

ELECTROCARDIOGRAPHIC AND ECHOCARDIOGRAPHIC FINDINGS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND THEIR CORRELATION WITH DISEASE SEVERITY: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is frequently complicated by cardiac involvement in the form of chronic cor pulmonale, right ventricular hypertrophy (RVH), and pulmonary arterial hypertension (PAH), which influence morbidity and prognosis. Electrocardiography (ECG) and echocardiography are complementary, non-invasive tools for detecting this involvement. The aim and objective to study the electrocardiographic and echocardiographic changes in patients with COPD and to correlate these changes with the severity of the disease. **Materials and Methods:** This prospective observational study was conducted in the Department of General Medicine, Government Medical College and General Hospital, Kadapa, over 18 months in 100 clinically and spirometrically confirmed COPD patients above 30 years of age. All patients underwent detailed history, clinical examination, chest radiography, 12-lead ECG, spirometry, and 2D Doppler echocardiography. Severity was graded by the GOLD classification. ECG was assessed for right ventricular hypertrophy (RVH) criteria, and systolic pulmonary artery pressure (PAP) was estimated by Doppler echocardiography using the modified Bernoulli equation in patients with ECG evidence of RVH. **Results:** Of 100 patients, 68% were male and 32% female (ratio 2:1); 64% were aged ≥ 50 years. Sixty-six percent had a positive smoking history, predominantly >20 pack-years. On GOLD grading, 48% had moderate and 34% had severe airflow obstruction. Chest radiography most commonly showed increased bronchovascular markings (54%). ECG evidence of RVH was found in 62% of patients, right axis deviation (51%) and R/S ratio >1 in V1 (54%) being the commonest individual signs. Doppler echocardiography confirmed elevated systolic PAP in 46% of the total cohort, corresponding to 75% of those with ECG evidence of RVH. **Conclusion:** Cardiac involvement is common in COPD and correlates with disease severity, smoking exposure, and advancing age. A simple 12-lead ECG is a practical first-line screening tool for right heart involvement in COPD, with echocardiography valuable for confirming and quantifying pulmonary arterial hypertension in those with a positive screen.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD), encompassing chronic bronchitis and emphysema, is among the most common respiratory illnesses in developing countries and is projected to become the third leading cause of death and the fifth leading cause of disability-adjusted life years lost worldwide.^[1] Despite this substantial burden, COPD

has historically received comparatively less research attention than other airway diseases such as asthma and lung cancer, and remains one of the most underfunded diseases in relation to its share of the global burden of disease.^[2]

Cigarette smoking is overwhelmingly the most important risk factor for the development of COPD, and the risk of disease and its severity rise with increasing pack-years of exposure.^[3] A major determinant of morbidity and mortality once COPD

is established is the cardiac involvement that accompanies progressive airflow limitation — chronic cor pulmonale, right ventricular hypertrophy (RVH), and pulmonary arterial hypertension (PAH).^[4]

The pathogenesis of pulmonary hypertension in COPD is multifactorial. Alveolar hypoxia, hypercarbia, and acidaemia produce pulmonary vasoconstriction; hyperinflated lungs compress the pulmonary vasculature; emphysematous destruction reduces the cross-sectional area of the pulmonary vascular bed; and hypoxia-driven polycythaemia increases blood viscosity and cardiac output. With time, these haemodynamic insults produce fixed structural remodelling of the pulmonary arterial wall, converting an initially reversible rise in pressure into sustained pulmonary hypertension.^[5]

The electrocardiogram (ECG), although relatively insensitive for detecting pulmonary hypertension, remains an inexpensive and universally available bedside screening tool. Signs such as a rightward P-wave axis (P pulmonale), right axis deviation, the S1, S2, S3 pattern, low-voltage QRS complexes, and an R/S ratio >1 in lead V1 reflect right atrial and right ventricular overload and carry independent prognostic significance in COPD.^[6] Echocardiography, in contrast, is nearly twice as sensitive as clinical examination for detecting cor pulmonale and permits non-invasive estimation of systolic pulmonary artery pressure from the tricuspid regurgitant jet velocity using the modified Bernoulli equation.^[7] Given the prognostic importance of cardiac involvement in COPD and the relative paucity of Indian data correlating electrocardiographic and echocardiographic abnormalities with clinical and spirometric disease severity, this prospective observational study was undertaken to evaluate ECG and echocardiographic changes in patients with COPD and their relationship to disease severity.

Aims and Objectives

To study the electrocardiographic and echocardiographic changes in patients with chronic obstructive pulmonary disease and to correlate these changes with the severity of the disease.

MATERIALS AND METHODS

Study design and setting: This was a prospective observational study conducted in the Department of General Medicine, Government Medical College and General Hospital, Kadapa, over a period of 18 months, following approval from the Institutional Ethics Committee (IEC No. 20/GMC/KDP/2023). Information was collected on a predesigned proforma administered by the interview technique, and written informed consent was obtained from all participants.

