Original Research Article

Grading and frequency of pulmonary hypertension in chronic obstructive pulmonary disease patients

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Abstract

Background: India is a developing country, and due to industrialization, the incidence of COPD has been increasing day by day. Pulmonary hypertension (PH) is most common complication of Chronic Obstructive Pulmonary Disease (COPD). Those who are exposed to long term biomass smoke and tobacco smoke develop COPD. **Aims and Objective**: To detect Frequency and Grading of PH in COPD patients. **Material and methods:** COPD patients are diagnosed with the help of pulmonary function test (PFT) and duration of exposure to biomass or tobacco smoke. PH is detected with the help of 2D ECHO. Other causes of PH are excluded. **Observation**: Frequency of PH is more common in female (53.4%) than male (46.6%). Out of 60 patients 36.7 % developed mild, 36.6% developed moderate and 26.7 % developed severe PH. Frequency of PH in moderate COPD is 58.3%, Severe COPD is 33.3% and very severe COPD is 3.3%. **Conclusion**: There is increased incidence of PH in COPD due to Biomass smoke (53.4%) as compared to Tobacco smoke (46.6%). PH in COPD has female preponderance. COPD is more commonly associated with mild to moderate PH. Severe PH is associated with advanced disease and poor outcome. Endothelial dysfunction, Inflammation, Hyperinflation and Chronic hypoxia are affiliated with PH in COPD patients.
Key Words: pulmonary hypertension, chronic obstructive pulmonary.

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INTRODUCTION

COPD is most common cause of mortality in developing country due to Environmental pollution, Biomass or tobacco smoke, industrialization etc. In India, the mortality rate was 102.3 per 100,000 and in world it was 6,740,000/day out of world total of 27,756,000 day[1]. Multiple studies show that there is increased trend of COPD and its complications such as PH, Left ventricular failure, right ventricular failures, They are associated with significant limitation of daily activity and poor standard of living. In rural areas, the incidence of COPD is more for those who are exposed to biomass smoke as compared to tobacco smoke. Biological fuel generates biomass smoke after combustion. Biomass smoke contains all type of gases which are responsible for development of COPD. Incidence of COPD due to biomass smoke is higher as compare to tobacco smoke, so biomass smoke is the major risk factor for development of COPD in rural areas. This study is designed for detection of PH in COPD patients with history of exposure to biomass smoke (BS), tobacco smoke (TS) with the help of investigations such as PFT and 2D ECHO.

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MATERIAL AND METHOD

We included patients those who either attended medicine OPD or got admitted in medicine wards of Bharati Hospital, Sangli. Study was approved by local ethical committee. In Inclusion criteria we selected those patients who are chronically exposed to biomass smoke and tobacco smoke. We excluded patients with suboptimal ECHO study because of any reason, Patients with other co-morbid conditions like chest deformity, Bronchiectasis, Pulmonary Fibrosis, Lung Cancer, Sleep Apnoea, Obesity (BMI more than 35), Primary cardiac illness, clinically unstable patients and Pregnant women. Cumulative exposure to biomass smoke is expressed in hour-year and tobacco smoking is expressed in pack-year. Diagnosis of COPD is performed with the help of PFT. In PFT FEV1 (Forced Expiratory Volume in one second) and FVC (Forced Vital Capacity) is measured. The functional criteria for detection of irreversible airflow obstruction is FEV1/FVC <70%, FEV1 <80% predicted and without asthma as assessed by clinical history and response to bronchodilators (change, 12% in FEV1 following 400 µg of inhaled salbutamol)[2]. Pulmonary Function Tests: Post-bronchodilator spirometry was performed with a flow sensitive spirometer (spiro axel). At least three reproducible maximal expiratory efforts starting from complete inspiration were obtained; the best FEV1 was used to calculate FEV1 as percent predicted. FVC and FEV1 were expressed as percentage of predicted (FVC %, FEV1 %). Echocardiography performed by same cardiologist with 10years experience. Echocardiography was done with vivid machine and 5s probe was used. The difference between right ventricular systolic pressure and right atrial pressure was taken for measurement of tricuspid jet velocity. Simplified Bernoullis Equation was used for measurement of Pulmonary arterial hypertension (Systolic Pulmonary Arterial Pressure (PASP)= 4 x V^2 + Right Atrial Mean Pressure). Right Atrial Mean Pressure was estimated to be 5 mm Hg when diameter of inferior Vena Cava < 1.7cm and > 50% decrease in diameter with inspiration, 10 mm Hg when IVC was more > 1.7 cm with normal inspiratory collapse > 50% and 15 mm Hg when IVC is > 1.7 cm and inspiratory collapse is < 50%[3]. In the 4th World Symposium on Pulmonary Hypertension, the Working Group on Diagnosis and Assessment of Pulmonary Arterial Hypertension defined pulmonary arterial pressure is more than 35mmHg is PH[4]. PH is divided in three groups Mild (35-50mmHg). Moderate(50-70mmHg) and severe(>70mmHg)[3]

