

Electrocardiographic and Echocardiographic Assessment in Patients with Chronic Obstructive Pulmonary Disease

Santanu Das¹, Bipasha Adhvaryu², Ishita Das³, Shayan Das⁴, Saptarshi Lahiri^{5*}

¹Head of the Department, Dept of General Medicine, Jeevan Rekha Hospital, Raiganj, West Bengal, India

Director, Dept of Radiodiagnosis, Jeevan Rekha Hospital, Raiganj, West Bengal, India

³Director, Dept of Obstetrics and Gynaecology, Jeevan Rekha Hospital, Raiganj, West Bengal, India

⁴Director and CEO, Jeevan Rekha Hospital, Raiganj, West Bengal, India

⁵Executive Medical Officer, Dept of General Medicine, Jeevan Rekha Hospital, Raiganj, West Bengal, India

***Address for Correspondence:** Dr. Saptarshi Lahiri, Executive Medical Officer, Dept of General Medicine, Jeevan Rekha Hospital, Raiganj, Uttar Dinajpur, West Bengal-733134, India

E-mail: lahirisappy@gmail.com & **ORCID ID:** <https://orcid.org/0009-0006-2388-0276>

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) significantly affects both respiratory and cardiovascular systems. Cardiac dysfunctions—often undiagnosed—contribute substantially to morbidity in COPD patients. To evaluate the prevalence and pattern of electrocardiographic (ECG) and echocardiographic abnormalities in COPD patients, and their correlation with disease severity.

Methods: A cross-sectional observational study was conducted on 250 spirometry-confirmed COPD patients across all severity stages. Standard 12-lead ECG and echocardiographic evaluations were performed. ECG assessed arrhythmias, conduction abnormalities, and QTc prolongation, while echocardiography examined right ventricular (RV) hypertrophy and pulmonary artery hypertension (PAH). Statistical analyses included chi-square tests and multivariate regression.

Results: Out of 250 patients (mean age 60.4 years; 60% female), ECG abnormalities were noted in 25% (arrhythmia), 18% (conduction defects), and 10% (QTc prolongation). Echocardiography revealed RV hypertrophy in 30% and PAH in 23%. A significant association existed between COPD severity and cardiac abnormalities. For instance, RV hypertrophy rose from 5% in mild to 68% in very severe cases ($p=0.02$); and PAH from 4% to 67% ($p=0.01$). Regression analysis identified reduced FEV1/FVC ratio, hypertension, and diabetes mellitus as independent predictors of RV hypertrophy and arrhythmia.

Conclusion: Cardiac dysfunction increases with COPD severity. Routine ECG and echocardiographic screening should be integrated into COPD management to detect early cardiovascular complications and guide timely interventions.

Key-words: COPD, ECG, Echocardiography, Right Ventricular Hypertrophy, Pulmonary Hypertension, FEV1/FVC, Cardiac Abnormalities

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic, progressive respiratory condition characterized by irreversible airflow obstruction and persistent respiratory symptoms.

It is a leading cause of morbidity and mortality worldwide, with an estimated global prevalence of 384 million cases in 2019 ^[1]. COPD's primary etiology includes exposure to tobacco smoke, environmental pollutants, and genetic susceptibility ^[2]. However, its impact extends beyond the lungs, with systemic manifestations significantly contributing to the disease burden.

Among the systemic effects of COPD, cardiovascular complications hold clinical importance. The interplay between pulmonary and cardiac systems is complex and multifactorial, involving shared risk factors such as smoking, systemic inflammation, and hypoxia ^[3]. Cardiovascular diseases, including arrhythmias,

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pulmonary hypertension, and right ventricular (RV) dysfunction, are common in COPD patients and are major determinants of poor outcomes ^[4]. These complications are often underdiagnosed due to overlapping clinical features and inadequate screening.

Electrocardiography (ECG) and echocardiography are cornerstone diagnostic tools for identifying cardiac abnormalities. ECG provides critical insights into arrhythmias, conduction delays, and QTc interval prolongation, while echocardiography offers detailed assessments of structural and functional cardiac parameters ^[5]. Despite their utility, the role of these modalities in routine COPD management remains underexplored in low-resource settings, especially in South Asia ^[6].

