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Original article

Degree of Pulmonary Hypertension in Patients of COPD Attending a Tertiary Care Hospital

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Abstract

Introduction: Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality in India. The development of pulmonary hypertension (PH) in COPD adversely affects survival and exercise capacity and is associated with an increased risk of severe acute exacerbation. ***Aims and Objectives:*** Present study aimed to describe the distribution of echocardiographically assessed pulmonary artery systolic pressure in the COPD patients attending pulmonary medicine OPD, to estimate the proportion of PH among such patients and to identify associated factors. ***Methodology:*** This Cross sectional study was done on 145 patients of COPD fulfilling the inclusion criteria were included in this study. These patients were evaluated for the presence of PH using chest X-ray, 2D-Echocrdiography and electrocardiogram. ***Results:*** Pulmonary hypertension was present in 17 (29.8%) patients with moderate, 36 (60%) with severe and 18 (72%) with very severe COPD. Mild PH was seen in 52(73%), moderate in 11(15.4%) and severe in 8(11.2%) patients. Patient exposed to both smoking and biomass fuel smoke had more frequent occurrence of PH (56.8%) as compared to exposure to single risk factor of either tobacco smoke (47.1%) or biomass fuel (52.4%). Thus overall proportion of PH among reported cases of COPD patients was 48.6% in our study. ***Conclusion:*** With the increase in severity of COPD the proportion of PH rises. This study emphasizes the early detection of COPD and the importance of screening for PH through clinical assessment and non- invasive techniques 2-D Echocardiography.

Keywords: ***Chronic obstructive pulmonary disease, pulmonary hypertension, echocardiography.***

Introduction

COPD is the fourth leading cause of death and is projected to be the 3rd leading cause by 2020 [1]. The prevalence of PH in stable COPD patients varies from 10-30% [2]. PH is defined as a mean pulmonary artery pressure of ≥ 25 mmHg at rest [3,4]. The ideal method to diagnose PH is right heart catherization. However in the absence of this facility, transthoracic Doppler echocardiography is a non- invasive screening test for detection of PH which fairly correlates the catherization findings.

The development of PH in COPD adversely affects survival and exercise capacity and is associated with an increased risk of severe acute exacerbation; therefore its early detection is mandatory to prevent heart failure [5].

Aims and Objectives

This study aimed to describe the distribution of echocardiographically assessed pulmonary artery systolic pressure in the COPD patients attending pulmonary medicine OPD, to estimate the proportion of PH among such patients and to identify associated factors.

Methodology

145 patients of COPD proven by spirometry as per latest GOLD Guidelines [6] attending the outpatient department of pulmonary medicine, were evaluated for presence of PH using chest X-ray, 2D-Echocardiography and electrocardiogram.

We included patients with age more than 40 years and with history of smoking with pack years of more than 20 and /or exposure to biomass fuel smoke. A significant biomass exposure was defined as a subject with more than 80 hours/year with a minimum exposure of 10 years. For patients with dual exposure of biomass and smoking, biomass fuel smoke exposure of ≥80 hours/years with a minimum exposure of 10 years with a pack years of ≥20 is considered significant dual exposure.

We excluded patients below 40 years of age, patients with other pulmonary diseases like tuberculosis, bronchiectasis, interstitial pulmonary disease and bronchial asthma, patients with unstable cardio respiratory status defined as occurrence of respiratory failure, broncho pulmonary infection or congestive cardiac failure in previous two months, structural diseases of heart e.g. valvular heart disease, congenital heart disease and cardiomyopathy, ischemic heart disease like typical angina pectoris

or prior myocardial infarction,patients with primary pulmonary hypertension and hepatic and renal insufficiency.

The parameters noted on echocardiography with the use of 2-D, Color Doppler and M mode were size of right atrium and ventricle, Grade of PH, presence or absence of tricuspid regurgitation signifying severity of PH and pulmonary artery pressure estimation

Pulmonary artery pressure was estimated based on peak tricuspid regurgitation velocity (TRV) and right atrial pressure (RAP) as per Bernoulli Equation

Pulmonary artery pressure = Right Ventricular Systolic Pressure (RVSP)

$RVSP=4v^2+RAP$

V = tricuspid regurgitant velocity, RAP = Right atrial pressure

Right atrial pressure was estimated based on the diameter and respiratory variation in diameter of the inferior vena cava (IVC) [7]. A normal RA pressure of 3mmHg (normal range of 0-5mmHg) is when IVC diameter is <2.1cm that collapses >50%.High RA pressure of 15mmHg (normal range 10-20 mmHg) is suggested when IVC is <20% collapsible. An intermediate value of 8mmHg is used when IVC diameter and collapsibility does not fit the above mentioned values.

