ORIGINAL RESEARCH

A study to determine Right Heart changes and Pulmonary Artery Pressure in stable COPD patients as per GOLD criteria

[1]Vibhor Dubey, [2]Adnan Hamza, [3]Sachet Dawar, [4]RC Meena

[1]Resident, [4]Professor & Head, Department of Respiratory Medicine, National Institute of Medical Science & Research, NIMS University, Jaipur, Rajasthan, India
[2]Senior Resident, Department of Respiratory Medicine, Govt. Medical College, Srinagar, Jammu & Kashmir, India
[3]Assistant Professor, Department of Respiratory Medicine, Noida International Institute of Medical Sciences, GautamBudh Nagar, Uttar Pradesh, India

Correspondence:
VibhorDubey
Resident, Department of Respiratory Medicine, National Institute of Medical Science & Research, NIMS University, Jaipur, Rajasthan, India
Email:vibhordubey2010@gmail.com

Abstract

Background: Chronic Obstructive Pulmonary Disease (COPD) has considerable effects on cardiac functions primarily affecting the pulmonary vasculature and then right ventricle along with left ventricle. These cardiac effects complicate the clinical course of COPD and correlate inversely with survival.

Aim: A cross sectional study was conducted in stable COPD patient at National InstitueOf Medical Science & Research, Jaipur with the following aims and objectives:-To study the right heart changes in stable COPD patients and to determine pulmonary arterial pressure in stable COPD patient.

Methodology: In all, 260 outdoor and indoor patients attending the Department of Respiratory Medicine with clinical history consistent with COPD and diagnosed so as per GOLD guidelines, between January 2018 to June 2019 and fulfilling the inclusion criteria were included in our study. All the included subjects were evaluated in detail as regards to a detailed clinical history and physical examination, Peripheral Blood Tests: Complete Blood Counts, Urea, Creatinine, SGOT, SGPT, Fasting Blood Sugar. Overnight and spot sputum sample was tested for Acid Fast Bacilli by Ziehl-Neelsen method. Other tests included Urine routine and microscopic examination, Chest roentgenograph (Postero-Anterior view), Spirometry and post-bronchodilator spirometry,Electrocardiography and 2D Echocardiography.

Results: Males outnumbered females in our study (61.53% and 38.46% respectively). The mean age of the patients was 62.38 ± 11.03 years. Mean BMI was 20.5 ± 2.39 & about 44.61% of our study subject were underweight.Bidi (70.38%) was the more common type of smoking in our COPDpatients.The majority of COPD cases had an MRC score grade 3 (45.76%). Cough (96.92%), expectoration (96.15%) & S.O.B. (98.46%) were almost universal. All ECG & 2D Echo parameters (such as right axis deviation, right ventricular hypertrophy and right ventricular dilatation) correlate with the severity of disease.Pulmonary hypertension was seen in 51.15% and their existed a linear correlation between Pulmonary hypertension& severity of the disease.

Conclusion: In lieu of the above findings, it is concluded that it is important to assess a patient of COPD not only with clinical, radiological, spirometric criteria but also with ECG & Echocardiography in order to assess the severity & outcome of disease.

Introduction

Chronic obstructive pulmonary disease (COPD) as defined by Global initiative for Obstructive Lung Disease (GOLD) is a complex respiratory disease of the airway characterized by persistent and usually progressive airflow limitation.[1]COPD is a common and disabling condition that has impacted over hundreds of millions of people globally. Currently, it is the fourth leading cause of mortality worldwide, responsible for more than 3 million deaths every year. Crude estimates suggest there are 30 million COPD patients in India.[2]

COPD and cardiovascular disease (CVD) share recognized risk factors such as ageing, history of cigarette smoking (or other exposures) and a sedentary lifestyle, and often coexist.[3,4,5]Patients with COPD and CVD are found to have high rates of morbidity, including worsening quality of life, dyspnoea and exercise intolerance, and thus they have a increased risk of hospitalisation for COPD and for CVD.[6,7]

About 50% of all hospitalization and nearly one-third of all deaths in cases whereForced expiratory volume in 1 second(FEV$_1$) is <50% of predicted are found to be in cases where COPD and CVD coexist.[4,1]Also their co-existence increases the risk of frequent exacerbations [8] and mortality.[3,6,9]In COPD, the right side of the heart is most commonly affected. [4] Right ventricular dysfunction and pulmonary vascular disease are well known to complicate the clinical course of COPD and correlate inversely with survival.