Study population: A total of 100 patients with COPD, above 30 years of age, attending the outpatient clinic or admitted as inpatients, were enrolled consecutively.

Inclusion criteria

Patients above 30 years of age with a clinical diagnosis of COPD — chronic cough, chronic sputum production, and dyspnea that was progressive, persistent, worse on exertion, and worsened during respiratory infections, with a history of exposure to recognised risk factors (principally cigarette smoking) — who satisfied spirometric criteria for the disease were included. The diagnosis was confirmed by spirometry: a post-bronchodilator FEV1 <80% of the predicted value together with an FEV1/FVC ratio <70% confirmed the presence of airflow limitation that was not fully reversible.

Exclusion criteria

Patients with other respiratory diseases (bronchial asthma, pulmonary tuberculosis, bronchiectasis, lung malignancy) and those with valvular heart disease, coronary artery disease, systemic hypertension, cardiomyopathy, or HIV/AIDS were excluded, in order to avoid confounding of the cardiac and pulmonary vascular findings under study.

Investigations

All patients underwent a detailed history and clinical examination, and were subjected to routine haematological and biochemical investigations, urine analysis, sputum examination (Gram stain, AFB stain, culture and sensitivity), the Mantoux test, ELISA for HIV, and arterial blood gas analysis. Chest radiography (postero-anterior and lateral views), a standard 12-lead electrocardiogram, spirometry, and 2-dimensional trans-thoracic echocardiography with Doppler assessment of pulmonary artery pressure were performed in all patients. Spirometry was performed according to American Thoracic Society recommendations,⁸ and severity of airflow obstruction was graded using the GOLD classification into mild, moderate, severe, and very severe categories on the basis of the post-bronchodilator FEV1 % predicted.^[9]

Electrocardiographic criteria

The 12-lead ECG was assessed for P pulmonale (P-wave amplitude >2.5 mm in leads II, III, aVF, or >1.5 mm in V1–V3), frontal-plane QRS/P-wave axis deviation, the S1, S2, S3 pattern, low-voltage QRS complexes, the Lead I sign, the R/S ratio in V1 and V6, and right bundle branch block. Right ventricular hypertrophy (RVH) was diagnosed when two or more of the following were present: right axis deviation greater than +110°, an R wave in V1 ≥7 mm, an R/S ratio in V1 >1, an R/S ratio in V5 or V6 ≤1, an S wave in V5 or V6 >2 mm, or a qR pattern in V1.^[10]

Echocardiographic criteria

Two-dimensional and Doppler echocardiography were performed in all patients who showed ECG evidence of RVH. The systolic pulmonary artery pressure was estimated by applying the modified Bernoulli equation ($P = 4V^2$, where V is the peak velocity of the tricuspid regurgitant jet) and adding the derived pressure gradient to the estimated mean right atrial pressure.^[11] Pulmonary arterial hypertension was defined as a systolic pulmonary

artery pressure greater than 30 mmHg, or a mean pulmonary artery pressure greater than 20 mmHg.

Statistical analysis

Data were entered into a predesigned format and analysed using descriptive statistics; results are expressed as frequencies and percentages, and are presented in tabular form.

RESULTS

Of the 100 patients with COPD studied, the majority (44%) were aged 50–59 years, followed by 33% aged 40–49 years, 20% aged ≥60 years, and only 3% aged 30–39 years (Table 1); COPD was thus predominantly a disease of the middle-aged and elderly, and was uncommon below the age of 40 years.

Table 1: Age distribution

Age (years)	Predominant bronchitis	Predominant emphysema	Total	%
30–39	3	0	3	3
40–49	24	9	33	33
50–59	33	11	44	44
≥60	10	10	20	20

Sixty-eight of the 100 patients were male and 32 were female, giving a male:female ratio of approximately 2:1. [Table 2]

Table 2: Gender distribution

Gender	Predominant bronchitis	Predominant emphysema	Total	%
Male	46	22	68	68
Female	24	8	32	32

Sixty-six of the 100 patients (66%) had a positive smoking history, most commonly with more than 20

pack-years of exposure; of the 34 non-smokers, 24 (71%) were female. [Table 3]

Table 3: Smoking pattern (pack-years)

Pack-years	Predominant bronchitis	Predominant emphysema	Total	%
>30	24	12	36	36
20–30	16	6	22	22
<20	6	2	8	8
Non-smoker	24	10	34	34

On GOLD grading of the post-bronchodilator FEV1%, moderate disease (48%) and severe disease (34%) accounted for the majority of cases, with only

12% classified as mild and 6% as very severe [Table 4]; most patients had an FEV1/FVC ratio in the range of 40–60%.