Echocardiographic probability of pulmonary hypertension in symptomatic patients as per Lang RM et al is shown in table 1 [8].

Table: 1

Peak tricuspid regurgitation velocity (m/s)	Presence of other echo PH signs	Echocardiographic probability of pulmonary hypertension
2.9-3.4 >3.4	Yes Not required	High
≤2.8 or not measurable 2.9-3.4	Yes No	Intermediate
≤2.8 or not measurable	No	Low

Grading of PH was done according to American heart association guidelines [9]. 36-50mmHg was considered as mild, 51-60 mmHg was considered as moderate and >60 mmHg was considered as severe

Results

Out of 145 subjects included in our study, the variables which were observed are listed in table 2

Table 2

Variables studied	**No of cases n**	**%**
Male	76	53%
Female	69	47%
Age group 40-55yrs	22	15%
Age group 56-70 yrs	77	53%
Age group 70 yrs	46	32%
Tobacco smokers	104	72%
Biomass fuel smoke exposure	42	28%
Dual exposure to tobacco and biomass smoke	37	25%

Table 3: The association between pulmonary hypertension and severity of COPD

Severity of COPD	**Presence of PH(n)**	**Total number of cases (N)**	**%**	**'p 'value**
Mild COPD	0	3	0%	0.078
Moderate COPD	17	57	29.8%	<0.001
Severe COPD	36	60	60%	0.022
Very severe COPD	18	25	72%	0.017

It was observed that as severity of COPD increased the proportion of cases with pulmonary hypertension also increased. These findings were statistically significant as shown in table 3 (p value <0.05).

Table 4: The association between grades of COPD and severity of pulmonary hypertension

Severity of COPD	**Mild PH n / total no of COPD cases (n%)**	**Moderate PH n /total no of cases (n%)**	**Severe PH n /total no of cases (n%)**	**'p' value**
Moderate COPD	14 / 57 (24%)	2 / 57 (3%)	1 / 57 (1.7%)	<0.001
Severe COPD	31 / 60 (51.6%)	1 / 60 (1.6%)	4 /60 (6%)	
Very Severe COPD	7 / 25 (28%)	8 / 25 (32%)	3 / 25 (12%)	
Total no of cases of PH	52	11	8	

Most of the moderate to severe COPD cases had mild degree of PH, whereas most of the patients with very severe COPD had moderate degree of PH. It was thus observed that severity of pulmonary hypertension increased with increasing severity of COPD, which was statistically significant (<p value 0.001) as shown in table 4.

Table 5: The Association between risk factors and pulmonary hypertension

Risk factors	PH present (n)	Total number of cases (N)	%	'P' value
Biomass fuel smoke exposure	22	42	52.4%	0.564
Tobacco Smoker	49	104	47.1%	0.564
Combined Smokers and biomass fuel smoke exposure	21	37	56.8%	0.252

It was observed that patients having dual exposure to smoking and biomass fuel had more incidence of pulmonary hypertension (56.8%) as compared to single risk factor of either biomass fuel (52.4%) or tobacco smoking (47.1%) alone. However these results were statistically not significant.

Discussion

Pulmonary Hypertension (PH) is defined as an increase in mean pulmonary arterial pressure (PAPm) is ≥25 mmHg at rest assessed by right heart catherization (RHC) [2]. PH in COPD affects survival and exercise capacity and is associated with increased risk of acute exacerbation. The pulmonary pressure increases during exercise, REM sleep and exacerbations which, eventually leads to right heart failure

In present study, out of 145 patients, 3 (0.2%) had mild, 57 (39%) had moderate, 60 (41.3%) had severe and 25 (17%) had very severe COPD. Pulmonary hypertension was present in 17(29.8%) patients with moderate, 36(60%) with severe and 18(72%) with very severe COPD patients. Pulmonary hypertension was not seen in our patients with mild COPD. Thus overall prevalence of PH among reported cases of COPD patients in our study was 48.6%. These results were statistically significant (p value <0.05). A similar study done by Shruthi Reddy et al[10] found that the prevalence of PH among COPD patient was 50%. In a study by Gupta NK et al[11], the prevalence of PH in mild, moderate and very severe COPD were 16.67%, 54.55%, 60% and 83.33%. Approximately 25% patients with COPD eventually develop cor pulmonale [12].

Out of 71 total cases of PH, we had 52(73%) cases with mild,11(15.4%) with moderate and 8 (11.2%)with severe PH. A similar study done observed mild PH in 50.2%, moderate in 9.8%, severe in 3.7% cases of COPD [13]. Hyperinflation in COPD, compresses the alveolar vessels resulting in pulmonary vasoconstriction, destruction of vascular bed, vascular remodeling and polycythemia thus increasing the pulmonary vascular resistance, ultimately resulting in the development of PH [14]. Thus the incidence of PH is directly proportional to severity of airflow limitation.

