ORIGINAL RESEARCH PAPER

General Medicine

ECG PATTERNS PREDICTING SEVERITY OF COPD RELATED PULMONARY ARTERY HYPERTENSION

KEY WORDS: COPD, Pulmonary Hypertension, ECG, Echocardiography, Right Ventricular Dysfunction

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is frequently complicated by pulmonary hypertension and right heart dysfunction, which significantly worsen symptoms, functional status, and survival. Electrocardiography (ECG) and echocardiography offer accessible tools for detecting these cardiovascular abnormalities, yet their correlation with COPD severity remains underused in routine practice. The present study aimed to evaluate electrocardiographic and echocardiographic abnormalities in COPD patients and assess their association with disease severity and pulmonary artery pressure. **Methods:** This prospective study included 100 diagnosed COPD patients evaluated through clinical examination, post-bronchodilator spirometry, 12-lead ECG, and transthoracic echocardiography. COPD severity was classified according to GOLD staging. Pulmonary hypertension was defined as pulmonary artery systolic pressure (PASP) ≥35 mmHg. Statistical analysis was performed using SPSS version 26.0. **Results:** ECG abnormalities were detected in 84% of patients, with P pulmonale (39%), right axis deviation (34%), and right ventricular hypertrophy (30%) being the most common findings. Pulmonary hypertension was present in 68% of the cohort, with mean PASP showing a progressive rise from Stage I (31.2 ± 8.7 mmHg) to Stage IV (49.8 ± 13.1 mmHg). RV dysfunction and RV dilatation were found in 31% and 36% of patients, respectively. A strong positive correlation existed between GOLD stage and PASP ($r = 0.604$, $p < 0.001$), and the association between COPD severity and PH prevalence was statistically significant ($p = 0.002$). **Conclusion:** Cardiovascular abnormalities are highly prevalent in COPD and increase with disease severity. ECG and echocardiography provide valuable insight into pulmonary hypertension and right heart involvement, supporting their routine use in COPD evaluation for early detection and improved management.

INTRODUCTION

Chronic Obstructive Pulmonary Disease is a major public health concern and ranks as the third leading cause of death worldwide, with an estimated 3.23 million deaths annually [1-3]. More than 384 million people are affected globally, and prevalence varies by region according to the Global Burden of Disease Study [4]. In India, COPD remains a significant burden, with prevalence in adults ranging between 6 and 10 percent and nearly 30 million individuals living with the disease [5].

COPD is characterized by persistent respiratory symptoms and airflow limitation that arise from structural changes in the bronchi, small airways, and lung parenchyma, most commonly due to long-term exposure to noxious particles or gases [6]. It is increasingly recognized as a systemic disorder that extends beyond the lungs, contributing to multiple complications, particularly cardiovascular disease [7]. Cardiovascular involvement accounts for a large share of morbidity and is the leading cause of death among patients with early to moderate COPD [8].

Pulmonary hypertension is one of the most important cardiovascular complications of COPD, reported in 20 to 90 percent of patients across various disease stages. It develops through a combination of chronic hypoxemia, hypoxic pulmonary vasoconstriction, vascular remodeling, and destruction of the pulmonary vascular bed [9,10]. As pulmonary hypertension progresses, it places pressure overload on the right ventricle, resulting in right ventricular hypertrophy and, eventually, cor pulmonale. These changes substantially worsen functional capacity and survival.

Electrocardiography remains a simple and widely available tool for detecting cardiac involvement in COPD. Common abnormalities include P pulmonale, right axis deviation, right ventricular hypertrophy, right bundle branch block, and low-voltage QRS complexes [11,12]. These findings reflect the hemodynamic stress imposed by chronic pulmonary hypertension. Reported prevalence of ECG abnormalities ranges from 23 to 69 percent, depending on disease severity and population characteristics [13,14].

Transthoracic echocardiography is the preferred non-invasive method for estimating pulmonary artery pressure and assessing right ventricular structure and function [15,16]. However, its routine use is still limited in many clinical settings.

There is limited evidence from Indian tertiary care centers on the correlation between ECG patterns and the severity of COPD-related pulmonary artery hypertension. This study evaluates ECG changes in COPD patients and examines how these patterns relate to the severity of pulmonary hypertension.