Reduction of the pulmonary vascularbed and hypoxia-induced pulmonary vasoconstriction leads toincrease in pulmonary vascular resistance and pulmonary arterial hypertension (PAH). Clinical studies haveshown that hypoxemia is one of the major determinantsof pulmonary hypertension.[10,11] Different patternsof hemodynamic abnormalities have been describedin COPD. Patients with a severe obstructive ventilatoryimpairment but with relatively normal arterialblood gas values do not usually demonstrate pulmonaryhypertension during rest. Patients with hypoxemiatypically demonstrate pulmonary hypertensionat rest accompanied by clinical and ECG evidence ofRight Ventricle(RV) hypertrophy. Post-mortem findings provide furtherevidence of the relation between hypoxemia andthe development of RV hypertrophy.[10,12,13]

From the above evidence it is clear that there is a direct correlation between Pulmonary Hypertension in patients with COPD.Several investigational tools currently exist for the assessment of pulmonary vascular disease in COPD, but right heart catheterization remains the gold standard.[14]Yet, echocardiography provides a rapid, non-invasive and accurate method to evaluate the RV functions along with left ventricular function.[4]

Material & methods

All outdoor and indoor patients attending the department of Respiratory Medicine with clinical history consistent with Chronic Obstructive Pulmonary Disease (COPD) and diagnosed so as per GOLD guidelines (GOLD-2017), during January 2018 to January 2020 were enrolled. Approval of the Institutional Ethical Committee was obtained. A written informed consent was taken from all the patients after explaining the study protocol.

Ours was a cross sectional study conducted with the aim to determine pulmonary arterial pressure and right heart changesin stable COPD patients and also to find out co-existing left heart changes, if any.
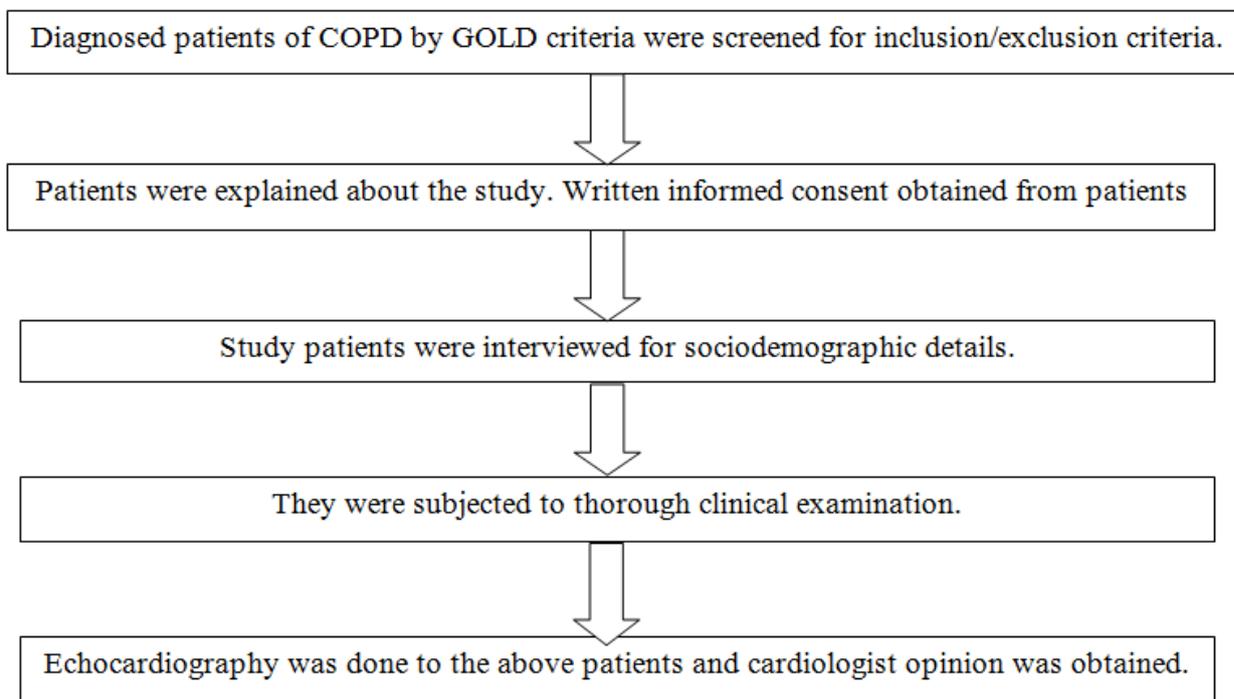
Stable COPD patients (as per GOLD criteria) aged 40 years and above willing to participate in study were included. Patients with history of Acute Exacerbation of COPDor systemic steroid use in past 3 months, present or past history of wheeze, chest tightness, eye allergy, nasal allergy or skin allergy, suggesting bronchial asthma, other illnesses like active pulmonary tuberculosis, malignancy, renal or hepatic disease and those who refused consent were excluded from the study.

