

To study the correlation of Forced Expiratory Volume at 1st second (FEV₁) with Severity of Pulmonary Hypertension At A Tertiary Care Hospital in Southern Rajasthan.

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Abstract

Introduction: Pulmonary Hypertension is an increase in the pulmonary arterial, venous and capillary pressure together known as the lung vasculature, leading to shortness of breath, dizziness, fainting, chest pain, pedal oedema, ascites and other symptoms all of which are exacerbated by exertion. It is generally considered that the severity of the respiratory disease increases the risk of Pulmonary Hypertension. There is a wide variation in the prevalence of Pulmonary Hypertension due to chronic respiratory diseases.

Aim: To find the correlation of Forced Expiratory Volume at 1st second (FEV₁) with Severity of Pulmonary Hypertension.

Methodology: The study was a prospective study conducted in the Department of Pulmonary Medicine of a tertiary care hospital. A total of 240 patients were selected randomly, of which 40 were lost to follow up due to various reasons.

Result. In our study there is significant inverse correlation between FEV₁ and PHTN (mPAP >25 mmHg) using Pearson correlation (p value < 0.001), that is as FEV₁ decreases, pulmonary hypertension increases.

Conclusion. The Spirometry values declined with severity of pulmonary Hypertension. As the spirometry parameters deteriorated the number of patients having pulmonary hypertension increases.

Keywords: Pulmonary hypertension, Chronic respiratory diseases, Forced Expiratory Volume at 1st second (FEV₁).

Introduction.

Pulmonary Hypertension (PH) is defined by mean pulmonary artery pressure, mPAP \geq 25 mm Hg at rest measured by right heart catheterization.⁽¹⁾ It can lead to serious condition with a markedly decreased exercise tolerance and heart failure. Pulmonary Hypertension complicates course of chronic respiratory diseases. There is an increased understanding for this intervening complication and its association with functional impairment, greater oxygen requirement, quality of life and prognosis. The

severity of Pulmonary Hypertension in chronic respiratory diseases tends to be mild to moderate but it may be severe in some cases. Many factors are involved in the pathogenesis of Pulmonary Hypertension. It is generally considered that the severity of the respiratory disease increases the risk of Pulmonary Hypertension.^(1,2) The severity of Pulmonary Hypertension is classified on the basis of mean pulmonary artery pressure as Grade 1 mild (25 to 40 mm Hg), Grade 2 moderate (41 to 55 mm Hg) and Grade 3 severe (> 55 mm Hg).⁽³⁾

Pulmonary function tests are interpreted by comparing individual patient data with reference values of normal subjects⁽⁴⁾. Analysis begins with evaluation of the ratio of FEV₁ to Vital Capacity (VC). While historically, the ratio of FEV₁ to FVC served as the basis for distinguishing obstructive disorders from normality or restrictive diseases. If the ratio is less than the lower limit of normal and the VC is at or above the lower limit of normal, the pattern is obstructive. If Total Lung Capacity (TLC) is not at or above lower limit of normal, a mixed obstructive-restrictive pattern is suggested. If FEV₁/VC and VC are each equal to or greater than the respective lower limits of normal, spirometry is considered as normal. There is a wide variation in the prevalence of Pulmonary Hypertension due to chronic respiratory diseases.^(2,3) This study aims at finding the correlation of Forced Expiratory Volume at 1st second (FEV₁) with Severity of Pulmonary Hypertension.

Methodology.

The study was a prospective study conducted in the Department of Pulmonary Medicine of a tertiary care hospital in Southern Rajasthan. Before proceeding for the study, the required proforma and plan of the study were submitted to the Ethics committee for Research on Human Subjects of the institute and were approved.

Patient with underlying respiratory diseases were screened for presence of Pulmonary Hypertension, by symptomatology, chest X-ray PA view, ECG changes ('p' pulmonale and right axis deviation), clinical finding i.e. loud P₂, split S₂, tricuspid regurgitation, parasternal impulse, raised JVP, etc. If on these basic investigations, Pulmonary Hypertension was suspected, 2D ECHO was advised. Based on 2D echo findings, Pulmonary Hypertension was classified as mild (grade 1), moderate (grade 2) and severe (grade 3), that is corresponding to mPAP values of 25-40, 41-55, >55 mm Hg respectively.

