ORIGINAL RESEARCH PAPER

Volume-8 | Issue-1 | January-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

STUDY OF PULMONARY HYPERTENSION IN CHRONIC RESPIRATORY ILLNESS

Medicine

Shivprasad Kasat Assistant Professor, Department of Respiratory Medicine, MGM Medical College and Hospital, Aurangabad.

Rekha Bolla* Resident, Department of Respiratory Medicine, MGM Medical College and Hospital, Aurangabad. *Corresponding Author

Ashish Deshmukh Professor and HOD, Department of Respiratory Medicine, MGM Medical College and Hospital, Aurangabad.

Sunil Jadhav Associate Professor, Department of Respiratory Medicine, MGM Medical College and Hospital, Aurangabad.

Hafiz Deshmukh Assistant Professor, Department of Respiratory Medicine, MGM Medical College and Hospital, Aurangabad.

KEYWORDS

INTRODUCTION:

Chronic obstructive lung disease (COPD) and diffuse parenchymal lung diseases (DPLD), including idiopathic pulmonary fibrosis (IPF) and sarcoidosis, are associated with a high incidence of pulmonary hypertension (PH), which is linked with exercise limitation and a worse prognosis.[1] Pulmonary hypertension, 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. Anatomical evidence of right-ventricular hypertrophy can be found at autopsy in up to 40% of patients with COPD. Pulmonary hypertension in COPD progresses over time and its severity correlates with the degree of airflow obstruction and the impairment of pulmonary gas exchange[2-7].Pulmonary hypertension a common complication in lung disease. In the latest revised classification of pulmonary hypertension (PH), chronic lung diseases or conditions with alveolar hypoxia are included in WHO Group III of PH-related diseases. Alveolar hypoxia and thereby PH may occur in distinct conditions including: parenchymal lung disease, chronic airway diseases, ventilatory control abnormalities, residence at high altitude, progressive neuromuscular diseases and mixed obstructive and restrictive lung diseases.[7]

Alveolar hypoxia which is considered to be a potent stimulus for pulmonary vasoconstriction. This operates at the endothelial level and is one of the most important pathways leading to PH development in chronic lung diseases. Alveolar hypoventilation leads to the precipitation of acute pulmonary vasoconstriction in some regions of the lungs, and vasodilation in others, causing physiological shunt.[8]

Hypoxia leads to pulmonary vasoconstriction which in turn leads to an increase in pulmonary vascular resistance. There are basically Two important mechanisms which are postulated to support this phenomenon. One is that the vasoconstriction is achieved either through activation of a vasoconstrictor pathway or inactivation of a the vasodilator pathway, or alternatively via the effects of hypoxia on the vascular smooth muscle [9].

In study by Scharf et al. saw in its patients with severe COPD, to have 60% elevated pulmonary capillary wedge pressures. PH is associated with exercise limitation among patients with COPD. In a study of 362 pre-transplant patients with COPD, PH (mean PAP ≥25mmHg) was seen to be associated with shorter 6- minute walking distance (6MWD) after adjustments for demographics and lung function.[10,11] Yet in another study by Thabut et al.[12] in a cluster analysis identified a subgroup of COPD patients with out-of-proportion PH associated with severe hypoxia .

The primary respiratory condition and PH may both be associated with dyspnea, the later often goes unrecognised. Therefore, data on PH prevalence in each of these conditions is very limited. Hence the current study was planned to see for the prevalence and also to see the association between the two.[13]

METHODOLOGY

The study is a cross sectional observational study conducted at MGM Hospital Aurangabad after obtaining permission of the institutional ethics committee.

The study data is from January 2017 to January 2018.

The survey was conducted by taking written and informed consent from the patients.