All recruited patients were subjected to routine investigations: Haemoglobin %, Total count, Differential count, Erythrocyte Sedimentation Rate(ESR), Urine albumin, sugar, microscopy, Sputum for Acid fast bacilli and gram stain, random blood sugar, blood urea, serum creatinine

All cases were subjected to pulmonary function test using spirometric evaluation of Forced expiratory volume in one second (FEV_1), Forced vital capacity (FVC), Ratio of FEV_1 / FVC. The best of three attempts were taken for analysis. FEV_1 is decreased in obstructive lung diseases, FVC is normal or slightly reduced. COPD severity was assessed by combining the symptomatic assessment with the patient's spirometric classification, comorbidities and /or risk of exacerbations. A 12 lead ECG was taken in all the patients under study and the following pointswere noted: 'P' wave changes, standard criteria for RVH taken into consideration, Low voltage complexes, Poor progression of 'R' waves, Incomplete Right Bundle Branch Block(RBBB) and Arrhythmias, if any.

All patients were then subjected to echocardiographic examination including 2-D and M-mode echocardiography to note the presence of pulmonary hypertension, right ventricular hypertrophy, right ventricular dilatation and right ventricular failure. The following points were noted:Pulmonary artery diameter, Evidence of pulmonary hypertension on M-mode examination of pulmonary valve, Right ventricular hypertrophy (RVH), Right ventricular and diastolic dimension, Right atrial dilatation, Right ventricular failure, Tricuspid regurgitation, RV wall motion abnormality, Dilatation of inferior venacava and hepatic veins. The presence of right ventricular dilation, right ventricular hypertrophy or right ventricular failure was taken as evidence of cor-pulmonale.

Flow diagram of study design

Diagnosed patients of COPD by GOLD criteria were screened for inclusion/exclusion criteria.

↓

Patients were explained about the study. Written informed consent obtained from patients

↓

Study patients were interviewed for sociodemographic details.

↓

They were subjected to thorough clinical examination.

↓

Echocardiography was done to the above patients and cardiologist opinion was obtained.

Observation & results

260 Patients with irreversible airway obstruction were recruited in the study subject to their written consent & following inclusion & exclusion criteria. The data so collected were analysed for statistical validity using software SPSS 16 (Statistical Package for the Social Sciences). The observations made were tabulated.

Table 1: Distribution of subjects according to various study parameters

S.No.	Parameter		No. Of patients	Percentage
1.	Sex	Male	160	61.53%
		Female	100	38.46%
2.	Age (in years)	40-50	32	12.30%
		50-60	72	27.69%
		60-70	89	34.23%
		>70	67	25.76%
3.	Body Mass Index (BMI (Kg/m^2)	Underweight (<18.50)	116	44.61%
		Normal (18.50-24.50)	142	54.61%
		Over weight (>24.50)	02	0.76%
4.	Life style	Sedentary	117	45%
		Non-Sedentary	143	55%
5.	Type Of Smoking	Bidi	183	70.38%
		Bidi/Hukkah	36	13.84%
		Hukkah	41	15.76%
6.	Smoking Index	<500	30	11.53%
		500-1000	167	64.23%
		≥1000	63	24.23%
7.	Chief Complaints	Dyspnoea	257	98.84%
		Cough	252	96.92%
		Expectoration	250	96.15%

Table 1 shows that 61.53% of the patients in our study were males and 38.46% were females.The mean age was 62.38 ± 11.03 years, range 40-85 years. The maximum incidence of COPD in this study is among the age group 50-69 years i.e. in the 5th&6th decade of life. No patients were less than 40 years, only 19 of the patients were ≥ 80 years.116 (44.61%) patient wereunderweight whereas BMI was in normal range in 142 (54.61%) patients.Only two (0.76%) patients were overweight.About 143 (55%) patients had non-sedentary life style whereas about 117 (45%) had sedentary life style.Bidi smoked by 183(70.38%) patients is the most common type of smoking followed by those smoking both bidi and Hukkah. The majority of cases 167(64.23%)were seen in 500-1000 smoking index followed by63 (24.23%) in ≥1000 smoking index and30 (11.53%) in <500 smoking index.

257 (99.84%) patients in this study had history of breathlessness at presentation, which is the most common symptom followed by cough in 252 (96.92%) and expectoration in 250 (96.15%) respectively. Most of the patients had multiple complaints.

Table 2:Distribution of subjects according torespiratory history, Spirometry findings and GOLD criteria

S.No.	Parameter		No. Of patients	Percentage (%)
1.	Severity of Disease	Mild	11	4.23
		Moderate	110	42.30
		Severe	111	42.69
		Very Severe	28	10.76
2.	mMRC Grade	Grade 1	111	42.69
		Grade 2	30	11.53
		Grade 3	119	45.76
3.	No. Of Exacerbations	0-1	127	48.84
		>1 - <2	129	49.61
		>3	04	01.53

4.	No. Of Hospitalizations	0	115	44.23
		>0	145	55.76
5.	GOLD criteria (A,B,C,D)	A	87	33.46
		B	28	10.76
		C	24	09.23
		D	121	46.53

From Table 2 it is apparent that maximum number of patients had moderate to severe airflow obstruction at the time of presentation and only 4.23% had mild disease. Majority of cases reportedmodified Medical Research Council (mMRC)grade3dyspnoea.One to twoexacerbations in last year occurred in majority of cases (49.61%) followed by 0-1 exacerbations 129 (48.84%) and >3 exacerbations occurred only in 04 (1.53%) cases in our study.115 (44.23%) patient had no history of hospitalization in past 2 years whereas as about 145 (55.76%) had history of hospitalization in past 2 years. It is clear that maximum number of patient 121 (46.53%) were in group D of ABCD criteria whereas group C had minimum number 24 (09.23%) patients.