As per the smoking status our keen observation was that patients exposed to both smoking and biomass fuel smoke had more frequent occurrence of PH (56.8%) as compared to exposure to single risk factor of either tobacco smoke (47.1%) or biomass fuel (52.4%). However these results were statistically not significant. Biomass is composed of mixture of gases and particles that penetrate deeply into the lung and cause morphologic and biochemical changes and are risk factors for COPD [15,16]. In a study by Rivera et al vascular changes were prominent in both groups, but more severe in the biomass exposed group which could explain why pulmonary hypertension is seen more in biomass exposed group [17]. The pathophysiology of the development of PH in COPD patients is due to chronic exposure of airways to noxious particles like tobacco smoke and biomass fuel smoke. Hence combined exposure to smoking and biomass increases the risk of PH. A similar study done by bunyamin et al showed that frequency of PH is higher in patients exposed to biomass fuel than tobacco smoke [18].

Conclusion

Pulmonary Hypertension is one of the most common complication of COPD. COPD associated PH is recognized as a poor prognostic marker. It is associated with increased risk of exacerbation, worsened exercise capacity, poor oxygenation and increased mortality. As severity of COPD increases the proportion of cases with increased severity of PH increases. Severe PH increases the right ventricular afterload and eventually leads to heart failure. This study emphasizes the early screening of patients of COPD for PH through clinical assessment and non invasive testing like 2-D Echocardiography.

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Conflict of interest

None

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References

[1] Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age group in 1990 and 2010; a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*.2012; 380(9859): 2095-128.

[2] Naeije R, MacNee W, Calverley P, et al. Chronic obstructive pulmonary disease,2nd edition. London. *Arnold Heath Sciences*. 2003:228-242.

[3] Galie N, Hoeper MM, Humbert M, et al. Guidelines for the diagnosis and treatment of pulmonary hypertension: the Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). *Eur Respir J*. 2009; 34(6):1219–63.

[4] McLaughlin VV, Archer SL, Badesch DB, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension" A report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians, American Thoracic Society and the Pulmonary Hypertension Association. *J Am Coll Cardiol*. 2009; 53(17):1573–619.

[5] Thomas L Petty. The history of COPD. *Int J COPD*. 2006;1(1) :3-14.

[6] Global initiative for Chronic Obstructive Lung Disease Strategy for the Diagnosis, Management and Prevention of Chronic Obstructive Pulmonary Disease Updated 2019, available online at www.goldcopd.org.com

[7] Fisher MR,Forfia PR, Chamera E, et al. Accuracy of Doppler Echocardiography in the hemodynamic assessment of pulmonary hypertension. *Am J Respir Crit Care Med*.2009; 179(7):615-621.

[8] Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015;16(3):233-271.

[9] 2015 ERS/ERS Guidelines for the Diagnosis and treatment of Pulmonary Hyperetension.*Eur heart J*.2015.

[10] Shruthi Reddy, Rajender and Nihin Reddy. Prevalence of Pulmonary Hypertension in COPD patients; A retrospective observational study. *Int journal of integrative Medical Science*.2016; 3(5):285-2.

[11] Gupta NK,Agrawal RK, Srivastav AB et al. Echocardiographic evaluation of heart in chronic obstructive pulmonary disease patient and its correlation with severity of disease. *Lung India*.2011; 28:105-9.

[12] J.L. Wright, L. Lawson, P.D. Pare et al. The structure and function of the pulmonary vasculature in mild COPD. The effect of oxygen and exercise. *The American Review of Respiratory disease*. 1983;128(4) :702-707.

[13] Thabut G, Dauriat G,Stern JB et al. Pulmonary hemodynamics in advanced COPD candidates for lung volume reduction surgery or lung transplantation. *Chest*. 2005;127(5):1531-1536.

[14] Fishman AP.State of the art: chronic cor pulmonale. *Am Rev Respir Dis*. 1976; 114(4):775-94.

[15] Zelikoff JT, Chin Chen L, Cohen MD et al. The toxicology of inhaled wood smoke. *J toxicology Environment Health B crit Rev*.2002; 5(3):269-282.

[16] Hu G, Zhou Y, Tian J et al. Risk of COPD from exposure to biomass smoke: A metaanalysis.*Chest*.2010;138(1):20-31.

[17] Rivera RM, Cosio MG, Ghezzo H et al. Comparison of lung morphology secondary to cigarette and biomass smoke. *Int J Tuberc* Lung Dis. 2008;12(8):972-7.

[18] Medhat Soliman, Hussen Heshmat, Yousif Amen et al. Detection of right sided heart changes and pulmonary hypertension in COPD patients. *Egyptian Journal of Chest diseases and Tuberculosis*.2015;64(2): 335-341.