STUDY AIMS AND OBJECTIVE:

To study the correlation between hypoxia and pulmonary hypertension in chronic respiratory illness like COPD, ILD, Bronchiestasis and fibrocavitatory diseases

INCLUSION CRITERIA

- Age above 40 years
- Patients with chronic respiratory illness and PAH
- All the patients who give consent to perform 2D echo and ABG will be included in the study

EXCLUSION CRITERIA:

- Patients having acute respiratory illness
- Patients with acute myocardial infarction

SAMPLE SIZE

The final sample size of the study after considering the inclusion and exclusion criteria was 152 patients.

PROTOCOL FOR DATA COLLECTION

Survey was conducted in three phases:

1. Interview of the subjects
2. Arterial blood gas analysis and
3. 2D ECHO

INTERVIEW OF THE SUBJECTS

A short history was administered face to face to the subject relating age, sex, occupation, smoking history and respiratory symptoms.

ARTERIAL BLOOD GAS ANALYSIS

An **arterial-blood gas (ABG)** test measures the amounts of arterial gases, such as oxygen carbon dioxide. An ABG test requires that a small volume of blood be drawn from the radial artery with a syringe and a thin needle, but sometimes the femoral artery in the groin or another site is used. The blood can also be drawn from an arterial catheter. An ABG test measures the blood-gas tension values of the arterial partial pressure of oxygen, and the arterial partial pressure of carbon dioxide, and the blood's pH. In addition, the arterial oxygen saturation can be determined. Such information is vital when caring for

patients with critical illnesses or respiratory disease.

2D ECHO:

Echocardiography with Doppler studies is the most useful first line investigation in a patient presenting with clinical features suggestive of pulmonary hypertension
It facilitates:

1) Estimation of pulmonary artery systolic pressure to determine if PH is present.
2) Assessment of cardiac cause of PH
3) Assessment of severity of RV dysfunction
4) Assessment of prognostic variables

Estimating Pulmonary Artery Systolic Pressure

Echocardiographic evaluation of pulmonary artery systolic pressure (PASP) relies on the fact that PASP approximates right ventricular systolic pressure (RVSP) in the absence of right ventricular outflow obstruction. The most accurate echocardiographic method for estimating (PASP) uses the simplified Bernoulli equation to obtain a systolic trans-valvular pressure gradient
DPRV-RA = 4(VTR)2
Where VTR is the velocity of the tricuspid regurgitant jet
Severity of pulmonary hypertension grading as:
Mild=25-40mmHg
Moderate=41-55mmHg
Severe = > 55 mmHg
The results were analysed using Microsoft excel and whenever applicable appropriate tests were done such as t test and chi-square to see for association. Percentage and graph was used to present the descriptive statistics.

RESULTS :

The study included 152 patients with chronic respiratory illness, all the patients were above the age group of 40 years. Majority of the patients belong to the age group of 61-70 years of age. The mean age among the participants was 66.5±12.02 years. There were majority males (71.7%) and males: female ratio was found to be 2.5:1. The study had 99(65.2%) smokers and 10(6.6%) non smokers. There were 43(28.2%) who were exposed to chulla smoke.(table 1).

Table no 1: Distribution of patients according to smoke exposure status.

Smoking	Number	Percent
Smokers	99	65.2
Non -smokers	10	6.6
Chulla exposure	43	28.2
Total	152	100

The study had majority patients of COPD. There were 87 males and 33 females of COPD. The study had 6 males and 4 females who had interstitial lung disease(ILD) and 8 males and 4 females with bronchiectasis and 7 males and 3 females of fibro-cavitatory disease.
The figure 1 shows in detail the sex-wise distribution of patients and their diseases.

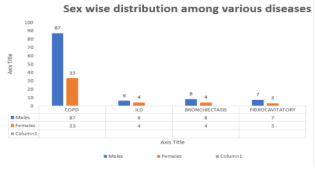

Figure 1: Sex wise distribution of patients according to their disease

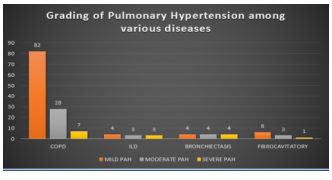

Figure 2: Distribution of patients according to severity of PAH and disease.