Table3:Distribution of subject as per ECG and Echocardiographic findings.

S.No.	Parameter		No. Of patients	Percentage (%)
1.	ECG Changes	RAD	124	47.69%
		RVH	120	46.15%
		P-pulmonale	117	45%
		RBBB	06	2.30%
2.	2 D Echo Changes	RVD	127	48.84%
		RVH	83	31.92%
		RHF	00	00
		Cor-pulmonale	127	48.84%
3.	Pulmonary Hypertension	Present	133	51.15%
		Absent	127	48.84%
4.	Left Heart Changes	LVSD	00	00
		LVDD	127	48.84%

Table 3 shows 46.15% of the patients had ECG evidence of right ventricular hypertrophy in the study. Right Axis Deviation (RAD) was seen in 47.69% and is the most common ECG abnormality.45% of the patients in the study had p-pulmonale. RBBB was found only in 2.30%. 48.84% of the patients in this study had echocardiographic evidence of cor-pulmonale. 48.84% of the patients had right ventricular dilatation (RVD) and 31.92% patients had right ventricular hypertrophy (RVH). None of the patients had echocardiographic features of RV failure.The range of pulmonary artery pressure was from 15-55mmHg. Pulmonary hypertension (PH) was found in 133 (51.15%) of subjects.The mean ejection fraction among the subjects was 58.58 ± 3.62. None of the subject had left ventricular systolic dysfunction (LVSD) whereas left ventricular diastolic dysfunction (LVDD) was present in 127 (48.84%) patients.

Table 4: Correlation of ECG findings with severity of the disease

Severity	Mild (n=11)		Moderate (n=110)		Severe (n=111)		Very severe (n=28)	
	No.	%	No.	%	No.	%	No.	%
RAD	00	00	42	38.18	62	55.85	20	71.42
RVH	00	00	40	36.36	60	54.05	20	71.42
P-pulmonale	00	00	40	36.36	57	51.35	20	71.42
RBBB	00		00	00	00	03	2.70	03	10.71

Table 4 shows that in the mild category no patient had ECG changes whereas in the moderate category 38.18%of the patients had some ECG changes, 38.18%(42/110) of the patients had RAD, 36.36% (40/110) of the patients had ECG evidence of RVH, 36.36% (40/110) of the patients had 'p' pulmonale. In the severe category 55.85% of the patient had ECG changes, 55.85%   (62/111) of the patients had RAD, 54.05% (60/111) of the patients had ECG evidence of RVH, 51.35% (57/111) of the patients had 'p' pulmonale.In the very severe category 71.42% of the patient had ECG changes. 71.42% (20/28) of the patients had RAD, evidence of RVH and 'p' pulmonale, whereas only 3 patients had RBBB.

Table 5:Correlation of Echocardiography findings with severity of the disease.

Severity	Mild (n=11)		Moderate (n=110)		Severe (n=111)		Very severe (n=28)	
	No.	%	No.	%	No.	%	No.	%
RVD	00	00	45	40.90	62	55.85	20	71.42
RVH	00	00	30	27.27	40	36.03	13	46.42
RHF	00	00	00	00	00	00	00	00
Cor-pulmonale	00	00	45	40.90	62	55.85	20	71.42

Table 5 shows that in the mild group no patient had evidence of cor-pulmonale. In the moderate group 40.90% (i.e.45/110) had evidence of cor-pulmonale, 40.90% & 27.27% had RVD & RVH respectively.In the severe group,55.85% (i.e.62/111) had evidence of corpulmonale, 55.85% & 36.03% had RVD & RVH respectively.In the very severe group, 71.42% (i.e.62/111) had evidence of corpulmonale, 71.42% and 46.42% had RVD & RVH respectively.The echo signs of RV dilatation, RVH and cor-pulmonalecorrelate significantly with the severity of the disease (p<0.0001).

Table 6: Distribution of Pulmonary Hypertension according to the severity of COPD

Severity	Pulmonary hypertension	Percentage
MILD (n=11)	00	00
MODERATE (n=110)	49	44.54%
SEVERE (n=111)	63	56.75%
VERY SEVERE (n=28)	21	75%

As observed in Table 6, pulmonary hypertension among mild, moderate, severe and very severe COPD was 00%, 44.54%, 56.75% and 75%respectively(P <0.001). Thus, incidence of PH increases significantly with severity of disease.