Table 4: Severity of airflow obstruction (GOLD classification)

Severity (post-bronchodilator FEV1% predicted)	Predominant bronchitis	Predominant emphysema	Total	%
Mild (>80%)	3	9	12	12
Moderate (50–80%)	32	16	48	48
Severe (30–50%)	28	6	34	34
Very severe (<30%, or <50% with respiratory failure)	4	2	6	6

Chest radiography most commonly showed increased bronchovascular markings (54%), followed by emphysematous changes (32%), cardiomegaly

(12%), and radiographic evidence of pulmonary hypertension (12%). [Table 5]

Table 5: Chest radiograph findings

CXR finding	Predominant bronchitis	Predominant emphysema	Total	%
Increased bronchovascular markings	48	6	54	54
Emphysematous changes	8	24	32	32
Cardiomegaly	8	4	12	12
Evidence of pulmonary hypertension	12	0	12	12

Electrocardiographic abnormalities were common [Table 6] Right axis deviation was the single commonest finding (51%), followed by an R/S ratio >1 in V1 (54%), P pulmonale (23%), an R/S ratio <1 in V6 (10%), right bundle branch block (10%), the Lead I sign (5%), low-voltage QRS complexes (4%),

the S1, S2, S3 pattern (4%), left axis deviation (4%), ventricular ectopics (3%), and multifocal atrial tachycardia (2%). Overall, ECG criteria for right ventricular hypertrophy were satisfied in 62 of the 100 patients.

Table 6: Electrocardiographic findings

ECG change	Predominant bronchitis	Predominant emphysema	Total	%
P pulmonale	19	4	23	23
Right axis deviation	40	11	51	51
Left axis deviation	4	0	4	4
RBBB	2	8	10	10
Low voltage QRS	1	3	4	4
Lead I sign	4	1	5	5
R/S ratio in V6 <1	8	2	10	10
R/S ratio in V1 >1	42	12	54	54
S1, S2, S3 pattern	3	1	4	4
Ventricular ectopics	2	1	3	3
Multifocal atrial tachycardia	2	0	2	2

Table 7: Systolic pulmonary artery pressure on Doppler echocardiography

Systolic PAP (mmHg)	Predominant bronchitis	Predominant emphysema	Total	%
31–40	14	7	21	21
41–50	9	4	13	13
51–60	5	1	6	6
61–70	4	1	5	5
71–80	1	0	1	1

DISCUSSION

In the present study, COPD was predominantly a disease of middle and old age, with 64% of patients aged 50 years or older and only 3% below the age of 40 years, consistent with earlier Indian community-based data showing that COPD is rare below the age of 30–40 years and increases in prevalence with advancing age.^[12] This age-related increase most plausibly reflects the cumulative effect of smoking pack-years and other environmental exposures, the tendency of patients to under-report early symptoms and present to hospital only once disease is advanced, and improving life expectancy that allows more patients to survive into the age range at which COPD becomes clinically manifest.

The male:female ratio in this study was 2:1, at the higher end of the range of 1.32:1 to 2.60:1 and above the pooled median ratio of about 1.6:1 reported across Indian population studies.^[3] Comparable south Indian data have shown a fairly consistent ratio of around 1.6:1 over time,^[12] while the male preponderance tends to be more marked in urban populations, most plausibly reflecting the higher prevalence of tobacco smoking among Indian men; under-recognition of COPD in women has also been described as a contributing factor to this apparent gender disparity.^[13]

Sixty-six percent of patients in this cohort had a positive smoking history, broadly comparable with the smoker:non-smoker prevalence ratios of 61.6–91.1% (median 82.3%) reported across Indian population studies,³ and severity of airflow obstruction increased with increasing pack-years of exposure, in keeping with the well-established dose-response relationship between cigarette consumption and COPD risk, and with the demonstrated benefits of smoking cessation on long-term outcomes.^[14] Notably, almost three-quarters of the non-smokers in this study were women, underscoring that exposures other than direct tobacco smoking — indoor biomass

fuel combustion among them — remain an important contributor to COPD among Indian women.^[15]

Radiologically, increased bronchovascular markings were the commonest finding, consistent with the chronic-bronchitis-predominant phenotype of this cohort, while a substantial minority showed emphysematous change or radiographic signs of pulmonary hypertension. It is well recognised that a normal chest radiograph does not exclude significant emphysema; high-resolution computed tomography has demonstrated emphysema in a majority of heavy smokers with normal plain radiographs.^[16]