Table 2: distribution of patients based on PAH severity.

PAH severity	Males N (%)	Females N (%)	Total (%)
Mild	66 (43.4)	28 (18.4)	94 (61.8)
Moderate	28 (18.4)	10 (06.6)	38 (25.0)
Severe	15 (9.9)	05 (03.3)	20 (13.2)
Total	109 (71.7)	43 (28.3)	152(100)

The study also shows association between severity of PAH and PO2. The study shows that there was significant association between the means and standard deviation of patients of severe PAH and mild PAH. (P=<0.001). Table 3 shows mean and deviation of PO2.the study saw significant association between smoking and PO2.

Table 3: Mean and standard deviation of different factors

Factor	Mean	Standard deviation	
Pa O2 of all patients	93.2	5.09	P = < 0.001
Pa O2 of smokers	63.2	5.09	
PaO2 of non smokers	71.45	10.11	
Pa O2 mild PAH	72.75	6.85	P = < 0.001
Pa O2 moderate PAH	64.25	10.67	
Pa O2 severe PAH	53.0	18.38	

DISCUSSION

Pulmonary hypertension in disease states associated with decreased alveolar partial pressure of oxygen (PO2) is considered to develop from persistent hypoxic vasoconstriction and subsequent vascular remodelling[14]. In the study by Matthias [15]et al, mild PH was in 50.2% moderate in 9.8% and severe in 3.7% In our study we had mild cases of 61.8% and moderate cases of 25% and severe cases were 13.2%. the number of cases were more in the current study. The study by Chaouat [16]et al, stated that pulmonary hypertension to be a stronger prognostic factor in patients with COPD than FEV1 , hypoxemia or hypercapnia. In the current study we had association between Pulmonary hypertension and PaO2.[17]

The study by Burrows et al. in 1972 showed the COPD patients with, pulmonary hypertension to be associated with shorter survival rates. The current study also found association between PAH and PaO2.
Earlier studies performed in COPD patients have shown wide variation in the individual responses of the pulmonary circulation to changes in inspired oxygen concentration, but there was low correlation seen between Pa,O2 and Ppa. There was a suggestion made that mechanisms other than hypoxaemia should be at the origin of pulmonary hypertension in COPD.[18,19]

A variety of factors may contribute to the development and maintenance of pulmonary hypertension in COPD. The most significant of which are the remodelling of pulmonary vessels and hypoxic pulmonary vasoconstriction. Patients with end-stage COPD and cor pulmonale there were changes in pulmonary muscular arteries and precapillary vessels which explained the irreversible increase of pulmonary vascular resistance. Pulmonary vascular abnormalities in patients with mild-to-moderate disease mainly consist of thickening of the intima of pulmonary muscular arteries which reduces the lumen size and an increased proportion of muscularised arterioles [18-23].

Conclusion

The study shows that mostly the patients of chronic disease having PAH belong to COPD. Majority were males. There were majority who had smoking or chulla exposure and there was association between smoking and PO_2. There was also association of severity of PAH and PO_2. The study shows that there was increase in severity of PAH when the PO_2 decreases.

REFERENCES

1. Chaouat A, Bugnet A-S, Kadaoui N, et al. Severe pulmonary hypertension and chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2005;172:189–94.
2. Fishman AP. State of the art: chronic cor pulmonale. Am Rev Respir Dis 1976; 114: 775–794.
3. Weitzenblum E, Kessler R, Oswald M, Fraisse Ph. Medical treatment of pulmonary hypertension in chronic lung disease. Eur Respir J 1994; 7: 148–152.
4. Pietra GG. Pathology of the pulmonary vasculature and heart. In: Cherniack NS, ed. Chronic obstructive pulmonary disease. Philadelphia, W.B. Saunders, 1991; pp. 21–26
5. Weitzenblum E, Sautegeau A, Ehrhart M, Mammosser M, Hirth C, Roegel E. Long-term course of pulmonary arterial pressure in chronic obstructive pulmonary disease. Am Rev Respir Dis 1984; 130: 993–998.
6. Scharf SM, Iqbal M, Keller C, Criner G, Lee S, Fessler HE. Hemodynamic characterization of patients with severe emphysema. Am J Respir Crit Care Med 2002; 166: 314–322.