Table 7: Comparison ofseverity of airway obstruction with Left Ventricular Diastolic Dysfunction (LVDD)

Severity	No. Of patients	Percentage
Mild	00	00
Moderate	38	34.54
Severe	65	58.55
Very severe	24	85.71
		P<0.001

LVDD was found in 127(48.84%) subjects of the study population. We studied the distribution of LVDD among subjects in relation to severity of airway obstruction. LVDD was noted in none of the patient with mild COPD, 38(34.54%) of moderate COPD, 65 (58.5%) of severe COPD and 24 (85.71%) of very severe COPD subjects. There was significant correlation between the severity of COPD and LVDD (P value <0.001).

Discussion

In this study the male to female ratio was 1.6:1, i.e. males formed 61.53 % (160/260) of the study subjects. Male predominance in COPD patients is an established fact and can be attributed to higher prevalence of smoking in them.[15] Maximum number of COPD patients (161/260) in this study were in the age group of 60-69 years with mean age 62.38 ± 11.03 years. Patients between 60-69 years form the maximum number of OPD & IPD patients mainly because of the longer duration of tobacco exposure and repeated respiratory tract infections, which would have compromised their quality of life.

In the present study, 53.46% (139/260) of the patients had FEV1 <50% of the predicted, i.e. majority had severe obstructive disease. The correlation between symptoms and severity of airway obstruction is an established fact. Thus, when the FEV1 fall to < 40%, patients start experiencing breathlessness on exertion, as per British Thoracic Society (BTS) and GOLD.[16]

In this study, 46.15 % (120/260) of the patients had ECG evidence of RVH, as per criteria used by Braunwald.[17] The incidence of RVH by ECG varies in different studies due to the use of different criteria for RVH by the different authors.[18,19,20,21,22,23,24,25] Our findings correlate with the findings of F.J.C. Millard (45.7%) and Murphy and Hutcheson (43.66%) and Gupta and Khastgir (50%).

Right axis deviation was present in 47.69% (124/260) of the patients in the present study. All the patients with RVH had RAD (100%). According to Murphy & Hutcheson,[26] right axis deviation is one of the most reliable criteria of RVH, and is more common in patients with RVH secondary to COPD than in those with RVH secondary to congenital heart disease and has a specificity of 95%. P pulmonale was evident in 45% of the cases. Thus, findings of p-pulmonale in this study are similar to Gupta and Khastgir[19] (43.3%) and Silver calatayud (46.2%).[27]

In the present study, only six (2.30%) patients had evidence of RBBB. This is similar to the other studies. Fowler et al [28] (1/15patients), Padmavathi& Raizada,[24] (4/544 patients) and Chappell (1966)[29] (2/122 patients). Milnor (1957) [30] is of the opinion that presence of RBBB is more commonly due to coronary disease than RVH but can also be found in persons without heart disease. The incidence of all the ECG findings, increased as the severity of the disease (as measured by FEV1 and graded according to GOLD criteria) increased. Statistical correlation, was found with right axis deviation, RVH, and 'P' pulmonale, (p < 0.0001).

Caird and Wilcken (1962) [25], observed that 'p' pulmonale and evidence of RVH are much more frequent when FEV1 falls below 45% of normal than above it. The likely explanation (given by them) is that a ventilatory capacity of < 45% of predicted is inadequate to maintain normal blood gases in the face of inequalities of ventilation and perfusion (V/Q ratios) and it has been repeatedly shown that pulmonary hypertension is correlated with degree of arterial oxygen desaturation and hypecapnea. M. K. Tandon[31] also found increasing incidence of p-pulmonale, right axis deviation of QRS and dominant S in V5/6 with increasing severity as defined by FEV1/FVC.

Spodick and Co-workers suggested that the electrocardiographic changes accompanying increasing airway obstruction and arterial blood gases in chronic bronchitis and emphysema are due to several mechanisms: Hyperinflation of lungs, Depression of the diaphragm, Hypoxia and changes in body chemistry and Pulmonary hypertension. 48.84% (127/260) of the patients in our study had echocardiographic evidence of cor-pulmonale, comprising of RV dilatation, RV hypertrophy, RA dilatation, RV failure, and/or interventricular septum motion abnormality. Findings in our study, correlates well with most of the findings in the study by Himelmann et al [32]. The incidence of all the echocardiographic findings increased as the severity of the disease increased, i.e. maximum incidence was found in the most very severely affected group of patients. All the findings had statistically significant correlation with severity of disease. ('P' <0.0001)

