FREQUENCY OF PULMONARY HYPERTENSION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

Amir Suleman, Azmat Abdullah, Raza Ullah, Hamid Nisar, Zafar Iqbal, Shahida Naz, Saeed Maqsood

Department of Pulmonology, Pakistan

Address for correspondence:
Amir Suleman
Department of Pulmonology, Pakistan

E-mail:
amirsuleman2013@gmail.com

ABSTRACT

Objective: Chronic obstructive pulmonary disease (COPD) is characterized by progressive, irreversible airflow limitation and chronic inflammation caused by inhalation of cigarette smoke or other noxious particles. It is associated with a number of morbidities and complications. This study was conducted to determine the frequency of pulmonary hypertension in COPD.

Materials Methods This was descriptive, cross sectional study was carried out in the Department of Pulmonology, Ayub Teaching Hospital, Abbottabad - Pakistan over six months duration, from July to December, 2015.

Methodology: Diagnosed cases of COPD of either gender and aged 35 years and above were included in the study through consecutive, non-probability sampling technique. Patients with asthma, pulmonary hypertension, ischemic heart disease, collagen vascular disease were excluded from the study. The sample size was calculated using the WHO software for sample size, assuming 50%[1]proportion of pulmonary hypertension in COPD patients, 95% confidence interval and 7% margin of error.

Results: A total of 196 patients were enrolled in the study. 151(77%) were male and 45(23%) were female. Patients were divided in age groups as; 32(16.3%) patients were in age 35-50 years, 39(19.9%) patients in 51-60 years, 62(31.6%) patients in 61-70 years and 63(32.1%) patients were above 70 years. Mean age was 64.96 years. 43.4% of patients have Moderate COPD (FEV1:50%-69%), 30.6% have severe COPD (FEV1:30%-49%) and 26 % have very severe COPD (FEV1< 30%).PHT was found in 45.4% of COPD patients.

Conclusion: Patients with COPD had a significant prevalence of pulmonary hypertension, and failure to address this may lead to complications.

Key Words: COPD, Pulmonary hypertension, FEV1

This article may be cited as: Suleman A, Abdullah A, Ullah R, Nisar H, Iqbal Z, Naz S, Maqsood S. Frequency of pulmonary hypertension in chronic obstructive pulmonary disease patients. Pak J Chest Med 2017; 23 (4):151-5.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a disease that is characterized by progressive, irreversible airflow limitation and chronic inflammation.[2] Spirometry is the gold standard diagnostic test to confirm fixed airflow limitation.[3] COPD is a prevalent condition associated with high mortality, morbidity. It reduces quality of life and is caused by chronic inflammation due to inhalation of cigarette smoke or other noxious particles which in turn lead to remodeling of the small airways or destruction of the lung parenchyma.[4]COPD is one of the leading health burdensWorldwide, resulting in 3.0 million deaths annually. According to World Health Organization (WHO) statistics, 210 million people are suffering from COPD.[5] COPD is the 3rd leading cause of death in the world. The prevalence of COPD varies from 8% to

20% in the world.[6]

A number of morbidities and complications are associated with COPD. The presence of these complications represents a poor prognosis for patients with the COPD unless treated and managed in time. Pulmonary hypertension is one of important complication of COPD.[7] It is defined asmean pulmonary arterial pressure more than 25 mm Hg. The prevalence of pulmonary hypertension is high among COPD patients.[8] Pulmonary hypertension is usually mild to moderate. But, in 5–10% of patients with end stage COPD have severe pulmonary hypertension and at the end they develop right heart failure. Pulmonary hypertension (PH) secondary to chronic obstructive pulmonary disease (COPD) is placed in group 3 in WHO classification.[9]

Pulmonary hypertension in COPD is associated with an increased risk of acute exacerbations and thus increased mortality and morbidity. Depending on the Severity of COPD and the method of measuring the pulmonary artery pressure (echocardiography versus right heart catheterization), the prevalence of PH in stable COPD varies from 20 to 91%.[10] In another study prevalence of pulmonary hypertension in patient with severe COPD was 34%.[11] The prevalence of PH secondary to COPD also depends upon the etiology of COPD. In a retrospective study prevalence of COPD cases secondary to Biomass smoke was (59.1%) and that due to tobacco smoke it was (50%).[12] Pulmonary hypertension in COPD is assumed to be due to hypoxic pulmonary vasoconstriction leading to permanent medial hypertrophy.