MATERIAL & METHODS

This study followed a prospective observational design. It was carried out in the Department of Pulmonary Medicine at Index Medical College, Hospital and Research Centre, Indore. Data collection was completed over a defined study period after institutional ethics approval. A total of 100 adults with a confirmed diagnosis of chronic obstructive pulmonary disease were enrolled. Patients were included when they presented for evaluation of exertional breathlessness, chronic cough, or suspected pulmonary hypertension. COPD diagnosis was based on clinical assessment supported by spirometry according to GOLD criteria.

Inclusion Criteria

- Age 40 years and above
- Clinically and spirometrically diagnosed COPD
- Patients willing to undergo ECG and echocardiographic evaluation
- Stable patients without an acute exacerbation in the preceding four weeks

Exclusion Criteria

- Known congenital or acquired structural heart disease
- Primary pulmonary hypertension
- Cor pulmonale due to non-COPD causes
- Significant arrhythmias interfering with ECG interpretation
- Patients with incomplete clinical or investigation records

Methodology

Each participant underwent a detailed history and physical examination. Clinical parameters included duration of COPD, smoking history (pack-years), oxygen saturation, and presence of cor pulmonale features. Breathlessness severity was graded using the mMRC scale. Spirometry was performed to record FEV_1, FVC, and FEV_1/FVC ratios.

Pulmonary Function Testing

Pulmonary function testing was performed using a computerized spirometer following ATS and ERS guidelines. Post-bronchodilator spirometry was used to confirm airflow limitation. COPD severity was graded according to GOLD staging. Mild disease was defined as an FEV of 80 percent or more of predicted. Moderate airflow limitation corresponded to an FEV between 50 and 79 percent. Severe disease included patients with an FEV between 30 and 49 percent, while very severe COPD referred to an FEV of less than 30 percent of predicted. These categories were used to compare electrocardiographic and echocardiographic findings across disease stages.

Electrocardiographic Evaluation

A standard 12-lead ECG was recorded for each patient in the resting state, using a calibrated machine with a paper speed of 25 mm/s and voltage settings of 10 mm/mV. ECGs were assessed for P pulmonale, right axis deviation, right ventricular hypertrophy, right bundle branch block, and low-voltage QRS complexes. Two experienced physicians interpreted the tracings independently, and discrepancies were resolved by mutual agreement.

Echocardiographic Assessment

Transthoracic echocardiography was performed to evaluate pulmonary artery systolic pressure and assess right heart structure and function. Severity of pulmonary hypertension was categorized as mild (30–49 mmHg), moderate (50–69 mmHg), or severe (70 mmHg or above). Measurements included right ventricular dimension, right atrial size, tricuspid regurgitant jet velocity, and estimated pulmonary artery systolic pressure.

Outcome Measures

The primary objective was to determine the ECG patterns associated with pulmonary hypertension in COPD. A secondary objective was to evaluate how these ECG findings varied across different stages of COPD severity.

Statistical Analysis

All collected data were entered into a structured database and analysed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performedusing chi-square tests for categorical variables and independent t-tests or one-way ANOVA for continuous variables, depending on distribution. Correlation between ECG patterns and pulmonary hypertension severity was assessed using appropriate statistical tests. A p-value below 0.05 was considered statistically significant.

RESULTS

The study enrolled 100 patients with confirmed COPD, with a mean age of 63.1 ± 9.6 years (range 41–84 years). Males made up 74% of the cohort. Most participants were between 60 and 69 years of age (36%), followed by those in the 50–59 year group (30%). A history of smoking was present in 78% of the patients, and the mean smoking exposure was 25.8 ± 11.9 pack-years. Spirometric evaluation showed that the majority of patients (73%) were classified in GOLD Stages II and III, reflecting moderate to severe disease, which is typical of cases presenting to a tertiary care hospital. [Figure 1] GOLD Stage II (40%) was the most common category, followed by Stage III (33%), Stage I (17%), and Stage IV (10%). The mean FEV_1 was 51.6 ± 17.9 percent of the predicted value, and the average FEV_1/FVC ratio was 0.57 ± 0.11, indicating persistent airflow limitation in the study population. [Table 1]

Table 1: Demographic and Clinical Characteristics of Study Population (N = 100)