Electrocardiographic evidence of right ventricular hypertrophy was found in 62% of patients in this study, considerably higher than the 24% and 15% prevalence of a markedly deviated P-wave axis reported in two earlier series,^[17] but consistent with the well-documented insensitivity and marked inter-study variability of individual ECG criteria for cor pulmonale in COPD.^[18] Right axis deviation and an R/S ratio >1 in V1 were the most frequent individual abnormalities in this study, in keeping with prior descriptions of right axis deviation, tall R waves in the right precordial leads, and P pulmonale as the electrocardiographic hallmarks of chronic cor pulmonale.^[6] A P-wave axis of 70° or more has been proposed as a screening criterion for COPD, while an axis of 90° or more has been linked to advanced disease and independently predicts reduced long-term survival, together with the S1, S2, S3 pattern.^[18] Kok-Jensen, studying 228 patients with COPD, similarly found survival to be markedly poorer in those with a QRS axis of +90° to 180° combined with a tall P wave in lead II, only 37–42% of such patients surviving four years.^[19]

Echocardiographically, 46% of the total cohort — and 75% of those with ECG evidence of RVH — had confirmed pulmonary arterial hypertension by Doppler estimation of systolic pulmonary artery pressure. This finding illustrates the complementary relationship between ECG and echocardiography in COPD: ECG signs of RVH are highly specific but

relatively insensitive for pulmonary hypertension, whereas Doppler echocardiography, though considerably more sensitive, may still under- or over-estimate directly measured right heart pressures, particularly as pulmonary pressures rise.^[20] The pathogenesis of pulmonary hypertension in this setting is multifactorial — hypoxic pulmonary vasoconstriction, structural remodelling and loss of the pulmonary vascular bed, compression of vessels by hyperinflated lungs, and increased blood viscosity secondary to hypoxia-driven polycythaemia all contribute, chronic alveolar hypoxia being considered the single most important driver of the vascular remodelling that ultimately produces fixed pulmonary hypertension.^[21]

The correlation between electrocardiographic and echocardiographic abnormalities and the severity of airflow obstruction was evident in this cohort, with a greater burden of right heart abnormalities observed among patients with more severe spirometric impairment. This is in keeping with earlier longitudinal observations that pulmonary artery pressure rises in proportion to the degree of airflow obstruction as COPD advances; in a five-year follow-up of COPD patients, Weitzenblum and colleagues demonstrated a slow but steady annual rise in pulmonary artery pressure, with a somewhat faster rate of rise among those without pulmonary hypertension at baseline.^[22]

Right bundle branch block, low-voltage QRS complexes, and the S1, S2, S3 pattern were relatively uncommon in this study (10%, 4%, and 4% respectively), consistent with earlier reports describing these signs as less specific and less consistently associated with adverse outcomes than P-wave axis deviation and the standard criteria for right ventricular hypertrophy.¹⁸ Ventricular ectopics and multifocal atrial tachycardia were similarly infrequent (3% and 2% respectively); cardiac arrhythmias in COPD have been more strongly linked to episodes of acute respiratory decompensation and hypoxaemia than to the chronic stable state, which may explain their low prevalence in this predominantly stable, outpatient-based cohort.^[23]

Limitations

This study has certain limitations. It was conducted at a single tertiary-care centre with a modest sample of 100 patients, which may limit the generalisability of the findings to the wider COPD population. The cross-sectional, observational design does not allow assessment of how electrocardiographic and echocardiographic abnormalities evolve with disease progression over time. Echocardiography was performed only in patients who showed ECG evidence of right ventricular hypertrophy rather than in the entire cohort, which may have led to an underestimation of the true prevalence of pulmonary arterial hypertension among patients with a normal screening ECG. Right heart catheterisation, the reference standard for measurement of pulmonary artery pressure, was not performed, and Doppler-derived estimates of systolic pulmonary artery

pressure are known to correlate less well with directly measured pressures as pressures rise.^[20] Finally, potential confounders such as body mass index, arterial blood gas parameters, and exercise capacity were not analysed for their independent contribution to the electrocardiographic and echocardiographic abnormalities observed.

CONCLUSION

Cardiac involvement is common in patients with COPD and correlates with the severity and duration of the underlying airway disease. In this study, COPD was more frequent among men and in the middle-aged and elderly, and was strongly associated with a smoking history of more than 20 pack-years. Electrocardiographic abnormalities suggestive of right ventricular hypertrophy — principally right axis deviation, an R/S ratio >1 in V1, and P pulmonale — were present in more than 60% of patients, and three-quarters of these were confirmed to have raised pulmonary artery pressure on Doppler echocardiography. These findings support the use of a simple 12-lead ECG as a practical, inexpensive first-line tool for screening patients with COPD for cardiac involvement, with echocardiography reserved for confirmation and quantification of pulmonary arterial hypertension in those with a positive screen. Given the prognostic significance of cor pulmonale in COPD, routine ECG screening — followed by echocardiography where indicated — should be considered an integral part of the clinical evaluation of patients with this disease.

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