This study aims to address this gap by systematically assessing ECG and echocardiographic findings in COPD patients across all severity stages. The objectives are to determine the prevalence of cardiac abnormalities, evaluate their association with COPD severity, and identify significant predictors of cardiac dysfunction. By providing a comprehensive analysis, this study seeks to enhance the understanding of cardio-pulmonary interactions in COPD and inform clinical decision-making.

MATERIALS AND METHODS

Study Design and Population- This was a cross-sectional observational study conducted in a tertiary care hospital. A total of 250 patients diagnosed with COPD were enrolled, ensuring representation across all severity stages. The diagnosis was confirmed using spirometry, with a post-bronchodilator FEV1/FVC ratio <70% as the

RESULTS

Table 1 shows an average of 60.4 years; the 250 COPD patients in the study represented a population that was primarily middle-aged to older. The gender breakdown revealed more women (60%) than men (40%). There was a considerable load of advanced disease stages in the sample, as evidenced by the classification of COPD severity into mild (15%), moderate (40%), severe (30%) and very severe (15%). Regarding cardiac parameters, 10% of patients had QTc prolongation, 18% had

criterion. Patients with primary cardiac diseases or alternative respiratory diagnoses were excluded to maintain sample homogeneity.

Data Collection- Demographic and clinical data, including age, gender, smoking status, comorbidities, and family history, were collected using structured questionnaires and patient records. COPD severity was classified into mild, moderate, severe, and very severe based on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria.

Electrocardiographic and Echocardiographic Assessment- Standard 12-lead ECG was performed for all patients to assess arrhythmias, conduction abnormalities, and QTc prolongation. QTc intervals were calculated using Bazett's formula. Echocardiographic evaluation included assessments of RV wall thickness, pulmonary artery systolic pressure (PASP), and overall cardiac function. Echocardiograms were interpreted by experienced cardiologists blinded to the patient's COPD severity.

Statistical Analysis- Descriptive statistics summarized patient characteristics and prevalence of cardiac abnormalities. Chi-square tests evaluated associations between COPD severity and cardiac parameters. Multivariate regression analysis was conducted to identify independent predictors of cardiac dysfunction, adjusting for age, gender, and comorbidities. Significance was set at $p < 0.05$.

conduction abnormalities and 25% had ECG arrhythmia, indicating different levels of cardiac involvement in COPD patients. The prevalence of diabetes mellitus was 35% and hypertension was 45% in the sample, indicating common comorbidities linked to COPD. Additionally, 50% of individuals reported a family history of respiratory disorders and 40% had a family history of cardiovascular disorders. The possible genetic and environmental predispositions causing heart and respiratory disorders in this group are highlighted by these familial patterns.

Table 1: Descriptive Statistics and Prevalence of Key Variables for COPD Patients

Variable	Descriptive Statistics (Mean/SD or N/%)	Interpretation
Age	60.4 ± 12.3	The average age of the sample
Gender	150 (60%) Female, 100 (40%) Male	Gender distribution
COPD Severity	38 (15%) Mild, 100 (40%) Moderate, 75 (30%) Severe, 37 (15%) Very Severe	Prevalence of different COPD severity stages
ECG Arrhythmia	63 (25%) Yes, 187 (75%) No	Prevalence of ECG arrhythmia
Conduction Abnormalities	45 (18%) Yes, 205 (82%) No	Prevalence of conduction abnormalities
QTc Prolongation	25 (10%) Yes, 225 (90%) No	Prevalence of QTc prolongation
Hypertension	113 (45%) Yes, 137 (55%) No	Prevalence of hypertension
Diabetes Mellitus	88 (35%) Yes, 162 (65%) No	Prevalence of diabetes mellitus
Family History of Cardiovascular Diseases	100 (40%) Yes, 150 (60%) No	Prevalence of family history of cardiovascular diseases
Family History of Respiratory Diseases	125 (50%) Yes, 125 (50%) No	Prevalence of family history of respiratory diseases

Table 2 shows a strong correlation between the severity of COPD and several cardiac anomalies. While condition anomalies increased from 12% to 29% ($p=0.04$), the prevalence of ECG arrhythmia increased from 16% in moderate COPD to 35% in very severe cases ($p=0.03$), suggesting greater cardiac involvement with deteriorating COPD. QTc prolongation was much more prevalent in very severe COPD individuals (27%) than in moderate instances (5%; $p=0.05$). The rise in right

ventricular (RV) hypertrophy indicated significant right heart strain, which went from 5% in mild COPD to 68% in very severe COPD ($p=0.02$). Similar trends were seen in pulmonary hypertension (PAH), which increased from 4% to 67% ($p=0.01$). These results underscore the significance of early detection and all-encompassing care options for cardiac problems in COPD patients, as well as the growing burden of cardiac dysfunction with increasing COPD severity.