Variable	Category	Frequency (%)
Age (years)	40–49	10 (10%)
	50–59	30 (30%)
	60–69	36 (36%)
	≥70	24 (24%)
Gender	Male	74 (74%)
	Female	26 (26%)
Smoking Status	Current/Former Smokers	78 (78%)
	Non-smokers	22 (22%)
GOLD Stage	Stage I	17 (17%)
	Stage II	40 (40%)
	Stage III	33 (33%)
	Stage IV	10 (10%)

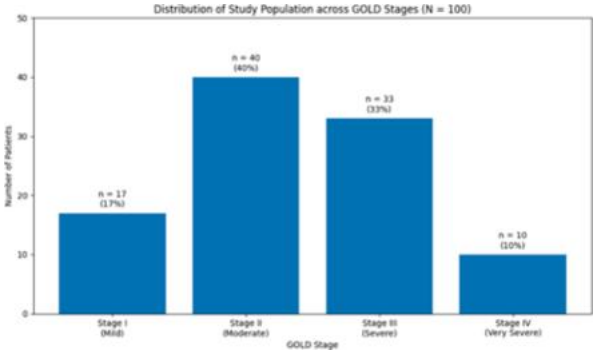

Figure 1: Distribution of Study Population across GOLD Stages (N=100)

ECG abnormalities were observed in 84% of patients, with right heart–related changes being the most common. P pulmonale was the most frequent finding (39%), followed by right axis deviation (34%) and right ventricular hypertrophy (30%). Other abnormalities included right bundle branch block (18%), ST–T changes (25%), low-voltage QRS complexes (17%), sinus tachycardia (22%), and atrial fibrillation (6%). [Table 2]

Table 2: Specific Electrocardiographic Abnormalities in COPD Patients (N = 100)

ECG Parameter	Present	Absent
P pulmonale	39 (39%)	61 (61%)
Right Axis Deviation	34 (34%)	66 (66%)
Right Ventricular Hypertrophy	30 (30%)	70 (70%)
Right Bundle Branch Block	18 (18%)	82 (82%)
ST–T Wave Changes	25 (25%)	75 (75%)
Low Voltage QRS	17 (17%)	83 (83%)
Sinus Tachycardia	22 (22%)	78 (78%)
Atrial Fibrillation	6 (6%)	94 (94%)
Any ECG Abnormality	84 (84%)	16 (16%)

Echocardiography showed pulmonary hypertension in 68% of patients, with a mean PASP of 38.6 ± 11.8 mmHg. Mild PH was the most common form (57.4%), followed by moderate (27.9%) and severe PH (14.7%). Right ventricular dilatation and dysfunction were noted in 36% and 31% of patients, respectively, while right atrial enlargement occurred in 43%. Tricuspid regurgitation was present in 89%. Left ventricular

systolic dysfunction was identified in 9% and diastolic dysfunction in 47% of the cohort. [Table 3]

Table 3: Echocardiographic Findings in COPD Patients (N = 100)

Parameter	Frequency (%) or Mean ± SD
Mean PASP (mmHg)	38.6 ± 11.8
Pulmonary Hypertension (≥35 mmHg)	68 (68%)
Severity of PH (n=68)	
Mild (35–49 mmHg)	39 (57.4%)
Moderate (50–69 mmHg)	19 (27.9%)
Severe (≥70 mmHg)	10 (14.7%)
RV Dilatation	36 (36%)
RA Dilatation	43 (43%)
RV Dysfunction (TAPSE <17 mm)	31 (31%)
Tricuspid Regurgitation	89 (89%)
LV Systolic Dysfunction	9 (9%)
LV Diastolic Dysfunction	47 (47%)

The prevalence of pulmonary hypertension increased steadily with advancing COPD severity, rising from 41.2% in Stage I to 100% in Stage IV. Moderate and severe stages showed the highest burden, with 60.0% of Stage II patients and 81.8% of Stage III patients affected. Chi-square analysis demonstrated a significant association between GOLD stage and pulmonary hypertension (χ^2 = 14.92, p = 0.002), indicating that the likelihood of developing PH increases as airflow limitation worsens. [Table 4, Figure 2]

Table 4: Pulmonary Hypertension Across GOLD Stages (N = 100)

GOLD Stage	PH Present	PH Absent	Total
Stage I	7 (41.2%)	10 (58.8%)	17
Stage II	24 (60.0%)	16 (40.0%)	40
Stage III	27 (81.8%)	6 (18.2%)	33
Stage IV	10 (100%)	0 (0%)	10
Total	68 (68%)	32 (32%)	100
Chi-square: χ^2 = 14.92, df = 3, p = 0.002			