The aim of this study is to find out the frequency of pulmonary hypertension in patients presenting with COPD in our population. The results of this study will provide an objective evidence of secondary pulmonary hypertension frequency and will highlight the need for preventive strategies.

MATERIAL AND METHODS

OPERATIONAL DEFINITIONS: Pulmonary hypertension is defined as mean pulmonary arterial pressure greater than 25mm Hg measured by Doppler echocardiography.

COPD: Post bronchodilator Spirometry showing FEV1 <80% predicted or FEV1/FVC ratio <70%.

This cross sectional study was carried out in pulmonology department (wards/OPDs) of Ayub Teaching Hospital Abbottabad over six months duration from 01-07-2015 to 31-12-2015. A total of 196 patients were included through consecutive, non-probability sampling technique. Sample size was calculated by WHO sample size calculator assuming 50%[6]proportions of pulmonary hypertension, 95% confidence interval and 7% margin of error. Diagnosed cases of COPD of either gender and aged 35 years and above were included in the study. Patients with asthma, pulmonary hypertension, ischemic heart disease, collagen vascular disease were excluded.

All new cases with COPD presenting to pulmonology OPD/wards were enrolled in study. The purpose and benefits of the study was explained to the patients and informed written consent was taken from patients. Detailed history and clinical examination followed by relevant investigation was done. Doppler echocardiography of all patientswas done by an expert cardiologist. All the information was recorded on a proforma. Exclusion criteria were followed to control confounders and bias in the study results.

Statistical analyses were carried out using SPSS-15. Quantitative variables like age weredescribed as Mean±Standard deviation. Frequencies and percentages were calculated for categorical variables like gender, pulmonary hypertension.

RESULTS

A total of 196 patients of COPD were inducted in the study. 151(77%) were male and 45(23%) were female. Age distribution among 196 patients was analyzed as: 32(16.3%) patients were in age range 35-50 years, 39(19.9%) patients were in age range 51-60 years, 62(31.6%) patients were in age range 61-70 years and 63(32.1%) patients were above 70 years.Mean age of the COPD patients was 64.96(±10.6SD)years.

Pulmonary hypertension was found in 89 (45.4%) of patients in our study while rest of 107 (54.6%) didn't have pulmonary hypertension. Among patients with PHT, 34.1% were having moderately severe COPD, 45% with severe COPD and 64.7% were having very severe COPD.

In our study, the risk factors associated with second-

Table 1: Gender distribution among COPD subjects

	Frequency	Percent
Male	151	77.00%
Female	45	23.00%
Total	196	100.0%

Table 2: Descriptive Statistics of Age of COPD patients

Age in years	Frequency	Percent
35-50	32	16.3%
51-60	39	19.9%
61-70	62	31.6%
Above 70 years	63	32.1%
Total	196	100.0%

Table 3: PHT in COPD patient

	Frequency	Percent
Yes	89	45.4
No	107	54.6
Total	196	100.0

ary pulmonary hypertension in COPD patients were severity of the disease at presentation.

DISCUSSION

COPD is expected to increase globally in the coming decades. The main reasons are the changing age distribution in all countries, with increased life expectancy and an ever increasing proportion of the population living to >60 years.[13] Mean age in our study 64.96 %(±10.6 SD). COPD is a disease of elderly and one contributor is annual decline in FEV1 that usually starts after 30 years of age.

Smoking is common and major risk factor for COPD.[14] Males are more at risk for developing COPD than females.[15] These findings are consistent with our study where majority (77%) COPD patients were male. Although this trend is changing in developed world where female's tendency towards cigarette smoking and subsequent development of COPD is increasing, cigarette smoking in developing countries like Pakistan is still low in females and that's the reason males COPD patients predominate.

