 (8.9%)
	50-59	28 (31.1%)
	60-69	35 (38.9%)
	≥70	19 (21.1%)
Gender	Male	66 (73.3%)
	Female	24 (26.7%)
Smoking Status	Current/Former Smoker	69 (76.7%)
	Non-smoker	21 (23.3%)
Pack-years (smokers)	Mean \pm SD	24.6 ± 12.3
GOLD Stage	Stage I (Mild)	17 (18.9%)
	Stage II (Moderate)	35 (38.9%)
	Stage III (Severe)	29 (32.2%)
	Stage IV (Very Severe)	9 (10.0%)
FEV1 (% predicted)	$Mean \pm SD$	52.3 ± 18.7
FEV1/FVC ratio	$Mean \pm SD$	0.58 ± 0.12
Total		90 (100%)

Table 1 shows demographic and clinical characteristics of 90 COPD patients with mean age 62.4 ± 9.8 years and male predominance (73.3%). Distribution across GOLD stages revealed majority had moderate to severe disease (71.1% in Stages II-III), with mean FEV₁ of $52.3\pm18.7\%$ predicted indicating significant airflow obstruction.

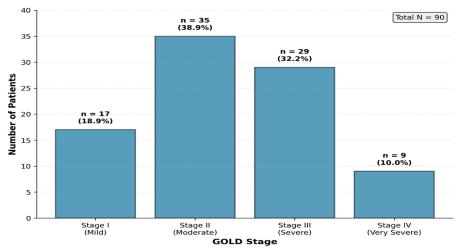


Figure 1: Distribution of Study Population across GOLD Stages (N=90)

Figure 1 shows the distribution of 90 COPD patients across GOLD severity stages with predominance of moderate to severe disease (71.1% in Stages II-III), reflecting tertiary care referral patterns with advanced cases requiring specialized management.

ECG abnormalities were detected in 74 patients (82.2%). P pulmonale, characterized by tall peaked P waves (≥2.5 mm) in inferior leads, was the most common finding, present in 36 patients (40.0%). Right axis deviation (QRS axis >+90°) was observed in 29 patients (32.2%), suggesting rightward displacement of cardiac axis due to right ventricular enlargement. Right ventricular hypertrophy, diagnosed using established voltage criteria, was present in 26 patients (28.9%), directly reflecting chronic pressure overload on the right ventricle. Right bundle branch block was identified in 18 patients (20.0%), indicating interventricular conduction delay. ST-T wave changes, primarily in right precordial leads, were noted in 22 patients (24.4%). Low voltage QRS complexes in limb leads, attributed to hyperinflated lungs acting as poor electrical conductors, were present in 15 patients (16.7%). Sinus tachycardia (heart rate >100 bpm) was documented in 19 patients (21.1%), and atrial fibrillation was detected in 8 patients (8.9%), indicating arrhythmic complications.

Table 2: Specific Electrocardiographic Abnormalities in COPD Patients (N=90)

ECG Parameter	Present	Absent
P pulmonale	36 (40.0%)	54 (60.0%)
Right Axis Deviation	29 (32.2%)	61 (67.8%)
Right Ventricular Hypertrophy	26 (28.9%)	64 (71.1%)
Right Bundle Branch Block	18 (20.0%)	72 (80.0%)
ST-T Wave Changes	22 (24.4%)	68 (75.6%)
Low Voltage QRS	15 (16.7%)	75 (83.3%)
Sinus Tachycardia	19 (21.1%)	71 (78.9%)
Atrial Fibrillation	8 (8.9%)	82 (91.1%)
Any ECG Abnormality	74 (82.2%)	16 (17.8%)

Table 2 illustrates specific electrocardiographic abnormalities detected in the study cohort, with 82.2% showing at least one abnormality. P pulmonale was most prevalent (40.0%), followed by right axis deviation (32.2%) and right ventricular hypertrophy (28.9%), reflecting chronic right heart involvement in COPD.