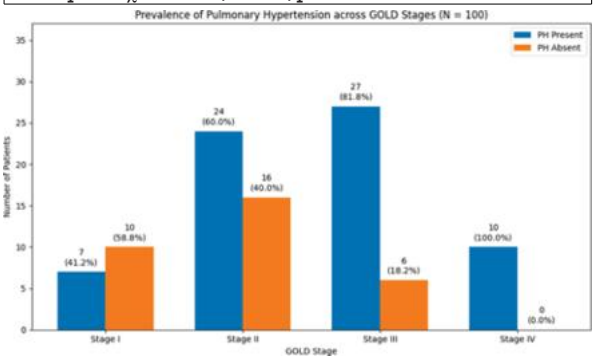

Figure 2: Prevalence of Pulmonary Hypertension across GOLD Stages

Mean PASP increased steadily with advancing COPD severity, rising from 31.2 mmHg in Stage I to 49.8 mmHg in Stage IV. This upward trend was statistically significant on ANOVA (F = 12.83, p < 0.001). A strong positive correlation was also observed between GOLD stage and PASP (r = 0.604, p < 0.001), indicating that higher spirometric severity is closely linked with elevated pulmonary artery pressure. [Table 5, Figure 3]

Table 5: Mean PASP Across GOLD Stages (N = 100)

GOLD Stage	N	Mean PASP (mmHg)	SD	95% CI
Stage I	17	31.2	8.7	27.0–35.4
Stage II	40	34.7	9.4	31.7–37.7
Stage III	33	43.5	12.6	38.8–48.2
Stage IV	10	49.8	13.1	41.0–58.6
Total	100	38.6	11.8	36.3–41.0
ANOVA: F = 12.83, p < 0.001				
Correlation: r = 0.604, p < 0.001				

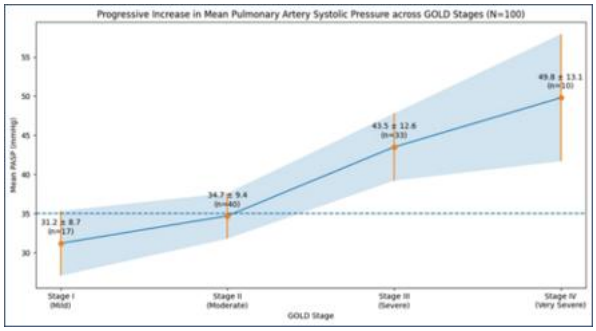

Figure 3. Mean Pulmonary Artery Systolic Pressure Progression Across GOLD Severity Levels

DISCUSSION

This prospective study evaluated electrocardiographic and echocardiographic abnormalities in 100 patients with diagnosed COPD and demonstrated a high burden of cardiovascular involvement. The findings clearly show that cardiac abnormalities increase as airflow limitation becomes more severe, reinforcing the need for systematic cardiac surveillance as part of COPD management.

The demographic pattern in our cohort, with a mean age of 63.1 years and marked male predominance (74%), aligns with established epidemiological trends in India and globally. [1] Smoking exposure was substantial, with 78% of patients being current or former smokers and a mean pack-year index of 25.8. These characteristics are consistent with smoking being the principal risk factor for COPD development and progression. The distribution of patients across GOLD stages, with 73% classified in Stages II and III, reflects typical tertiary care referral patterns, where moderate-to-severe cases are more commonly evaluated.

