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Original Research Article

Pulmonary Hypertension in Patient Suffering From Chronic Obstructive Pulmonary Disease

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Abstract

Chronic obstructive pulmonary disease is a leading cause of morbidity and mortality in adults all over the world. While other major causes of non-cancer mortality such as coronary artery disease and stroke have shown a consistent downward trend, COPD is the only one that continues to increase [1]. In the present study, Majority of patients were in the age group 61-70years. Out of 50 patients studied, 34 patients were male and 16 patients were females. Most of the patients are farmers by occupation followed by beedi worker and factory worker. Cough was present in 92% of patients, sputum production in 96% of patients, breathlessness in 88% of patients, weakness and fatigue was present in 54 % of patients and swelling of lower limbs in 30% of patients. Among 50 patients, 64% of patients were smokers and 36% were non-smokers. 82% of patients had use of accessory muscles of respiration, 52% patients had barrel shaped chest, chest movements were found to be minimal in 64% patients, rhonchi was present in 62% patients, crepitations were heard in 90% patients and clinical signs of pulmonary hypertension were present only in 36% patients.70% patients were found to have severe pulmonary hypertension, 28% had moderate pulmonary hypertension and mild pulmonary hypertension was present in 2%patients. Diagnosis of pulmonary hypertension in patients with COPD is difficult because patients diagnosed with COPD, and are stable, do not routinely undergo right heart catheterization – which makes PH more difficult to detect. In addition, the symptoms of both diseases are easily mistaken as the. While it is difficult to determine the prevalence of COPD with pulmonary hypertension, early detection can improve treatment and quality of life.

Keywords: Pulmonary Hypertension, Chronic Obstructive Pulmonary Disease, Cough, Breathlessness, Sputum.

INTRODUCTION

Chronic obstructive pulmonary disease is a leading cause of morbidity and mortality in adults all over the world. While other major causes of non-cancer mortality such as coronary artery disease and stroke have shown a consistent downward trend, COPD is the only one that continues to increase [1]. In the United States, in the year 2000, COPD was a major cause for health care utilisation with 8 million physician office/ hospital outpatient visits, 1.5 million emergency department visits, and 673,000 hospitalisations [2]. The epidemiological scenario is expected to worsen and the World Health Organization predicts that COPD will become the third leading cause of death (currently fourth) and the fifth leading cause of disability (currently twelfth) worldwide by the year 2020 [3, 4].

While the characteristic abnormality in COPD is an inflammatory state of the airways that occurs in response to exposure to noxious stimuli and results in airflow limitation that is only partially reversible, and is usually progressive, the pulmonary involvement extends beyond the airways. A major contributor to airflow limitation is emphysema, the dilation and destruction of airspaces distal to the terminal bronchiole in the lung parenchyma without accompanying inflammation. In recent years, pulmonary vascular pathology is increasingly being recognised as the third important lung involvement that contributes to the morbidity and mortality.

Pulmonary hypertension is a common complication of chronic obstructive pulmonary disease (COPD). Its presence is associated with shorter survival and worse clinical evolution. In COPD, pulmonary hypertension tends to be of moderate severity and progresses slowly. However, transitory increases of pulmonary artery pressure may occur during exacerbations, exercise and sleep. Right ventricular function is only mildly impaired with preservation of the cardiac output.

Structural and functional changes of pulmonary circulation are apparent at the initial stages of COPD. Recent investigations have shown endothelial dysfunction and changes in the expression of endothelium-derived mediators that regulate vascular tone and cell growth in the pulmonary arteries of

patients with mild disease. Some of these changes are also present in smokers with normal lung function. Accordingly, it has been postulated that the initial event in the natural history of pulmonary hypertension in COPD could be the lesion of pulmonary endothelium by cigarette-smoke products.

Long-term oxygen administration is the only treatment that slows down the progression of pulmonary hypertension in chronic obstructive pulmonary disease. Nevertheless, with this treatment pulmonary artery pressure rarely returns to normal values and the structural abnormalities of pulmonary vessels remain unaltered. Vasodilators are not recommended on the basis of their minimal clinical efficacy and because they impair pulmonary gas exchange. Recognition of the role of endothelial dysfunction in the physiopathology of pulmonary hypertension in chronic obstructive pulmonary disease opens new perspectives for the treatment of this complication.