The range of pulmonary artery pressure was from 15mmHg to 55mmHg. None of the patients had mean pulmonary artery pressure (mPAP) more than 55mmHg. Pulmonary hypertension(PH) was found in 133 (51.15%) of subjects. In our study the distribution of pulmonary hypertension among mild, moderate, severe and very severe COPD were 00%, 44.54%, 56.75% and 75% respectively and the prevalence of pulmonary hypertension was 51.15%. N.K.Gupta et al [16] evaluated 40 COPD patients with echocardiography and found pulmonary hypertension among 63% (n=17). Among their 40 COPD subjects, 45% had mild COPD, while moderate, severe and very severe COPD were 27.5%, 12.5% and 15% [33] respectively. The pulmonary pressure in COPD patients had shown to increase 0.4 to 0.6mm Hg per year. This finding illustrates that pulmonary hypertension in COPD progress slowly[16]. A recent small cohort reported that the frequencies of PH in mild, moderate, severe, and very severe COPD were 16.67%, 54.55 %, 60.00 %, and 83.33 %, respectively[34].

Fayngersh V et al [35] studied the prevalence of PH in 105 stable COPD patient and found 60% (n=63) of patients with pulmonary hypertension. Mammosser et al[36] in their study on COPD patients to assess pulmonary hypertension enlightened that subjects with pulmonary hypertension had lower FEV1 than those without pulmonary hypertension. The study by Higham et al[37] in assessing the pulmonary artery pressure by echo found significant association between severity of COPD and pulmonary hypertension($r = -0.26$, $P = 0.05$). Our study results were in agreement with the above mentioned studies. The prevalence of pulmonary hypertension increases as the severity COPD increases.

LVDD was noted in none of the patients with mild COPD, 38 (34.54%) of moderate COPD, 65(58.5%) of severe COPD and 24(85.71%) of very severe COPD subjects. There was significant correlation between the severity of COPD and LVDD ('P' <0.001). Left ventricular diastolic dysfunction had been documented in COPD with varying prevalence. The right and left ventricle share a common interventricular septum (ventricular interdependence). The increase in LV end diastolic pressure due to bulging of septum into left ventricle and increased afterload due to increase in intrathoracic pressure were the proposed mechanism. A study by Cutticaet al[38] found 54% prevalence of LVDD in COPD patients. Abroug F et al [39] in their study found 32% of patients with LVDD. Caram et al [34] from their study with 25 mild/moderate COPD and 25 severe/very severe COPD patient found mild left diastolic dysfunction among 88% of patients irrespective of COPD severity.

Boussuges et al.[41] found a prevalence of 76% vs. 35% of LVDD in COPD patients compared to controls.Yilmaz et al. in 2005[42] has conducted a study on 44 subject that were divided into two group group 1: 24 (without PH) & Group 2: 20 (with PH). Patient with cardio vascular risk factors such as HT, heart disease, valvular heart disease were excluded. They concluded that left ventricular diasystolic dysfunction was present in despite normal LVEF.

Gupta et al., in 2011[16] has conducted a study on 40 subjectand categorized them as per GOLD criteria as mild, moderate, severe and very severe. 7.5% of cases were found to have LVSD, whereas LVDD & LVH was present in 47.5% & 22.5% of cases respectively. Thus on the basis of their findings they concluded that prevalence of LVDD increases with the severity of disease of COPD (with the reduction in FEV1).

In our study all patients have preserved LVEF and prevalence of LVDD was still 48.84% in COPD patient which increases as the severity of disease increases. Thus, our findings correlate with Gupta et. al[16] and Yilmaz et. al. [42]

In this study the diagnosis of cor-pulmonale could be made in 46.15% (120/260) patients by electrocardiographic method and 48.84% (127/260) by echocardiographic method. The difficulty in interpreting the ECG criteria for RVH in COPD is due to the ECG changes seen can be the result of positional changes in the heart as a result of severe emphysema rather than RVH per se. The degree of RVH seen in COPD, is usually lower, as compared to that seen in congenital heart disease, for which most of the criteria were initially proposed.

Many studies have proved that echocardiography is more sensitive than electrocardiography in detecting RV dysfunction in COPD. Prakash R.[43]in his study found that ECG had a sensitivity of 31% and Specificity of 85% and ECHO had a sensitivity of 93% and specificity of 95% in detecting RV dysfunction. Putnik[44]in his study found that diagnosis of cor-pulmonale by ECG could be made in 78% and by ECHO in 95% of the patients. Oswald mammoser[45]found that sensitivity and specificity for diagnosis of pulmonary hypertension in patients with COPD by ECG was 51% and 86% respectively and by Echo was 78% and 78% respectively.Himelmann[32]in his study found that cor-pulmonale could be diagnosed in 39% of the patients by clinical methods and by echocardiography in 75%. This clearly shows that echocardiography is better than ECG or clinical methods, to detect the presence of corpulmonale in patients with COPD.