ECG abnormalities were detected in 84% of patients, a proportion higher than the 23.6% reported by Kumar et al. [13] in community-based COPD populations, but comparable to the higher rates documented in hospital-based cohorts such as those reported by Jatav et al. [14] and by similar tertiary care studies where abnormal ECG patterns were noted in more than 80% of patients. These differences across studies likely reflect variations in disease severity, as tertiary centers tend to evaluate patients with more advanced physiological impairment. In our study, P pulmonale was the most frequent abnormality (39%), which is higher than the 15.5–32.7% range described in earlier literature [17], indicating more pronounced right atrial overload. The frequency of right axis deviation (34%) and right ventricular hypertrophy (30%) aligned well with previously documented ranges of 28–38% and 23–40%, respectively [18]. Conduction-related abnormalities, including right bundle branch block (18%) and low-voltage QRS complexes (17%), were also in keeping with patterns attributed to hyperinflation and altered thoracic configuration in COPD [19]. Similar distributions of these abnormalities have been reported in other clinical studies evaluating hospitalized COPD patients.Pulmonary hypertension was present in 68% of patients, which lies within the broad 20–90% prevalence range reported across COPD populations depending on disease severity and diagnostic method. A meta-analysis by Zhang et al. [10] reported an overall PH prevalence of 39.2%, with rates rising sharply in severe disease. Studies evaluating moderate to severe COPD, including those conducted in comparable tertiary settings, have documented PH rates in the 60–70% range, similar to our findings. The increase in PH prevalence from 41.2% in Stage I to 100% in Stage IV in our data reflects the well-established relationship between airflow limitation and pulmonary vascular involvement, a trend frequently attributed to worsening hypoxemia, vascular

remodeling, and the loss of pulmonary vascular bed in advanced disease [17].

Mean pulmonary artery systolic pressure also increased progressively with COPD severity, rising from 31.2 mmHg in Stage I to 49.8 mmHg in Stage IV. This stepwise escalation is consistent with trends described in previous echocardiographic studies, including those reported by Fayngersh et al. [18], where PASP increased markedly with advancing disease stage. The strong positive correlation between GOLD stage and PASP observed in our study ($r = 0.604$, $p < 0.001$) further supports the role of spirometry not only in grading airflow limitation but also in anticipating pulmonary vascular complications.

Right heart structural and functional abnormalities were also frequent. Right ventricular dilatation was noted in 36% of patients, and right atrial enlargement in 43%, reflecting chronic pressure overload. Right ventricular systolic dysfunction, assessed using TAPSE, was present in 31%, aligning with the 20–35% range reported in earlier studies [19]. TAPSE reduction is known to carry prognostic weight, particularly in patients with coexisting pulmonary hypertension. Left ventricular diastolic dysfunction was documented in 47% of patients, similar to findings by Boussuges et al. [20], and may result from ventricular interdependence, chronic hypoxemia, systemic inflammation, and age-related myocardial changes.

The clinical implications of these findings are substantial. First, given the high burden of ECG abnormalities, a baseline ECG should be part of routine COPD assessment, particularly for patients in GOLD Stage II and above. Findings such as P pulmonale, right axis deviation, and features of RV hypertrophy should prompt further evaluation. Second, echocardiography should be considered early in the course of management because pulmonary hypertension and right ventricular dysfunction were common even in moderate disease. Early identification may allow interventions such as optimization of bronchodilator therapy, long-term oxygen therapy for hypoxemia, pulmonary rehabilitation, and consideration of pulmonary hypertension–specific management in selected cases. Third, the presence of cardiovascular abnormalities should influence risk stratification, exacerbation management, and follow-up intensity. Patients with PH or RV dysfunction may benefit from closer monitoring and discussions regarding advanced therapies in appropriate clinical contexts.

This study has several limitations. Being a single-center study, the findings may not capture regional variability. The sample size, though larger than several earlier studies, remains modest. The cross-sectional design does not permit assessment of progression over time. Although echocardiography is a widely accepted screening tool, right heart catheterization—considered the gold standard—was not performed. Exercise-induced hemodynamic changes were also not evaluated.

Future research should focus on longitudinal follow-up to determine how ECG and echocardiographic abnormalities evolve and how they influence clinical outcomes. Larger multicentric studies would improve generalizability and could evaluate cost-effectiveness of routine cardiac screening in COPD. Exploration of biomarkers for early detection of pulmonary vascular involvement would also contribute valuable insights.

CONCLUSION

This study highlights the substantial burden of electrocardiographic and echocardiographic abnormalities in COPD and demonstrates a clear progression of pulmonary hypertension and right heart involvement with advancing disease severity. ECG proved valuable for early detection of cardiac changes, while echocardiography identified significant structural and functional impairment, even in moderate COPD. The strong association between GOLD stage and pulmonary artery pressure underscores the need for routine cardiopulmonary evaluation. Early identification of cardiovascular complications offers opportunities for timely intervention, better risk stratification, and improved clinical outcomes. Integrating cardiac assessment into standard COPD care is essential for comprehensive patient management.

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