Pulmonary hypertension is a frequent complication in the natural history of chronic obstructive pulmonary disease (COPD). Its presence is associated with shorter survival rates and it has been identified as a predictive factor of worse clinical outcomes and frequent use of health resources. At the present time, there is no specific and effective treatment for this condition in COPD. However, recent advances in knowledge of the pathogenesis of pulmonary hypertensive states, along with the development of new and effective strategies in the treatment of pulmonary hypertension, open a new perspective that could be applicable in COPD.

OBJECTIVE

To evaluate pulmonary hypertension in COPD patients

METHODOLOGY

All patients who presented with history of cough, sputum, breathlessness or wheezing of more than 3 months duration to the medical outpatient or admitted in medical wards of ADICHUNCHANAGIRI INSTITUTE OF MEDICAL SCIENCES were subjected to pre and post-bronchodilator pulmonary function testing. These patients were evaluated for presence of pulmonary hypertension. This study period was from JANUARY 2015 to DECEMBER 2015 with 50 sample size.

RESULTS

Table-1: Age and Sex wise distribution of cases

Age group (years)	Male		Female		Total	
	No.	%	No.	%	No.	%
41-50	**03**	06	**01**	02	**04**	**08**
51-60	**13**	26	**04**	08	**17**	**34**
61-70	**10**	20	**08**	16	**18**	**36**
>70	**08**	16	**03**	06	**11**	**22**
Total	**34**	**68**	**16**	**32**	**50**	**100**

Majority of patients were in the age group 61-70years. Out of 50 patients studied, 34 patients were male and 16 patients were females.

Table-2: Occupation wise distribution of patients

Occupation	Number of patients	%
Agriculture	31	62
beedi worker	11	22
Factory worker	08	16
Total	50	100

Most of the patients are farmers by occupation followed by beedi worker and factory worker.

Table-3: distribution of patients based on Clinical symptoms

Clinical Symptoms	er of patients (n=50)	%
Cough	46	92.0
Breathlessness	44	88.0
Sputum	48	96.0
Fever	29	48.0
Weight loss	49	98.0
Swelling of lower limbs	15	30.0
Weakness and fatigue	27	54.0
Chest pain	24	48.0

Cough was present in 92% of patients, sputum production in 96% of patients, breathlessness in 88% of patients, weakness and fatigue was present in 54 % of patients and swelling of lower limbs in 30% of patients.

Table-4: distribution of patients based on Smoking status

Smoking status	r of patients (n=50)	%
No	18	36.0
Yes	32	64.0
Total	50	100.0

Among 50 patients, 64% of patients were smokers and 36% were non-smokers.

Table-5: Examination of respiratory system

Examination of respiratory system	ber of patients (n=50)	%
Use of accessory muscles	41	82.0
Barrel shaped chest	26	52.0
Chest movements		
Normal	15	30.0
Minimal	32	64.0
Rhonchi	31	62.0
Crepitations	45	90.0
CVS-PH	18	36.0

82% of patients had use of accessory muscles of respiration, 52% patients had barrel shaped chest, chest movements were found to be minimal in 64% patients, rhonchi was present in 62% patients, crepitations were heard in 90% patients and clinical signs of pulmonary hypertension were present only in 36% patients.

Table-6: Severity of Pulmonary hypertension

PASP	ber of patients (n=50)	%
Mild PH 20-30 mmHg	1	2.0
Moderate PH 30-50 mmHg	14	28.0
Severe PH >50 mmHg	35	70.0
Total	50	100.0

70% patients were found to have severe pulmonary hypertension, 28% had moderate pulmonary hypertension and mild pulmonary hypertension was present in 2% patients.

DISCUSSION

In the present study, Majority of patients were in the age group 61-70years. Out of 50 patients studied, 34 patients were male and 16 patients were females. Most of the patients are farmers by occupation followed by beedi worker and factory worker. Cough was present in 92% of patients, sputum production in 96% of patients, breathlessness in 88% of patients, weakness and fatigue was present in 54 % of patients and swelling of lower limbs in 30% of patients. Among 50 patients, 64% of patients were smokers and 36% were non-smokers. 82% of patients had use of accessory muscles of respiration, 52% patients had barrel shaped chest, chest movements were found to be minimal in 64% patients, rhonchi was present in 62% patients, crepitations were heard in 90% patients and clinical signs of pulmonary hypertension were present only in 36% patients.70% patients were found to have severe pulmonary hypertension, 28% had moderate pulmonary hypertension and mild pulmonary hypertension was present in 2% patients.