Conclusion

It can be safely concluded from our study that COPD is a fairly common disease among the patients reporting to Respiratory Medicine OPD, especially in males in 5th and 6th decade of life. It is important to assess a patient of COPD not only with clinical, radiological, spirometric criteria but also with ECG & Echocardiography in order to assess the severity and outcome of disease.

References

1. Global Initiative for Chronic Obstructive Lung Disease – Global Strategy forDiagnosis, Management, and Prevention of Chronic Obstructive Pulmonarydisease. http.//www.goldcopd.com
2. Emmanuel Weitzenblum. Chronic corpulmonale. Heart 2003 ; 89 : 225-230.
3. Divo M, Cote C, de Torres JP, et al. Comorbidities and risk of mortality in patients with chronic obstructive pulmonary disease. Am J RespirCrit Care Med 2012; 186: 155–161.
4. Smith MC, Wrobel JP. Epidemiology and clinical impact of major comorbidities in patients with COPD. Int J Chron Obstruct Pulmon Dis 2014; 9: 871–888.
5. Bhatt SP, Dransfield MT. AECOPD: acute exacerbations of chronic obstructive cardiopulmonary disease? Am J RespirCrit Care Med 2013; 188: 1046–1048.
6. Miller J, Edwards LD, Agusti A, et al. Comorbidity, systemic inflammation and outcomes in the ECLIPSE cohort. Respir Med 2013; 107: 1376–1384.
7. Mannino DM, Thorn D, Swensen A, et al. Prevalence and outcomes of diabetes, hypertension and cardiovascular disease in COPD. EurRespir J 2008; 32: 962–969.
8. Westerik JA, Metting EI, van Boven JF, et al. Associations between chronic comorbidity and exacerbation risk in primary care patients with COPD. Respir Res 2017; 18: 31.
9. Lahousse L, Tiemeier H, Ikram MA, et al. Chronic obstructive pulmonary disease and cerebrovascular disease: a comprehensive review. Respir Med 2015; 109: 1371–1380.
10. Oswald-Mammosser M, Apprill M, Bachez P, et al. Pulmonary hemodynamics in chronic obstructive pulmonary disease of the emphysematous type. Respiration 1991;58:304 –310
11. Saito H, Dambara T, Aiba M, et al. Evaluation of corpulmonale on a modified short-axis section of the heart by magnetic resonance imaging. Am Rev Respir Dis (1992);146:1576–1581.
12. M. Oswald-Mammosser, E. Weitzenblum, E. Quoix, et al,Prognostic factors in COPD patients receiving long-term oxygen therapy. Importance of pulmonary artery pressure, Chest 107 (1995) 1193–1198.
13. Incalzi RA, Fuso L, De Rosa M, et al. Electrocardiographic signs of chronic corpulmonale: a negative prognostic finding in chronic obstructive pulmonary disease. Circulation 1999;99:1600–1605

14. Cardiac Disease in Chronic Obstructive Pulmonary Disease Jeremy A. Falk1, Steven Kadiev2, Gerard J. Criner3, Steven M. Scharf4, Omar A. Minai5, and Philip Diaz

15. Badaran E, Ortega E, Bujalance C, Puerto LD, Torres M, Riesco JA. Smoking and COPD exacerbations. European Respiratory Journal 2012;40 (56):P1055

16. N.K.Gupta,Ritesh Kumar Agrawal,A.B.Srivastav,M.L.Ved. Echo evaluation of heart in copd patient and its co-relation with the severity of disease.Lung India.2011 apr-jun;28(2):105-109.

17. Vallerie V. McLaughlin, Stuart Rich. Cor-pulmonale. Chapter 54. Heart Disease – A text book of Cardiovascular Medicine. Eugene Braunwald, 6th edition, W. B. Saunders Company, Philadelphia. 2001 : 1936-1954

18. Ralph C. Scott, Samuel Kaplan, M.O.Fowler, Robert A Helm. Richard N Westscott, Isom C. Walker, William J. Stiles. The electrocardiographic pattern of right ventricular hypertrophy in chronic corpulmonale. Circulation 1955 ; X1 : 927-936.

19. R. W. Phillips. Providence R I. The electrocardiogram in Corpulmonale secondary to pulmonary emphysema. A study of 18 cases proved by autopsy.American Heart Journal, 1958 ; 56(3) : 352-371.

20. Padmavathi S, Pathak SN. Chronic cor-pulmonale in Delhi. A study of 127 cases.Circulation1959 ; 20 : 343-352.

21. Agarwal SK, Heiss G, Barr RG, Chang PP, Loehr LR, Chambless LE, Shahar E, Kitzman DW, Rosamond WD. Airflow obstruction, lung function, and risk of incident heart failure: the Atherosclerosis Risk in Communities (ARIC) study. Eur J Heart Fail 2012; 14:414–422.