A total of 240 patients were selected randomly, of which 40 were loss to follow up due to various reasons. Among the 200 patients studied the disease distribution was as follows : 80 COPD, 25 ILD, 30 Bronchiectasis, 50 Asthma, 5 Pneumoconiosis, 5 Sarcoidosis, 5 Obstructive Sleep Apnea associated with COPD. Patients with chronic respiratory disease of either sex who met the inclusion criteria (details below) and completed the spirometry including FEV₁, FVC and FEV₁/FVC ratio and 6 Minute Walk Test were selected.

Inclusion Criteria.

1. Chronic respiratory diseases such as Chronic Obstructive Lung Disease, Bronchiectasis, Interstitial Lung Diseases, Pneumoconiosis, Asthma.
2. 18 years.
3. Sex – Either Sex.

Exclusion criteria.

1. Infectious Diseases.
2. Debilitated Patient.
3. Congestive Heart Failure, Left Heart Failure, Recent Myocardial Infarction.
4. Valvular Heart Disease (Mitral Stenosis, Mitral Regurgitation).
5. Thrombo Embolic Disease.

Results.

In this study, the population of male was 132 (66%) and female was 68 (34%). In our study majority of male and female are of COPD, but using chi square test the significance is not significant.

In our study 18% of the patients had pulmonary hypertension.

Table 1. Patients classified as per the severity of Pulmonary Hypertension (PHTN):

Grades of PHTN	No. of patients	Percentage
1	28	76
2	07	19
3	02	05
Total	37	100

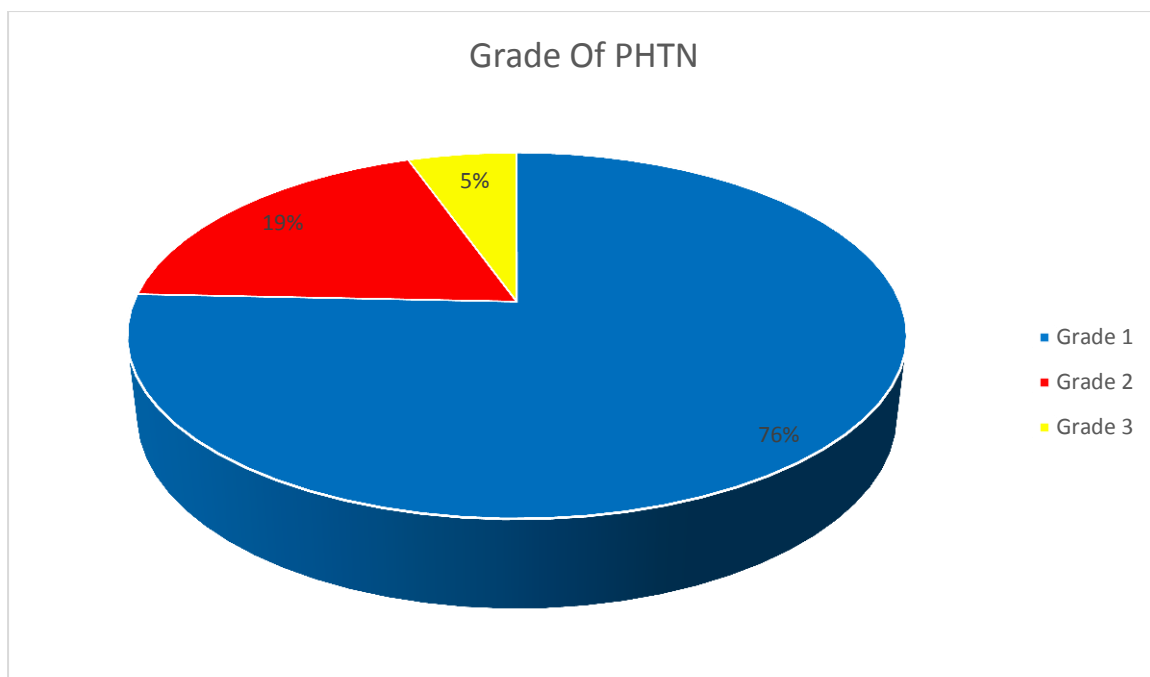


Figure 1 Grade of PHTN

In this study, grade 1 had 76%, grade 2 had 19% and grade 3 had 5% of the pulmonary hypertension patients.

Table 2. Comparison Of Mean Spirometric values in different grades of PHTN.