According to study by Gupta NK, a total 40 of patients of COPD were selected and staged by pulmonary function test (PFT) and evaluated by echocardiography. **Results:** On echocardiographic evaluation of COPD, 50% cases had normal echocardiographic parameters. Measurable tricuspid regurgitation (TR) was observed in 27/40 cases (67.5%). Pulmonary hypertension (PH), which is defined as systolic pulmonary arterial pressure (sPAP) > 30 mmHg was observed in 17/27 (63%) cases in which prevalence of mild, moderate, and severe PH were 10/17 (58.82%), 4/17 (23.53%), and 3/17 (17.65%), respectively. The frequencies of PH in mild, moderate, severe, and very severe COPD were 16.67%, 54.55%, 60.00%, and 83.33%, respectively. Right atrial pressure was 10 mmHg in 82.5% cases and 15 mmHg in 17.5% cases. Cor pulmonale was observed in 7/17 (41.17%) cases; 7.50% cases had left ventricle (LV) systolic dysfunction and 47.5% cases had evidence of

LV diastolic dysfunction defined as A ≥ E (peak mitral flow velocity of the early rapid filling wave (E), peak velocity of the late filling wave caused by atrial contraction (A) on mitral valve tracing). Left ventricle hypertrophy was found in 22.5% cases [5].

According to study by Higham MA, Presence or absence of tricuspid regurgitation (TR) was determined by Doppler echocardiography in 73 consecutive COPD patients attending a hospital outpatient clinic. Transtricuspid pressure gradient (TTPG) was calculated. PH was defined as TTPG ≥30 mmHg. Patients also underwent spirometry, forced expiratory volume in one second (FEV_1), single breath gas transfer (carbon monoxide transfer coefficient; (K_{CO}) and carbon monoxide diffusing capacity of the lung; $D_{L,CO}$) and arterial blood gas measurement. Measurable TR was observed in 56/73 patients (77%). There were no differences between the group in which TR was observed compared to that in which it was absent, with regard to age, smoking history nor pulmonary function variables. PH was seen in 31/56 cases (55%), with good reproducibility. There were statistically significant correlations of TTPG with FEV_1 (r=−0.26, p=0.05), K_{CO} (r=−0.31, p=0.04) and $D_{L,CO}$ (r=−0.42, p=0.006) expressed as % pred. Stepwise regression analysis showed that age and K_{CO} combined provide a multivariate model for prediction of TTPG [6].

Mild-to-moderate pulmonary hypertension is a common complication of chronic obstructive pulmonary disease (COPD); such a complication is associated with increased risks of exacerbation and decreased survival. Pulmonary hypertension usually worsens during exercise, sleep and exacerbation. Pulmonary vascular remodelling in COPD is the main cause of increase in pulmonary artery pressure and is thought to result from the combined effects of hypoxia, inflammation and loss of capillaries in severe emphysema. A small proportion of COPD patients may present with "out-of-proportion" pulmonary hypertension, defined by a mean pulmonary artery pressure >35-40 mmHg (normal is no more than 20 mmHg) and a relatively preserved lung function (with low to normal arterial carbon dioxide tension) that cannot explain prominent dyspnoea and fatigue. The prevalence of out-of-proportion pulmonary hypertension in COPD is estimated to be very close to the prevalence of idiopathic pulmonary arterial hypertension. Cor pulmonale, defined as right ventricular hypertrophy and dilatation secondary to pulmonary hypertension caused by respiratory disorders, is common. More studies are needed to define the contribution of cor pulmonale to decreased exercise capacity in COPD [7].

In 41 patients with stable COPD, we prospectively performed CWD and right heart catheterization. The mean value of PAPs for the entire group was 38.5 +/- 14.9 mm Hg. Pulmonary arterial hypertension (PAPs greater than or equal to 35 mm Hg) occurred in 51 percent (21/41) of patients. Doppler estimation of PAP was impossible in 34 percent (14/41) because of poor signal quality (n = 3), absence of Doppler-detected TR (n = 8), and inadequate TR Doppler signal (n = 3). The PAP could be estimated in 66 percent (27/41) of patients. A statistically significant correlation was found between the Doppler-estimated PAP and the catheter-measured PAPs (r = 0.65; p less than 0.001; SEE = 9 mm Hg). Therefore, CWD appears to be useful for the noninvasive estimation of PAP in patients with COPD [8].

CONCLUSION

Diagnosis of pulmonary hypertension in patients with COPD is difficult because patients diagnosed with COPD, and are stable, do not routinely undergo right heart catheterization – which makes PH more difficult to detect. In addition, the symptoms of both diseases are easily mistaken as the another. While it is difficult to determine the prevalence of COPD with pulmonary hypertension, early detection can improve treatment and quality of life.

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