22. FJC. Millard. The electrocardiogram in chronic lung disease. British heart journal1967; 29 : 43-50..

23. Gupta S, Khastgir T, Gupta MP, Sethi KK, Manoharan S. Clinical, Haemodynamic and Echocardiographic study in chronic corpulmonale. JAPI 1989 ; 37(6) : 373-376..

24. S. Padmavathi and VeenaRaizada. Electrocardiogram in chronic corpulmonale. British Heart Journal 1972 ; 34 : 658-667.

25. Francis I. Caird, David E.L., Wilcken. The electrocardiogram in chronicbronchitis with generalized obstructive lung disease. Its relations to ventilator function. The American Journal of Cardiology, 1962 ; 10 : 5-11.

26. Marvin L. Murphy, Fred Hutcheson. The electrocardiographic diagnosis of rightventricular hypertrophy in chronic obstructive pulmonary disease. Chest. 1974 ;65 : 622-627.

27. Harold M Silver, Juan B. Calatayud. Evaluation of QRS criteria is patients with COPD. Chest 1971 ; 59(2) : 153-159.

28. Noble O. Fowler, Clarke Daniels, Ralph Scott, Benigno SF, MoscheGueron. Theelectrocardiogram in corpulmonale with and without emphysema. The American Journal of Cardiology, 1965 ; 16 : 500-505.

29. A. G. Chappel. The electrocardiogram in chronic bronchitis and emphysema. Bri. Heart. J. 1966 ; 28 : 517-522.

30. W. R. Milnor. Electrocardiogram and Vectorocardiogram in right ventricularhypertrophy and right bundle branch block. Circulation 1957 ; XVI. : 348-367.

31. Tandon MK. Correlation of electrocardiographic features with airway obstruction in chronic bronchitis. Chest 63 (2) : 146-148.

32. Himelmann RB, Struve SN, Brown JK, Namnum P, Schiller NB. Improvedrecognition of corpulmonale in patients with severe chronic obstructive pulmonary disease. Am J. Med. 1988 ; 84 : 891-898.

33. Chaouat A, Naeije R, Weitzenblum E. Pulmonary hypertension in COPD. EurRespir J 2008; 32: 1371.

34. W. Matsuyama, R. Ohkubo, K. Michizono, et al. Usefulness of transcutaneous jugular venous Doppler in COPD patients. EurRespir J 2001; 17: 1128 -1131.

35. Fayngersh V, Drakopanagiotakis F, Dennis McCool F, Klinger JR. Pulmonary Hypertension in a Stable Community-Based COPD Population. Lung 2011;189:377-82.

36. M. Oswald-Mammosser, E. Weitzenblum, E. Quoix, et al,Prognostic factors in COPD patients receiving long-term oxygen therapy. Importance of pulmonary artery pressure, Chest 107 (1995) 1193–1198.

37. M. A. Higham, D. Dawson, J. Joshi, P. NihoyannoPaulos, N W Morell. Utility of echocardiography in assessment of pulmonary hypertension secondary to COPD. Eur. Respir. J. 2001; 17 : 350-355.

38. Cuttica MJ, Shah SJ, Rosenberg SR, Orr R, Beussink L, Dematte JE, Smith LJ,Kalhan R: Right heart structural changes Are independently associated with exercise capacity in Non-severe COPD. PLoS ONE 2011, 6:e29069.

39. Abroug F, Ouanes-Besbes L, Nciri N, et al. Association of left-heart dysfunction with severe exacerbation of chronic obstructive pulmonary disease: diagnostic performance of cardiac biomarkers. Am J RespirCrit Care Med. 2006;174(9):990-996.

40. Caram LM, Ferrari R, Naves CR, Tanni SE, Coelho LS, Zanati SG, et al Association between left ventricular diastolic dysfunction and severity of chronic obstructive pulmonary disease. Clinics (Sao Paulo). 2013;68:772–6.

41. 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 RespirCrit Care Med. 2000;162:670–5.

42. Yilmaz R, Gencer M, Ceylan E, Demirbag R. Impact of chronic obstructive pulmonary disease with pulmonary hypertension on both left ventricular systolic and diastolic performance. J Am SocEchocardiogr. 2005;18:873–81.

43. Prakash R. Echocardiographic diagnosis of right ventricular hypertrophy : correlation with ECG and Necropsy findings in 248 patients. Cathetcardiovasc. diagn1981 ; 7(2): 179-84.

44. Putnik M, Povazan D, Vindisjesic M. Electrocardiography and echocardiographyin the diagnosis of chronic corpulmonale (Article in Serbo Croatian (Roman)). Med Pregl, 1998 ; 51(11) : 528-31.

45. Oswald Mammosser M, Oswald T, Nyankiye E, DickeleMcCrange D, Weitzenblum E. Non invasive diagnosis of pulmonary hypertension in chronic obstructive pulmonary disease Comparison of ECG, radiological measurements, echocardiography and myocardial scintigraphy. Eur. J. Respir Dis. 1987 ; 71(5) : 419-29.