Factors that portend a poor prognosis include severity of airflow limitation, hypercapnea, and pulmonary hypertension.[16] Secondary pulmonary hypertension in COPD patients is a poor prognostic factor.[17] Pulmonary Hypertension Typicallyappears when airflow limitation is severe and is associated with chronic hypoxemia, the main pathophysiological cause being chronic alveolar hypoxia.[18]

The prevalence of pulmonary hypertension (PH) in COPD has not been accurately measured in large epidemiologic studies because of the risks and expense of invasive pressure measurement by right heart catheterization.Estimates of the prevalence of PH in patients with COPD vary widely based upon the definition of PH, the methods used to determine pulmonary pressures, and the physiologic characteristics of the studied population.In international studies the prevalence of PHT in COPD was different in different studies ranging from20 to 50%.[19] In our study pulmonary hypertension was found to be 45.4% in COPD patients. This is in contrast to a study Sertogullarindan B et al.where pulmonary hypertension was found to be 34% in COPD patients.[20] This lower frequency may be due to difference in risk factor for COPD, study population age and gender. This can be explained by the findings in study by Ongel EA et al.[6] that demonstrates that COPD due to cigarette smoking has high occurrence of PH (50%) than COPD due to other risk factors like biomass burn exposure (59.1%). This also explains the variation in findings of PH in COPD patients among gender as males are more commonly smokers than females. Increasing age is associated with increased frequency and severity of PH.

There is direct relationship between severity of airway obstruction detected by FEV1 and PHT. In our study PHT was 34.1%, 45% and 64.7%in moderate, severe and very severe COPD respectively.This finding is demonstrated in study by Chaouatet al.[18] where it is found that prevalence of pulmonary hypertension increases in patients with increasing COPD severity. Similar increased prevalence of PH was demonstrated by Scharf SM et al.[21] in his study where PH was found to be present in more than 90% of patients although the criteria for PH diagnosis was mPAP>20mmhg with mean value of 26.3 ± 5.2 mmHg. In Animal models of smoke-induced emphysema and Pulmonary Hypertension suggest that PH is due to hyperinflation and gas trapping that compress the pulmonary vessels.[22] It means that PHT is more common in severe and very severe COPD patients.

CONCLUSION

Most complications of COPD are associated with the severity of the COPD. To prevent these complications, all risk factors that accelerate the decline in FEV1 and severity of COPD should be sought and modified, if

possible. Failure to recognize and address these factors may lead to unnecessary escalation of the therapy and avoidable hospitalizations and complications.

Our study shows that a higher proportion of PHT is present in COPD patients especially in severe COPD. Effective smoking cessation counseling needs to be ensured from the very first encounter with COPD patient.Keeping in mind the impact on health and health care resources, it is essential to develop effective strategies for prevention of COPD complications.

LIMITATIONS

Our study has certain limitations that need to be overcome in the future.

We used Doppler echocardiography to diagnose PHT in COPD patients. Doppler echocardiography is not a very good modality to diagnose PHT.

Sample size was small. Study population was male predominant.