7. Sajkov D, Mupunga B, Bowden JJ, Petrovsky N. Pulmonary Hypertension in Chronic Lung Diseases and/or Hypoxia. InPulmonary Hypertension 2013.
8. Swenson ER. Hypoxic Pulmonary Vasoconstriction and Chronic Lung Disease. Advances in Pulmonary Hypertension. 2013 Oct 1;12(3).
9. Sylvester, J. T, Shimoda, L. A, Aaronson, P. I, & Ward, J. P. Hypoxic pulmonary vaso•constriction Physiol Rev (2012). , 92(1), 367-520.
10. Scharf, S. M, Iqbal, M, Keller, C, Criner, G, Lee, S, & Fessler, H. E. Hemodynamic characteristics of patients with severe emphysema. Am J Respir Crit Care Med (2002). , 166, 314-322.
11. Sims, M. W, Margolis, D. J, Localio, A. R, Panettieri, R. A, Kawut, S. M, & Christie, J.D. Impact of pulmonary artery pressure on exercise function in severe COPD. Chest (2009). , 136, 412-419.
12. Thabut, G, Dauriat, G, Stern, J. B, Logeart, D, Lévy, A, Marrash-chahla, R, et al. Pul•monary hemodynamics in advanced COPD candidates for lung volume reduction surgery or lung transplantation. Chest (2005). , 127, 1531-1536.
13. Orr, R, Smith, L. J, & Cuttica, M. J. Pulmonary hypertension in advanced chronic ob•structive pulmonary disease. Curr Opin Pulm Med (2012). , 18(2), 138-143.
14. Heath D, Smith P, Rios DJ, Williams D, Harris P. Small pulmonary arteries in some natives of La Paz, Bolivia. Thorax 1981; 36: 599–604.
15. Held M, Jany BH. Pulmonary hypertension in COPD. Respiratory care. 2013 Aug 1;58(8):e86-91.
16. Chaouat A, Naeije R, Weitzenblum E. Pulmonary hypertension in COPD. Eur Respir J 2008;32(5):1371-1385.
17. Barbera` JA, Riverola A, Roca J, et al. Pulmonary vascular abnormalities and ventilation-perfusion relationships in mild chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1994; 149: 423–429.
18. Peinado VI, Barbera` JA, Ramirez J, et al. Endothelial dysfunction in pulmonary arteries of patients with mild COPD. Am J Physiol 1998; 274: L908–L913.
19. Wright JL, Petty T, Thurlbeck WM. Analysis of the structure of the muscular pulmonary arteries in patients with pulmonary hypertension and COPD: National Institutes of Health nocturnal oxygen therapy trial. Lung 1992;170: 109–124.
20. Wilkinson M, Langhorne CA, Heath D, Barer GR, Howard P. A pathophysiological study of 10 cases of hypoxic cor pulmonale. Q J Med 1988; 249: 65–85.
21. Magee F, Wright JL, Wiggs BR, Pare´ PD, Hogg JC. Pulmonary vascular structure and function in chronic obstructive pulmonary disease. Thorax 1988; 43: 183–189.
22. Wright JL, Lawson L, Pare´ PD, et al. The structure and function of the pulmonary vasculature in mild chronic obstructive pulmonary disease. Am Rev Respir Dis 1983; 128: 702–707.
23. Hale KA, Niewoehner DE, Cosio MG. Morphologic changes in the muscular pulmonary arteries: Relationship to cigarette smoking, airway disease, and emphysema. Am Rev Respir Dis 1980; 122: 273–278.