

Chronic Obstructive Pulmonary Disease and Cardiac Comorbidities

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Abstract

Introduction: Chronic obstructive pulmonary disease (COPD) is a global health issue with cigarette smoking being an important risk factor. COPD, defined by global initiative for chronic obstructive lung disease (GOLD) as a preventable and treatable disease with some significant extrapulmonary effects, is a very common clinical entity in clinical practice, COPD is associated with significant extrapulmonary (systemic) effects among which cardiac manifestations are most common, COPD affects pulmonary blood vessels, right ventricle, as well as left ventricle leading to the development of pulmonary hypertension (PH), cor pulmonale (COR-P), right and left ventricular dysfunction, echocardiography provides a rapid, non-invasive, portable, and accurate method to evaluate cardiac functions, and early diagnoses and intervention for cardiac comorbidities would reduce mortalities. Hence, the present study was undertaken with the following aims and objectives: (1) To assess the cardiac changes secondary to COPD by echocardiography and (2) to find the correlation between echocardiography findings and the severity of COPD using GOLD guidelines.

Materials and Methods: A prospective study was conducted at the Department of Pulmonary Medicine, SVS Medical College, Mahabubnagar. A total of 45 patients of COPD according to GOLD guidelines were taken into the study; all patients underwent investigations such as chest X-ray posteroanterior view, electrocardiography, and spirometry followed by 2D echo.

Results: We investigated 42 male and 3 female patients ranging from 50 to 75 years of age, of these cases, 2 among Category D had left ventricular diastolic dysfunction changes, 3 from Category C and 10 from Category D were diagnosed with PH, and 4 from Category D had changes of COR-P.

Conclusion: Our study puts emphasis on early cardiac screening of all COPD patients which will be helpful in the assessment of the prognosis and will further assist in identifying the individual likely to suffer increased morbidity and mortality.

Key words: 2D echo, Chronic obstructive pulmonary disease, Cor pulmonale

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a global health issue with smoking being the most important risk factor. By 2020, it will be the third most leading cause of mortality and fifth leading cause of morbidity in the world.^[1,2] There is a crude estimate of about 30 million people in India suffering from COPD, and the death rate

is among the highest in the world, and data suggest that about 556,000, i.e., >20% of total 2,748,000 die in India annually.^[3]

Cardiovascular disease (CVD) is a major comorbidity in COPD. General population studies and studies in patients with COPD indicate that COPD is an important risk factor for ischemic heart disease and sudden cardiac death. There is evidence of an association between COPD and CVD, and although COPD and CVD clearly share common risk factors such as smoking, COPD has been described as an independent risk factor for the development of CVD.

COPD affects pulmonary blood vessels, right ventricle, as well as left ventricle leading to the development of pulmonary hypertension (PH), cor pulmonale (COR-P),

Access this article online



www.ijss-sn.com

Month of Submission : 12-2017
Month of Peer Review : 01-2018
Month of Acceptance : 01-2018
Month of Publishing : 02-2018

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right ventricular dysfunction, and left ventricular dysfunction. Ischemic heart disease is one of the main causes of mortality in COPD.^[4] The observed association between COPD and CVD may be explained due to the coexistence of shared risk factors such as smoking, age, sex, and inactivity. Coexistence of both diseases is very common and has diagnostic, therapeutic, and prognostic implications.^[5,6] Chronic bronchitis alone increases the risk of coronary deaths by 50%. Reduced ratio of forced expiratory volume in 1 s (FEV1) to forced vital capacity is also an independent risk factor for coronary events, increasing the risk by 30%. With every 10% decrease in FEV1, all-cause mortality increases by 14% and an increase in cardiovascular mortality by 28%.^[7] In more advanced COPD, CVDs account for 20–25% of all deaths.^[8]

Left ventricular diastolic dysfunction (LVDD) is defined as the inability of the ventricle to fill to a normal end-diastolic volume, both during exercise as well as at rest, while left atrial pressure does not exceed 12 mmHg.^[9-11] LVDD is a common phenomenon in COPD.^[12-14] Abnormal left ventricular function is seen in COPD due to many factors such as hypoxia, acidosis, ventricular interdependence, lung hyperinflation, and distension.

Left ventricular systolic dysfunction is a disorder characterized by failure of the left ventricle to produce adequate output despite an increase in distending pressure and end-diastolic volume.

PH is an increase of blood pressure in the pulmonary artery, pulmonary vein, or pulmonary capillaries, together known as the lung vasculature.