Grades of PHTN	FEV ₁ %	FVC %	FEV ₁ /FVC
1	52.14	64.07	68.18
2	37.71	53.43	68.86
3	26.50	40.0	65.50

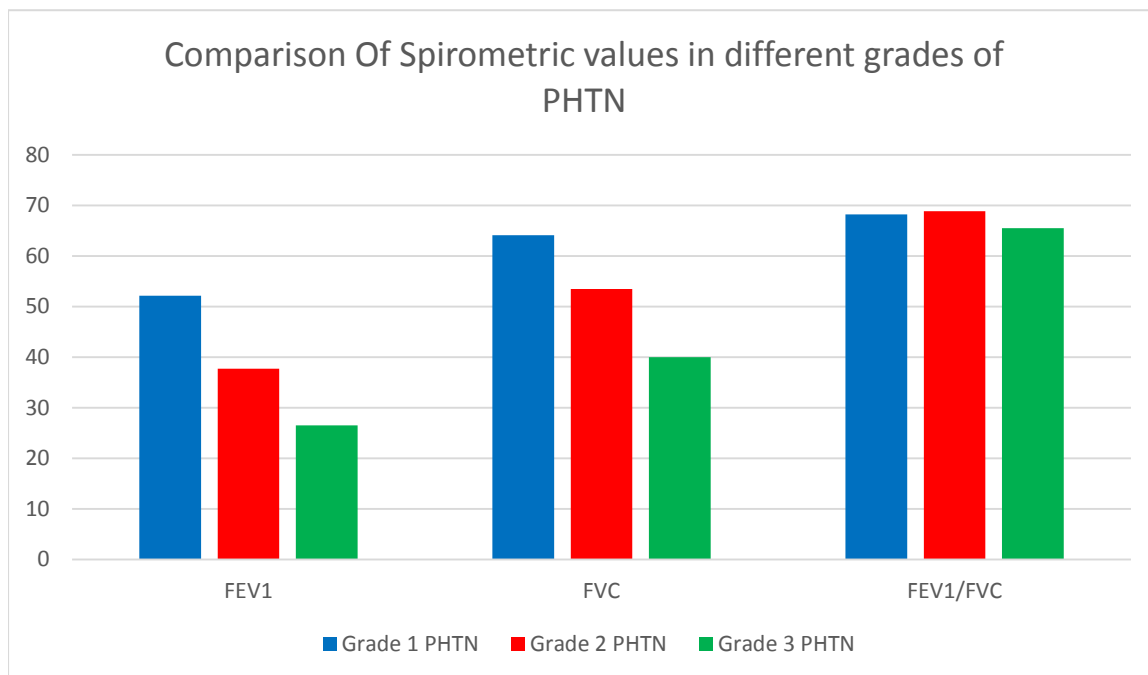


Figure 2 Comparison of Spirometric values in different grades of PHTN

In this study, as the severity of pulmonary hypertension increases the spirometric values goes on decreasing.

Table 3. Correlation of Pulmonary Hypertension with Forced Expiratory Volume at 1st second (FEV₁ %).

Correlations		
		PHTN (mPAP > 25 mmHg)
FEV ₁ %	Pearson Correlation	-0.443
	Sig. (2-tailed)	< 0.001
	N	50
**. Correlation is significant at the 0.01 level (2-tailed).		