Echocardiographic evaluation revealed pulmonary hypertension in 59 patients (65.6%) with mean PASP 37.8 ± 12.4 mmHg. Among patients with pulmonary hypertension, mild PH (35-49 mmHg) was most common (59.3%), followed by moderate PH (50-69 mmHg) at 25.4%, and severe PH (\geq 70 mmHg) at 15.3%. Right ventricular dilatation was present in 35.6%, right atrial dilatation in 42.2%, and right ventricular dysfunction in 30.0%. Tricuspid regurgitation was present in 90.0%. Left ventricular systolic dysfunction was present in 8.9% while diastolic dysfunction affected 48.9%.

Table 3: Echocardiographic Findings in COPD Patients (N=90)

Echocardiographic Parameter	Category/Range	Frequency (%) / Mean ± SD
Mean PASP (mmHg)	All patients	37.8 ± 12.4
Pulmonary Hypertension	PASP <35 mmHg (Normal)	31 (34.4%)
(PASP ≥35 mmHg)	PASP ≥35 mmHg (Elevated)	59 (65.6%)
Severity of PH (n=59)	Mild (35-49 mmHg)	35 (59.3%)
	Moderate (50-69 mmHg)	15 (25.4%)
	Severe (≥70 mmHg)	9 (15.3%)
RV Dilatation	RVID >42 mm	32 (35.6%)
	RVID ≤42 mm	58 (64.4%)
RA Dilatation	RA area >18 cm ²	38 (42.2%)
	RA area ≤18 cm ²	52 (57.8%)
RV Dysfunction	TAPSE <17 mm	27 (30.0%)
	TAPSE ≥17 mm	63 (70.0%)
Tricuspid Regurgitation	Present	81 (90.0%)
	Absent	9 (10.0%)
LV Systolic Dysfunction	LVEF <50%	8 (8.9%)
	LVEF ≥50%	82 (91.1%)
LV Diastolic Dysfunction	Present	44 (48.9%)
	Absent	46 (51.1%)

Table 3 describes comprehensive echocardiographic findings revealing pulmonary hypertension in 65.6% of patients with mean PASP of 37.8±12.4 mmHg. Right ventricular structural and functional abnormalities were common (RV dilatation 35.6%, RV dysfunction 30.0%), while left ventricular diastolic dysfunction affected 48.9% of patients.

A clear progressive trend was observed in pulmonary hypertension prevalence across GOLD stages, increasing from 41.2% in Stage I to 100% in Stage IV. Among GOLD Stage I patients, 7 out of 17 (41.2%) had pulmonary hypertension, predominantly of mild severity. In GOLD Stage II, 20 out of 35 patients (57.1%) demonstrated elevated PASP, with increasing proportion of moderate PH. GOLD Stage III showed 23 out of 29 patients (79.3%) with pulmonary hypertension, including higher prevalence of moderate to severe PH. All 9 patients (100%) with GOLD Stage IV disease had pulmonary hypertension, with majority showing moderate to severe elevation of PASP.

Table 4: Association between Pulmonary Hypertension and GOLD Stage (N=90)

GOLD Stage	PH Present	PH Absent	Total
Stage I (Mild)	7 (41.2%)	10 (58.8%)	17 (100%)
Stage II (Moderate)	20 (57.1%)	15 (42.9%)	35 (100%)
Stage III (Severe)	23 (79.3%)	6 (20.7%)	29 (100%)
Stage IV (Very Severe)	9 (100%)	0 (0%)	9 (100%)
Total	59 (65.6%)	31 (34.4%)	90 (100%)
Chi-square test: $\chi^2 = 12.828$, $df = 3$, $p = 0.005*$			

Table 4 demonstrates progressive increase in pulmonary hypertension prevalence across GOLD stages from 41.2% in Stage I to 100% in Stage IV. Chi-square analysis revealed statistically significant association between PH and disease severity (χ^2 =12.828, p=0.005), confirming that advanced COPD stages predict higher likelihood of pulmonary vascular involvement.