PH is defined as systolic pulmonary artery pressure (sPAP) >30 mmHg, and it is classified into mild, moderate, and severe grades as sPAP 30–50 mmHg, sPAP 50–70 mmHg, and sPAP >70 mmHg, respectively.^[15]

COR-P is defined as an alteration in the structure and function of the right ventricle caused by a primary disorder of the respiratory system. PH is the common link between lung dysfunction and the heart in COR-P. COR-P can develop due to various cardiopulmonary diseases. COR-P usually has a slow and chronic progression, but acute onset and life-threatening complications can occur.^[16]

Echocardiography provides a rapid, non-invasive portable, and almost accurate method to evaluate the right ventricle function, right ventricular filling pressure, tricuspid regurgitation, left ventricular function, and valvular functions.^[17] It has been studied that echocardiography measured pulmonary arterial pressure closely correlates with pressure measured by right heart catheterization.^[18,19]

This study was undertaken to evaluate cardiac function with echocardiography in COPD patients which may further help to assess the prognosis and assist in identifying the individuals likely to suffer increased morbidity and mortality.

MATERIALS AND METHODS

A prospective study was done at Tertiary Care Hospital, Mahabubnagar, Telangana. 45 patients were diagnosed COPD according to Global Initiative for Chronic Obstructive Lung Disease guideline. The subject included between the age of 50 and 75 years with informed and written consent.

The patients with pneumonia, tuberculosis, bronchial asthma, interstitial lung disease, carcinoma lungs, and other lung pathologies were excluded from the study.

The other category of patients who were excluded had a history of cardiac diseases such as ischemic heart disease, rheumatic heart disease, valvular heart diseases, congenital heart disease, and others.

All the patients were asked for the detailed history of respiratory as well as cardiovascular symptoms and were clinically examined for the signs of biventricular hypertrophy, cardiomegaly, PH, and heart failure.

Patients were investigated for routine investigations such as complete blood profile, renal function test, random blood sugar, electrocardiography, sputum for Gram stain, chest X-ray, and 2D echo.

Statistical Analysis

The statistical analysis of data has been done. The age and sex distribution of all patients, their body mass index (BMI), severity of COPD, echocardiography findings, the frequency of COR-P, and its relation to COPD have been represented graphically.

Correlation between the cardiac parameters on echocardiography findings and pulmonary parameters on spirometry findings has been done to find the relation and to estimate the risk of morbidity.

The mean and standard deviation (SD) of FEV1 and PH of all the patients has been calculated along with Pearson correlation value.

RESULTS

We investigated 42 male and 3 female patients in the age group of 50–75 years. Mean BMI was 19.59 kg/m². Of

45 patients, 24 (48%) patients were underweight. The mean pack years for smoking were 31.33. Mean \pm SD calculated for FEV1 was 49.05 ± 14.61 .

The most common finding on echocardiography was PH 13/45 (28.8%), next to which was LVDD 2/45 (4.5%). The study showed 28/45 (62%) of normal cases.

Of 13 patients of PH, there were 6 (46.15%), 3 (23.07%), and 4 (30.76%) patients of mild, moderate, and severe, respectively. Mean \pm SD calculated for PH ($n = 45$) was 35.59 ± 15.47 .

Of 2 patients with LVDD, 0 (0%) were of Grade 1, 1 (50.0%) of Grade 2, and 1 (50.0%) of Grade 3.

The severity of PH, COR-P, and LVDD was increasing with increasing severity of COPD [Figure 1].

DISCUSSION

There are various cardiac changes seen in the patients suffering from COPD. Right-sided cardiac dysfunction and PH are one of the main established complications described in many studies done worldwide. Cigarette smoking and other exposure factors lead to inflammatory changes which disrupt the vascular pulmonary endothelium, and on the other hand, changes of chronic bronchitis and emphysema lead to chronic hypoxic conditions which result into pulmonary artery remodeling and vasoconstriction. The other mechanism leading to the damage is the change in intrinsic pulmonary vasodilator substances such as prostacyclin synthase, decrease in endothelial nitric oxide synthase, and increase in endothelial 1. As a result, we see remodeling, changes in respiratory mechanics, and also, increase in blood viscosity. All these factors lead to PH. PH increases the afterload of the right ventricle and increase of the right ventricular work as well. If we summarize the whole mechanism of COPD, hypoxic vasoconstriction, and PH will result in right ventricular hypertrophy and even its dilatation giving a clinical presentation of the right heart failure [Table 1].