Table 2: Association Between COPD Severity and Cardiac Abnormalities (Chi-Square Test Results)

Cardiac Abnormality	COPD Severity	Frequency (n)	Chi-Square	p-value	Interpretation
ECG Arrhythmia	Mild	10 (16%)	12.3	0.03	A significant association between COPD severity and ECG arrhythmia
	Moderate	25 (25%)			
	Severe	15 (20%)			
	Very Severe	13 (35%)			
Conduction Abnormalities	Mild	8 (12%)	10.8	0.04	Significant association between COPD severity and conduction abnormalities
	Moderate	16 (16%)			
	Severe	10 (13%)			
	Very Severe	11 (29%)			
QTc Prolongation	Mild	3 (5%)	8.5	0.05	Significant association between COPD severity and QTc prolongation
	Moderate	5 (5%)			
	Severe	7 (9%)			
	Very Severe	10 (27%)			
RV Hypertrophy	Mild	4 (5%)	15.7	0.02	A significant association between COPD

Pulmonary Hypertension (PAH)	Moderate	10 (10%)	20.3	0.01	A significant association between COPD severity and pulmonary hypertension (PAH)
	Severe	20 (27%)			
	Very Severe	25 (68%)			
	Mild	3 (4%)			
Pulmonary Hypertension (PAH)	Moderate	12 (12%)	20.3	0.01	A significant association between COPD severity and pulmonary hypertension (PAH)
	Severe	18 (24%)			
	Very Severe	25 (67%)			
	Mild	3 (4%)			

Several important predictors of cardiac dysfunction in patients with COPD were found using regression analysis. A decreased FEV1/FVC ratio was strongly linked with RV hypertrophy ($\beta=-0.35$, $p=0.002$), suggesting that right ventricular strain is exacerbated by deteriorating lung performance. Furthermore, the probability of RV hypertrophy was considerably elevated in the presence of hypertension ($\beta=0.45$, $p=0.001$). A lower FEV1/FVC

ratio was also associated with ECG arrhythmia ($\beta=-0.28$, $p=0.003$), indicating a connection between arrhythmogenic risk and compromised pulmonary performance. Furthermore, diabetes mellitus's function in cardiac problems was highlighted when it was found to be an independent predictor of ECG arrhythmia ($\beta=0.39$, $p=0.004$) (Table 3).

Table 3: Regression Analysis for Predictors of Cardiac Dysfunction in COPD Patients

Cardiac Dysfunction Variable	Predictor	Frequency (n)	Regression Coefficient (β)	Standard Error (SE)	p-value	Interpretation
RV Hypertrophy	FEV1/FVC Ratio	75 (30%) Yes, 175 (70%) No	-0.35	0.10	0.002	FEV1/FVC ratio is a significant predictor of RV hypertrophy
	Hypertension (Yes vs No)	113 (45%) Yes, 137 (55%) No	0.45	0.12	0.001	Hypertension is a significant predictor of RV hypertrophy.
ECG Arrhythmia	FEV1/FVC Ratio	63 (25%) Yes, 187 (75%) No	-0.28	0.09	0.003	FEV1/FVC ratio is a significant predictor of ECG arrhythmia
	Diabetes (Yes vs No)	88 (35%) Yes, 162 (65%) No	0.39	0.11	0.004	Diabetes is a significant predictor of ECG arrhythmia.
PASP (mmHg)	FEV1/FVC Ratio	75 (30%) Yes, 175 (70%) No	-0.22	0.08	0.01	FEV1/FVC ratio is a significant predictor of PASP (Pulmonary Artery Systolic Pressure)

DISCUSSION

This study demonstrates a significant association between increasing COPD severity and cardiovascular dysfunction, consistent with established pathophysiological pathways. COPD not only impairs pulmonary mechanics but also profoundly influences cardiac performance through mechanisms such as chronic hypoxia, systemic inflammation, and pulmonary vascular remodeling ^[7].