REFERENCES

1. Andersen KH, Iversen M, Kjaergaard J, Mortensen J, Nielsen-Kudsk JE, Bendstrup E, et al. Prevalence, predictors, and survival in pulmonary hypertension related to end-stage chronic obstructive pulmonary disease. J Heart Lung Transplant 2012; 31:373-80.
2. Labonté L, Coulombe P, Zago M, Bourbeau J, Baglole CJ. Alterations in the Expression of the NF-κB Family Member RelB as a Novel Marker of Cardiovascular Outcomes during Acute Exacerbations of Chronic Obstructive Pulmonary Disease. PLoS One 2014 19; 9:e112965.
3. Overington JD, Huang YC, Abramson MJ, Brown JL, Goddard JR, Bowman RV, et al. Implementing clinical guidelines for chronic obstructive pulmonary disease: barriers and solutions. J Thorac Dis 2014; 6:1586-96.
4. Hassel E, Berre A, Skjulsvik A, Steinshamn S. Effects of exercise training on pulmonary vessel muscularization and right ventricular function in an animal model of COPD. Respir Res 2014 28; 15:117.
5. Song Q, Christiani DC, XiaorongWang, Ren J. The global contribution of outdoor air pollution to the incidence, prevalence, mortality and hospital admission for chronic obstructive pulmonary disease: a systematic review and meta-analysis. Int J Environ Res Public Health 2014 14; 11:11822-32.
6. Ongel EA, Karakurt Z, Salturk C, Takir HB, Burunsuzoglu B, Kargin F, et al. How do COPD comorbidities affect ICU outcomes? Int J Chron Obstruct Pulmon Dis 2014 17; 9:1187-96.
7. Hassel E, Berre A, Skjulsvik A, Steinshamn S. Effects of exercise training on pulmonary vessel muscularization and right ventricular function in an animal model of COPD. Respir Res 2014 28; 15:117.
8. Simonneau G, Robbins IM, Beghetti M, Channick RN, Delcroix M, Denton CP, et al. Updated clinical classification of pulmonary hypertension. J Am CollCardiol 2009; 54:S43-54.
9. Cuttica MJ, Kalhan R, Shlobin OA, Ahmad S, Gladwin M, Machado RF, etal.Categorization and impact of pulmonary hypertension in patients with advanced COPD. Respir Med 2010; 104:1877-82.
10. Andersen KH, Iversen M, Kjaergaard J, Mortensen J, Nielsen-Kudsk JE, Bendstrup E, et al. Prevalence, predictors, and survival in pulmonary hypertension related to end-stage chronic obstructive pulmonary disease. J Heart Lung Transplant 2012;31:373-80.
11. Sertogullarindan B, Gumrukcuoglu HA, Sezgi C, Akil MA. Frequency of pulmonary hypertension in patients with COPD due to biomass smoke and tobacco smoke. IntJ MedSci 2012; 9:406-12.
12. Ongel EA, Karakurt Z, Salturk C, Takir HB, Burunsuzoglu B, Kargin F, et al. How do COPD comorbidities affect ICU outcomes? Int J Chron Obstruct Pulmon Dis 2014 17; 9:1187-96.
13. Pauwels RA, Buist AS, Calverley PM, Jenkins CR, Hurd SS. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. Am J Respir Crit Care Med 2001; 163:1256-76.
14. Liu S, Zhou Y, Wang X, Wang D, Lu J, Zheng J, et al. Biomass fuels are the probable risk factor for COPD in rural South China. Thorax 2007; 62:889–97.
15. Buist AS, McBurnie MA, Vollmer WM, et al. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. Lancet 2007; 370: 741–750.
16. Burrows B, Earle RH. Course and prognosis of chronic obstructive lung disease. A prospective study of 200 patients.NEngl J Med. 1969 Feb 20; 280(8):397-404.

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

18. Chaouat, R. Naeije, E. Weitzenblum. Pulmonary hypertension in COPD. Eur Respir J 2008; 32: 1371–1385.

19. Chaouat A, Bugnet AS, Kadaoui N, Schott R, Enache I, Ducoloné A, Ehrhart M, Kessler R, Weitzenblum E. Severe pulmonary hypertension and chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2005 Jul 15; 172(2):189-94.

20. Sertogullarindan B, Gumrukcuoglu HA, Sezgi C, Akil MA. Frequency of pulmonary hypertension in patients with COPD due to biomass smoke and tobacco smoke. Int J Med Sci 2012; 9:406-12.

21. Scharf SM, Iqbal M, Keller C, Criner G, Lee S, Fessler HE, National Emphysema Treatment Trial (NETT) Group. Hemodynamic characterization of patients with severe emphysema.Am J Respir Crit Care Med. 2002 Aug 1; 166(3):314-22.

22. Wright JL. Relationship of pulmonary arterial pressure and airflow obstruction to emphysema. J Appl Physiol. 1993; 74:1320–4.