There are no exact data of PH prevalence in COPD; pulmonary artery pressures were seen elevated in about 20–90% of patients when measured by right heart catheterization, with evidence of changing severity in pulmonary hemodynamics with the severity in airflow obstruction. Two studies have shown an abnormal increase in MPAP (PPA) in COPD of 0.4–0.6 mmHg per year. These studies illustrate that PH in COPD progresses slowly and occur in mild as well as severe forms of disease.^[21,22]

Several studies have demonstrated the prognostic value of PH in COPD patients. Severe the PH, more poor is the prognosis, even in patients of COPD receiving long-term oxygen therapy. In one of the studies, showing the 5-year survival rate was 50% in patients with mild PH (20–30 mmHg), 30% in those with moderate and severe PH (30–50 mmHg), and 0% in small group of patients suffering from severe PH (>50 mmHg).

Comparison between Severity of COPD and Cardiac Changes

In our study, there is a direct linear correlation seen between PH and severity of COPD (FEV1).

COR-P was present in 8.8% of patients in our study. Approximately, about 25% of patients of COPD develop COR-P. One of the autopsy studies showed that 40% of patients of COPD had COR-P. In comparison, the results are matching to our study. The cause of LVDD in COPD patients could be due to chronic hypoxemia leading to the changes in myocardial relaxation, distension, and lung hyperinflation making the parietal pleura stiff and similarly the walls of cardiac fossa adding the load to the walls of the ventricle and also due to ventricular interdependence. In our study, LVDD was present in ~5% of patients as compared to 47.5% seen in the study done by Gupta *et al.*^[20]

Table 1: Whole mechanism

Findings	Category				Total	P
	A	B	C	D		
Normal	10	11	2	3	26	<0.0001
PAH	0	0	3	10	13	
COR-P	0	0	0	4	4	
LVDD	0	0	0	2	2	
LVSD	0	0	0	0	0	
L VH	0	0	0	0	0	
Total	10	11	5	19	45	

COR-P: Cor pulmonale, LVDD: Left ventricular diastolic dysfunction, LVSD: Left ventricular systolic dysfunction, LVH: Left ventricular hypertrophy

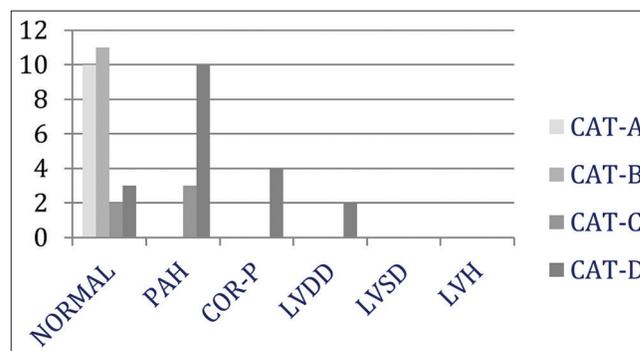


Figure 1: Simple bar diagram showing the presence of various cardiac morbidities in different categories. PH: Pulmonary hypertension, COR-P: Cor pulmonale, LVDD: Left ventricular diastolic dysfunction, LVSD: Left ventricular systolic dysfunction, LVH: Left ventricular hypertrophy

One of the limitations of our study was a small size, the other factors being not able to perform the right-sided heart catheterization or employ transesophageal echocardiography. Further, well-designed cohort studies and the use of future three-dimensional echocardiography with optimal sample size will be helpful in defining the role of echocardiography in COPD patients.

CONCLUSION

The study shows a high prevalence of cardiac comorbidities such as PH, COR-P, and LV dysfunction in COPD patients. The severity of complications increases with severity of COPD and makes a linear relation. This relation was also seen in Grade 2 and 3 LV dysfunctions and was not seen in Grade 1.

Hence, our study puts emphasis on cardiac screening of all COPD patients.