25 - PH Present PH Absent 23 (79.3%)

20 (57.1%)

21 (29.9%)

23 (79.3%)

24 (22.9%)

25 - PH Present PH Absent (9 < 0.05)

(58.8%)

36 (20.7%)

57 (41.2%)

58 (20.7%)

58 (20.7%)

58 (20.7%)

59 (100.0%)

69 (20.7%)

58 (20.7%)

59 (100.0%)

69 (20.7%)

Figure 2: Prevalence of Pulmonary Hypertension across GOLD Stages

Figure 2 illustrates the progressive increase in pulmonary hypertension prevalence with advancing GOLD stages, from 41.2% in Stage I to 100% in Stage IV. Chi-square analysis confirmed statistically significant association between disease severity and PH development (p=0.005).

Mean PASP showed progressive and statistically significant increase with advancing GOLD stages: Stage I 30.1±9.2 mmHg, Stage II 33.9±9.9 mmHg, Stage III 42.0±13.2 mmHg, and Stage IV 48.5±13.3 mmHg. The strong linear correlation between GOLD stage and mean PASP (Pearson correlation coefficient r=0.587, p<0.001) suggests that spirometric severity is a reliable predictor of pulmonary hypertension development in COPD patients.

Table 5: Mean Pulmonary Artery Systolic Pressure across GOLD Stages (N=90)

GOLD Stage	N	Mean PASP (mmHg)	SD	95% CI
Stage I (Mild)	17	30.1	9.2	25.3 - 34.9
Stage II (Moderate)	35	33.9	9.9	30.5 - 37.3
Stage III (Severe)	29	42.0	13.2	37.0 - 47.0
Stage IV (Very Severe)	9	48.5	13.3	38.9 - 58.1
Total	90	37.8	12.4	35.2 - 40.4
One-way ANOVA: $F = 11.704$, $df = 3.86$, $p < 0.001**$				
Pearson correlation: $r = 0.587$, $p < 0.001**$				

Table 5 demonstrates stepwise progression of mean PASP values across GOLD stages with highly significant statistical difference (F=11.704, p<0.001). Strong positive correlation exists between GOLD stage and PASP (r=0.587, p<0.001), indicating that spirometric severity reliably predicts pulmonary hypertension development in COPD patients.

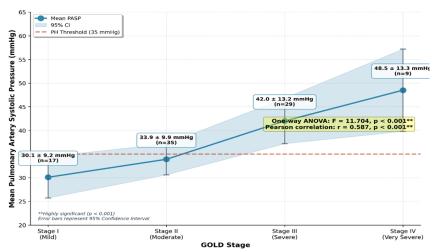


Figure 3: Progressive Increase in Mean Pulmonary Artery Systolic Pressure across GOLD Stages

Figure 3 demonstrates stepwise and statistically significant progression of mean PASP values across GOLD stages (ANOVA F=11.704, p<0.001). Strong positive correlation (r=0.587, p<0.001) between disease severity and PASP indicates that spirometric assessment can predict pulmonary vascular complications. The horizontal line at 35 mmHg indicates the threshold for pulmonary hypertension diagnosis.

DISCUSSION

This prospective study comprehensively evaluated electrocardiographic and echocardiographic abnormalities in 90 COPD patients, revealing high prevalence of cardiovascular complications correlating significantly with disease severity. The findings emphasize critical need for routine cardiac evaluation in COPD management.

Our study population demonstrated typical COPD demographics with male predominance (73.3%) and mean age 62.4 years, consistent with established epidemiological patterns. High smoking proportion (76.7%) aligns with smoking being principal COPD risk factor. Distribution across GOLD stages reflected tertiary care referral patterns with moderate to severe disease predominance.

ECG abnormalities present in 82.2% of our cohort were substantially higher than 23.6% reported by Kumar et al.[11] in community-based Indian COPD patients but comparable to 69% by Jatav et al.[12] in hospitalized patients. This variation likely reflects disease severity differences between community and hospital populations, with tertiary care naturally concentrating severe cases.

P pulmonale prevalence (40.0%) was considerably higher than 15.5-32.7% range in earlier studies,[15] suggesting significant right atrial enlargement secondary to chronic pressure overload. Right axis deviation (32.2%) was consistent with previous reports of 28-38% prevalence in COPD populations.[16] Right ventricular hypertrophy (28.9%) aligned with reported 23-40% range, though ECG has limited sensitivity for detecting RVH in COPD due to hyperinflation-related electrical insulation effects.[17] Right bundle branch block (20.0%) fell within 15-25% literature range, representing advanced conduction involvement. Low voltage QRS (16.7%) was attributed to increased anteroposterior chest diameter and hyperinflated lungs acting as poor electrical conductors.