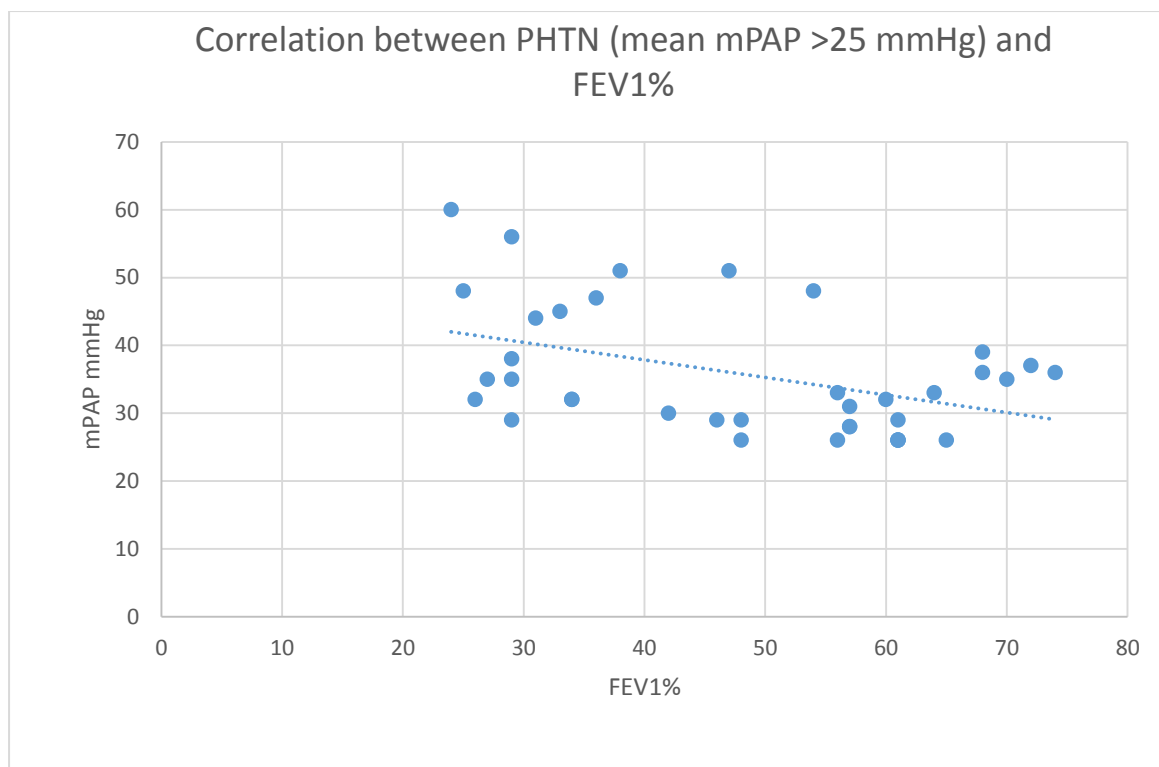


Figure 3 Correlation between PHTN (mean mPAP >25 mmHg) and FEV1%

In our study there is significant inverse correlation between FEV1 and PHTN (mPAP > 25 mmHg) using Pearson correlation (p value < 0.001), that is as FEV1 decreases, pulmonary hypertension increases.

Discussion

Pulmonary Hypertension in chronic respiratory diseases is a relatively common complication caused by complex pathophysiologic processes. In our study 200 Patients with chronic respiratory diseases were screened for evidence of Pulmonary Hypertension (PHTN). 66% out of these were males whereas the remaining 34% were females. As per the diagnosis, 40% were COPD, 25% Asthma, 15% Bronchiectasis, 12.5% ILD and 2.5% were Sarcoidosis, 2.5% Pneumoconiosis and 2.5% were Obstructive Sleep Apnea associated with COPD (OSA). Majority of the patients were in the age group of 51-60 years.

Out of 200 patients 37 i.e. 18% had Pulmonary hypertension. Pulmonary Hypertension secondary to chronic respiratory diseases had a male preponderance. In our study, 68 % of these patients were males and 32% were females. Based on 2D echo findings, Pulmonary Hypertension was classified as mild (grade 1), moderate (grade 2) and severe (grade 3), that is corresponding to mPAP values of 25-40, 41-55, >55 mm Hg respectively. Majority of the patients included in the study were classified as having Grade 1 Pulmonary Hypertension i.e 76%. 19% of patients were classified as Grade 2 and 5% were classified as Grade 3. As the Pulmonary Function parameters deteriorated the number of patients having Pulmonary Hypertension increased i.e. only 8% of patients with Pulmonary Hypertension had FEV₁% of more than 70% and 30% of patients with Pulmonary Hypertension had FEV₁% of less than 35%.

As Spirometry values deteriorated, there was an increase in the severity of pulmonary hypertension. Mean values of FEV₁ % decreased from 52% in grade1 to 38% in grade 2 and 27% in grade 3. The FVC% and FEV₁/FVC ratio also decreased with increasing severity of pulmonary hypertension. There is a significant inverse correlation in the FEV₁ % and pulmonary hypertension using Pearson correlation (p value <0.001). The results have been shown to be comparable with an earlier study conducted by Oswald-Mammosser M, Apprill M et al in 1991⁽⁵⁾; Thabut G, Dauriat G et al in 2005⁽⁶⁾ and Scharf S M, Iqbal M et al in 2002⁽⁷⁾ in which they showed that there is inverse correlation between FEV₁% and pulmonary hypertension.