REFERENCES

1. Gunen H, Hacievliyagil SS, Kosar F, Mutlu LC, Gulbas G, Pehlivan E, *et al.* Factors affecting survival of hospitalized patients with COPD. *Eur Respir J* 2005;26:234-41.
2. World Health Organisation. World Health Report. Geneva: World Health Organisation; 2000. Available from: <http://www.who.int/whr/2000/en/statistics.htm>. [Last accessed on 2017 Dec 12].
3. Lopez AD, Shibuya K, Rao C, Mathers CD, Hansell AL, Held LS, *et al.* Chronic obstructive pulmonary disease: Current burden and future projections. *Eur Respir J* 2006;27:397-412.
4. Anthonisen NR, Skeans MA, Wise RA, Manfreda J, Kanner RE, Connett JE, *et al.* The effects of a smoking cessation intervention on 14.5-year mortality: A randomized clinical trial. *Ann Intern Med* 2005;142:233-9.
5. Rutten FH, Moons KG, Cramer MJ, Grobbee DE, Zuihoff NP, Lammers JW, *et al.* Recognising heart failure in elderly patients with stable chronic obstructive pulmonary disease in primary care: Cross sectional diagnostic study. *BMJ* 2005;331:1379.
6. Rutten FH, Cramer MJ, Lammers JW, Grobbee DE, Hoes AW. Heart failure and chronic obstructive pulmonary disease: An ignored combination? *Eur J Heart Fail* 2006;8:706-11.
7. Sin DD, Man SF. Chronic Obstructive Pulmonary Disease as a Risk Factor for Cardiovascular Morbidity and Mortality. *Proceedings of the American Thoracic Society. Vol. 2. Symposium: Chronic obstructive Pulmonary Disease: A Disorder of the Cardiovascular and Respiratory Systems*; 2005. p. 8-11.
8. Sin DD, Anthonisen NR, Soriano JB, Agusti AG. Mortality in COPD: Role of comorbidities. *Eur Respir J* 2006;28:1245-57.
9. Kitzman DW, Little WC, Brubaker PH, Anderson RT, Hundley WG, Marburger CT, *et al.* Pathophysiological characterization of isolated diastolic heart failure in comparison to systolic heart failure. *JAMA* 2002;288:2144-50.
10. Rodeheffer R, Miller W, Burnett J. Pathophysiology of circulatory failure. In: Giuliani E, Gersh B, Megoon M, Hayes D, Schaff H, editors. *Mayo Clinic Practice of Clinical Cardiology*. 3rd ed. St. Louis: Mosby; 1996. p. 556-8.
11. Vasan RS, Benjamin EJ, Levy D. Prevalence, clinical features and prognosis of diastolic heart failure: An epidemiologic perspective. *J Am Coll Cardiol* 1995;26:1565-74.
12. Boussuges A, Pinet C, Molenat F, Burnet H, Ambrosi P, Badier M, *et al.* Left atrial and ventricular filling in chronic obstructive pulmonary disease. An echocardiographic and doppler study. *Am J Respir Crit Care Med* 2000;162:670-5.
13. Bhattacharyya P, Chowdhury SR, Nag S, Sarkar D, Ghosh G, Bardhan S, *et al.* Diastolic dysfunction in advanced COPD patients: Early results of an observational study. *Respirology* 2004;9:A98.
14. Rappaport E. Cor pulmonale. In: Murray JJ, Nadel JA, Mason RM, Boushey H, editors. *Textbook of Respiratory Medicine*. 4th ed. Philadelphia: W.B. Saunders; 2000. p. 1631-48.
15. Chemla D, Castelain V, Humbert M, Hébert JL, Simonneau G, Lecarpentier Y, *et al.* New formula for predicting mean pulmonary artery pressure using systolic pulmonary artery pressure. *Chest* 2004;126:1313-7.
16. Weitzenblum E, Chaouat A. Cor pulmonale. *Chron Respir Dis* 2009;6:177-85.
17. Daniels LB, Krummen DE, Blanchard DG. Echocardiography in pulmonary vascular disease. *Cardiol Clin* 2004;22:383-99, vi.
18. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by doppler ultrasound in patients with tricuspid regurgitation. *Circulation* 1984;70:657-62.
19. Tramarin R, Torbicki A, Marchandise B, Laaban JP, Morpurgo M. Doppler echocardiographic evaluation of pulmonary artery pressure in chronic obstructive pulmonary disease. A European multicentre study. Working Group on Noninvasive Evaluation of Pulmonary Artery Pressure. European Office of the World Health Organization, Copenhagen. *Eur Heart J* 1991;12:103-11.
20. Gupta NK, Agrawal RK, Srivastav AB, Ved ML. Echocardiographic evaluation of heart in chronic obstructive pulmonary disease patient and its co-relation with the severity of disease. *Lung India* 2011;28:105-9.
21. Kessler R, Faller M, Weitzenblum E, Chaouat A, Aykut A, Ducoloné A, *et al.* "Natural history" of pulmonary hypertension in a series of 131 patients with chronic obstructive lung disease. *Am J Respir Crit Care Med* 2001;164:219-24.
22. Oswald-Mammosser M, Weitzenblum E, Quoix E, Moser G, Chaouat A, Charpentier C, *et al.* Prognostic factors in COPD patients receiving long-term oxygen therapy. Importance of pulmonary artery pressure. *Chest* 1995;107:1193-8.

How to cite this article: Rao VK, Naseer MA, Reddy KS, Bhaskar L, Waghay P. Chronic Obstructive Pulmonary Disease and Cardiac Comorbidities. *Int J Sci Stud* 2018;5(11):53-56.

Source of Support: Nil, **Conflict of Interest:** None declared.