Pulmonary hypertension prevalence (65.6%) fell within wide reported range of 20-90% in COPD, influenced by disease severity, diagnostic criteria, and assessment methods. Zhang et al.'s[8] meta-analysis reported overall PH prevalence 39.2% across various COPD populations, while severe COPD studies demonstrated prevalence approaching 90%. Our cohort with predominant GOLD Stage II-III showed intermediate prevalence consistent with disease distribution.

Progressive PH increase across GOLD stages (41.2% Stage I to 100% Stage IV) demonstrated strong correlation between airflow obstruction and pulmonary vascular complications, consistently shown in literature.[15] Mean PASP stepwise progression from 30.1 mmHg (Stage I) to 48.5 mmHg (Stage IV) aligned with Fayngershet al.[16] findings of 28±11 mmHg in mild increasing to 42±16 mmHg in severe COPD. Strong linear correlation (r=0.587, p<0.001) between GOLD stage and PASP emphasizes spirometric assessment helps predict cardiovascular complications.

Right ventricular dysfunction (30.0%) fell within reported 20-35% range in COPD populations. TAPSE provided reliable RV function quantification, with reduced values associated with increased mortality.[18] RV dilatation (35.6%) and RA enlargement (42.2%) indicated significant right heart structural remodeling from chronic pressure overload.

Left ventricular diastolic dysfunction (48.9%) warrants attention with important prognostic implications. Multiple mechanisms contribute including ventricular interdependence, chronic hypoxemia effects, systemic inflammation, and

age-related changes. Boussugeset al.[17] reported similar prevalence, emphasizing comprehensive ventricular assessment importance.

The high cardiovascular abnormality prevalence has several clinical implications. First, systematic cardiovascular screening in COPD patients is needed, particularly moderate to severe disease. ECG, widely available and cost-effective, should be routine investigation. Specific findings like P pulmonale, right axis deviation, and RVH signs should prompt echocardiographic evaluation.

Second, echocardiography should be considered for GOLD Stage II and above given substantial pulmonary hypertension and RV dysfunction prevalence. Early detection allows targeted interventions including optimal COPD management, hypoxemia correction through supplemental oxygen, pulmonary rehabilitation, and pulmonary vasodilator therapy consideration in selected cases.

Third, cardiovascular abnormality presence should influence prognostic assessment and treatment decisions. Patients with documented PH and RV dysfunction require closer monitoring, aggressive exacerbation management, and potentially earlier advanced therapy consideration including transplantation evaluation.

Several study limitations merit consideration. Single-center design and modest sample size may limit generalizability. Cross-sectional design precludes longitudinal assessment. Right heart catheterization was not performed; however, echocardiographic PASP estimation is well-validated as screening tool. Exercise-induced changes were not assessed. Future longitudinal studies following COPD patients would provide valuable insights into cardiovascular complication temporal evolution and outcome impact. Studies evaluating routine screening cost-effectiveness and exploring novel biomarkers for early detection would strengthen evidence for universal implementation.

CONCLUSION

This prospective study demonstrates that electrocardiographic and echocardiographic abnormalities are highly prevalent among COPD patients in tertiary care settings, with P pulmonale, right axis deviation, and right ventricular hypertrophy being predominant ECG findings, while pulmonary hypertension showed strong positive correlation with disease severity as per GOLD classification. These findings emphasize that COPD should be viewed as a systemic disease with significant cardiovascular ramifications, necessitating routine incorporation of ECG and echocardiographic evaluation in management protocols to facilitate early complication detection and enable timely therapeutic interventions. Given the strong correlation between disease severity and cardiovascular abnormalities, echocardiographic screening should be prioritized for moderate to severe COPD patients, as integration of cardiovascular assessment into comprehensive COPD care has the potential to improve risk stratification, guide management decisions, and ultimately enhance patient outcomes.

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