In our study Spirometry values of FEV₁% increased from 48% to 51%, FVC increased from 61% to 64% and FEV₁/FVC improved from 68.16 to 68.89. Paired t test was used to evaluate the significance of improvement post treatment (p value was <0.05) i.e. there was significant improvement after treatment for 6 months. The

results are similar to a study conducted by Zisman DA, Schwarz M et al in 2010⁽⁸⁾, which showed improved oxygenation and lung functions with quality of life. In our study there was a significant positive correlation between FEV₁% and 6 MWD using Pearson correlation (p value <0.05), that is as FEV₁% increases, 6 MWD increases. The results are similar to a study done by Alhamad EH, Alanezi MO et al in 2009⁽⁹⁾, which showed significant correlation between 6 MWD and FEV₁%, FVC and TLCO. The prevalence of Pulmonary Hypertension secondary to COPD was found to be 20%. This prevalence is lower as compared to the studies conducted by Weitzenblum E, Hirth C et al in 1981⁽¹⁰⁾; who found prevalence of Pulmonary hypertension in COPD to be 35%. In their study pulmonary hypertension was defined as mPAP > 20 mm Hg, whereas in our study pulmonary hypertension was taken as mPAP > 25 mm Hg.

Conclusion.

The prevalence of Pulmonary Hypertension in males was greater than females. Pulmonary Hypertension was more common in older age groups. The severity of Pulmonary Hypertension increases with age. Mild Pulmonary Hypertension was the most common grade in chronic respiratory diseases. In our study based on 2D Echo findings the patients were divided into mild, moderate and severe Pulmonary Hypertension. 76% of the patients were having mild, 19% moderate and 5% severe Pulmonary Hypertension.

The Spirometry values declined with severity of pulmonary hypertension. As the Spirometry Parameters deteriorated the number of patients having Pulmonary Hypertension increases.

There was a significant inversely linear correlation between FEV₁ % and pulmonary hypertension. All patients were reevaluated after 6 months of therapy of the underlying

respiratory disease and of Pulmonary Hypertension in appropriate patients. They were evaluated with 2D echo, 6 MWT and Spirometry. The Spirometry, 6 MWT and 2D echo (Right Heart Changes and mPAP mmHg) parameters were found to improve significantly in entire study population.

Pulmonary Function Parameters and 6 Minute Walk Test can be used as prognostic markers in Pulmonary Hypertension.

References.

1. Hoepfer M.M.; The new definition of pulmonary hypertension. *Eur Respir J*.2009; 34: 790-791. 3
2. <http://www.who.int/classifications/icd10/>. November, 2010.
3. Khan MG. Pulmonary hypertension and cor pulmonale. In: Khan MG, Lynch JP III, eds. *Pulmonary Disease Diagnosis and Therapy: A Practical Approach*. Baltimore: Williams & Wilkins, 1997:603–616.
4. Pellegrino R, Viegi G, Brusasio V, et al : Interpretive Strategies for lung function test. *Eur Respir J* 26 : 948-968, 2005.
5. Oswald-Mammosser M, Apprill M, Bachez P, Ehrhart M, Weitzenblum E. Pulmonary hemodynamics in chronic obstructive pulmonary disease of the emphysematous type. *Respiration*. 1991; 58(5-6):304-10.
6. 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: 1531–1536.
7. Scharf S M, Iqbal M, Keller C, Criner G, Lee S, Fessler H E. Hemodynamic characterization of patients with severe emphysema. *Am J Respir Crit Care Med* 2002; 166: 314–322.
8. Zisman DA, Schwarz M, Anstrom KJ, et al. A controlled trial of sildenafil

- in advanced idiopathic pulmonary fibrosis. *N Engl J Med* 2010; 363:620–8.
9. Alhamad EH, Alanezi MO, Idrees MM, Chaudhry MK, AlShahraniAM, Isnani A, Shaikh S: Clinical characteristics and computed tomography findings in Arab patients diagnosed with pulmonary sarcoidosis. *Ann Saudi Med* 2009, 29(6):454-459.
10. Weitzenblum E, Hirth C, Ducolone A, Mirhom R, Rasaholinjanahary J, Ehrhart M. Prognostic value of pulmonary artery pressure in chronic obstructive pulmonary disease. *Thorax* 1981; 36: 752–758.