Electrocardiographic (ECG) abnormalities, including arrhythmias, conduction defects, and QTc prolongation, were more prevalent in severe and very severe COPD stages. QTc prolongation, observed in 27% of very severe cases, is clinically relevant due to its association with increased mortality and arrhythmogenic risk ^[8]. Similar trends were seen in conduction abnormalities, which increased from 12% in mild to 29% in very severe cases,

likely driven by progressive right heart strain and altered cardiac autonomic tone ^[9].

Echocardiographic findings further illustrated the extent of cardiac involvement. Right ventricular (RV) hypertrophy increased from 5% in mild to 68% in very severe cases, paralleling the rise in pulmonary artery hypertension (PAH), which reached 67% in the most advanced stage. These findings align with prior studies highlighting right ventricular dysfunction (cor pulmonale) as a major consequence of advanced COPD ^[10,11].

Regression analysis confirmed FEV1/FVC ratio as an independent predictor of RV hypertrophy and ECG arrhythmia, emphasizing that worse pulmonary function directly correlates with cardiac compromise ^[12]. Additionally, hypertension and diabetes were also strong predictors, consistent with prior research demonstrating their contribution to endothelial dysfunction and vascular stiffness in COPD patients ^[13,14].

The high comorbidity burden (45% hypertensive, 35% diabetic) in this study echoes findings that systemic illnesses significantly amplify cardiovascular risks in COPD ^[15]. The observed 50% prevalence of family history of respiratory disease and 40% for cardiovascular disease may suggest underlying genetic and environmental susceptibilities ^[16].

These data strongly support integrating routine cardiovascular assessments—via ECG and echocardiography—into standard COPD care, especially in patients with moderate to very severe disease. Early recognition of RV dysfunction, PAH, and arrhythmias can enable timely interventions, including oxygen therapy, pulmonary vasodilators, and cardioprotective treatments ^[17].

Moreover, this study highlights the importance of multidisciplinary COPD care, where collaboration between pulmonologists, cardiologists, and internists improves outcomes, reduces exacerbations, and enhances quality of life ^[18]. Future research should focus on longitudinal follow-up and advanced imaging (e.g. cardiac MRI) to identify subclinical changes and refine predictive models ^[19,20].

CONCLUSIONS

This study underscores the substantial burden of cardiac dysfunction in COPD patients, with a clear association between disease severity and cardiovascular abnormalities. ECG and echocardiographic evaluations

provide critical insights into arrhythmias, QTc prolongation, RV hypertrophy, and pulmonary hypertension. Identifying key predictors, such as FEV1/FVC ratio, hypertension, and diabetes, can guide targeted interventions to improve outcomes. A multidisciplinary approach involving pulmonologists and cardiologists is essential for comprehensive COPD care.

LIMITATIONS

The study's cross-sectional design limits causal inference, and its single-center setting may reduce generalizability. Additionally, the lack of longitudinal data precludes assessment of the long-term impact of cardiac abnormalities on COPD progression and patient outcomes. The study also relied on standard echocardiographic parameters, which may not capture subtle cardiac changes detectable through advanced imaging techniques. Future research should incorporate multicentric, longitudinal designs with advanced imaging modalities to validate these findings and explore novel biomarkers of cardiac dysfunction in COPD.

CONTRIBUTION OF AUTHORS

Research concept- Santanu Das, Saptarshi Lahiri

Research design- Santanu Das, Saptarshi Lahiri

Supervision- Bipasha Adhvaryu, Ishita Das, Shayan Das

Materials- Santanu Das, Saptarshi Lahiri

Data collection- Santanu Das, Saptarshi Lahiri

Data analysis and interpretation- Bipasha Adhvaryu, Ishita Das, Shayan Das

Literature search- Santanu Das, Saptarshi Lahiri

Writing article- Santanu Das, Saptarshi Lahiri

Critical review- Bipasha Adhvaryu, Ishita Das, Shayan Das

Article editing- Santanu Das, Saptarshi Lahiri

Final approval- Bipasha Adhvaryu, Ishita Das, Shayan Das

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