RESULTS

Total 60 patients were selected for study as per inclusion and exclusion criteria. Out of 60 patients 32(53.4%) were females and 28(46.6%) were males. All females and 2 males had history of BS exposure and 26 males had TS exposure. All the patients of COPD in our study show PH. The cumulative exposure time was min. 160 h-yr and max. 360 h-yr for BS and min 15 pack/years and max 105 pack/years for TS. Out of 60 patients 36.7 % developed mild, 36.6% developed moderate and 26.7 % developed severe PH. Out of 28 male patients 57.10% developed mild PH, 28.50% developed moderate PH and 14.70% developed severe PH. In females, out of 32 patients 21.80% developed mild PH, 40.60% developed moderate PH and 37.50% developed severe PH. (p 0.05) More severe form of PH was observed in females who had biomass smoke exposure, while mild form of PH was observed in males who were tobacco smoker. The difference was statistically significant.

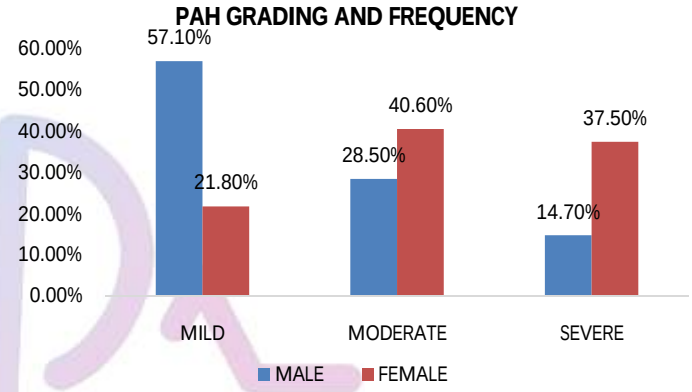

Figure 1:

Table 1:

PAH	MALE	FEMALE
MILD	16 (57.1%)	7 (21.80%)
MODERATE	8 (28.50%)	13 (40.60%)
SEVERE	4 (14.70%)	12 (37.50%)
Total	**28 (46.7%)**	**32 (53.30%)**

The chi-square statistic is 8.4833. the p value is 0.014384. the result is significant at p <0.05

DISCUSSION

The pathophysiology of the development of PH in COPD is not well understood and is multifactorial. The central stimulus to these processes remains chronic exposure of airways to noxious stimuli like tobacco and biomass smoke leading to chronic hypoxemia and endothelial dysfunction. Pathogenesis of pulmonary hypertension in COPD is that an endothelium-derived vasoconstrictor-dilator is imbalanced, mainly from a decreased endothelial nitric oxide expression and there is increased vascular endothelial growth factor with serotonin transporter expressions.[5-8] Chronic hypoxia is considered to major mechanism of causing PH in COPD.[9-10] BS is

composed of a relatively equal mixture of gases and particles and can penetrate deeply into the lung, producing a variety of morphologic and biochemical changes.[11,12] A recent meta-analysis, which reviewed risk of COPD from exposure to BS, concluded that BS exposure is a clear risk factor for COPD.[13] Most studies have reported a prevalence of PH in COPD to be between 30 % to 80 %.[10,14,15] In our study we found that all COPD patients were suffering from PH and female patients were more prone for development of PH due to BS exposure then males who were TS exposed. Newer therapies for treating these specific abnormalities are under trial and may be used in management option in severe PH.

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