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A STUDY TO ASSESS SERUM C-REACTIVE PROTEIN AND PULMONARY ARTERIAL SYSTOLIC PRESSURE CHANGES BY ECHOCARDIOGRAPHY IN STABLE CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

Respiratory Medicine

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ABSTRACT

Introduction: Patients with chronic obstructive pulmonary disease (COPD) have an ongoing systemic inflammation, which can be assessed by measuring serum C-reactive protein (CRP). The presence of pulmonary hypertension (PH) increases mortality and predicts exacerbations in patients with COPD. **Aims & Objectives:** To determine the C-reactive protein (CRP) levels and to assess the pulmonary arterial systolic pressure (PASP) by Echocardiography in patients with stable COPD. **Materials and Methods:** The present study was a Cross sectional study. This study was conducted from April 2021 to September 2022 at Dept. of Respiratory Medicine N. R. S. Medical College & Hospital, Kolkata. 94 patients were included in this study. **Results:** The mean CRP of our study population was 5.30 ± 2.11. 74 (79%) patients of our study had pulmonary arterial hypertension with mean PASP of 37.12 ± 16.36. Association of airflow obstruction severity with pulmonary hypertension was statistically significant ($p<0.0001$). **Conclusion:** Increased serum CRP level is associated with lung inflammation in stable COPD. Airflow obstruction severity correlates with the pathogenesis of pulmonary hypertension in COPD.

KEYWORDS

Chronic Obstructive Pulmonary Disease, Cardiovascular Disease, Echocardiography and C-reactive protein.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction.[1]

The main environmental exposures leading to COPD are tobacco smoking and the inhalation of toxic particles and gases from household and outdoor air pollution, but other environmental[2] and host factors (including abnormal lung development and accelerated lung aging) can also contribute.[3]

A diagnosis of COPD should be considered in any patient who complains of dyspnea, chronic cough or sputum production, a history of recurrent lower respiratory tract infections and/or a history of exposure to risk factors for the disease (see above) but forced vital capacity maneuver during spirometry showing the presence of a post-bronchodilator FEV1/FVC < 0.7 is needed to establish the diagnosis of COPD. The FEV1 also serves to determine the severity of airflow obstruction.[4]

COPD is a multidimensional disease, with several systemic manifestations and associations with several comorbid diseases. The most likely link between COPD and these extrapulmonary conditions is spillover of inflammatory mediators from the lung.[5]

Several inflammatory cytokines, including tumor necrosis factor-α (TNFα),interleukin (IL)-6, CXCL8 (IL-8), and IL-18, and acute phase proteins, such as C-reactive protein (CRP), serum amyloid A, and fibrinogen, are increased within the circulation of patients with COPD.[6]

Serum CRP as a systemic marker of ongoing lung inflammation could be used as a predictor of future COPD outcomes.[7] Increased serum CRP level is also associated with lung inflammation in stable COPD.[6]

Pulmonary hypertension and consequent right ventricular failure, cor pulmonale, in COPD patients, are usually the consequence of chronic alveolar hypoxia, with secondary contributions from destruction of the alveolar capillary bed, lung hyperinflation, and increased blood viscosity.[8]

The presence of pulmonary hypertension (PH) and cor pulmonale increases mortality and predicts hospital readmission for exacerbations in patients with chronic obstructive pulmonary disease (COPD). The standard for measuring pulmonary pressures has been right heart catheterization (RHC), but this test is invasive and costly. Transthoracic Doppler echocardiography (DE) has become a common method to estimate pulmonary artery pressures noninvasively.[9]

AIMS AND OBJECTIVES

General Objectives

To determine the C-reactive protein (CRP) levels and to assess the pulmonary arterial systolic pressure (PASP) by Echocardiography in patients with stable COPD.

Specific Objective

1. To determine any correlation of deranged CRP with stable COPD
2. To assess the pulmonary arterial systolic pressure (PASP) by Echocardiography in stable COPD patients
3. To assess the prevalence of pulmonary hypertension in stable COPD patients.
4. Correlation between pulmonary hypertension and severity of COPD patient.

MATERIAL AND METHODS

A Single centered cross sectional observational study was performed from April 2021 to September 2022 at Department of Respiratory Medicine, N.R.S. Medical College and Hospital, Kolkata to determine the CRP levels and to assess the pulmonary arterial systolic pressure by Echocardiography in patients with stable COPD.

Inclusion Criteria

1. Diagnosed Stable COPD patients irrespective of age and sex.

Exclusion Criteria

1. COPD patients with history of exacerbation within last 3 months.
2. Patients with hemodynamic instability.
3. Patients with documented heart disease.
4. Patients refusing to give informed consent regarding participation in the study.

94 cases of stable COPD were included in this study after obtaining written informed consent. Spirometry was performed in all cases and severity was assessed as per Gold Criteria. Serum C-reactive protein (CRP) level was measured. Pulmonary arterial systolic pressure (PASP) was assessed by Echocardiography.

RESULTS

In our study, 23 (24.5%) patients were ≤50 years of age, 41 (43.6%) patients were 51-60 years of age, 30 (31.9%) patient were ≥61 years of age. The mean age of our study population was 56.12 ± 6.62 (Mean ± SD). The patient population consisted of 63 (67.0%) male and 31 (33.0%) female. The mean FEV1 of our study population was 55.80 ± 18.73 (Mean ± SD). The mean CRP of our study population was 5.30 ± 2.11 (Mean ± SD). 74 (79%) patients of our study had pulmonary hypertension. The patient characteristics are shown in Table 1.

Table 1: Patient Characteristics

Patient Characteristics	
Age (years, Mean ± SD)	56.12 ± 6.62
Sex [male/female; n, (%)]	63 (67.0%)/ 31 (33.0%)
FEV1, % predicted (Mean ± SD)	55.80 ± 18.73
CRP, mg/dl (Mean ± SD)	5.30 ± 2.11
Pulmonary Hypertension [Yes/No; n, (%)]	74 (79%) / 20 (21%)

Among 94 patients, 10 (10.6%) patients had mild airflow obstruction, 52 (55.3%) patients had moderate airflow obstruction, 20 (21.3%) patients had severe airflow obstruction and 12 (12.8%) patients had very severe airflow obstruction as per gold severity criteria. Figure 1 shows distribution of severity of airflow obstruction.

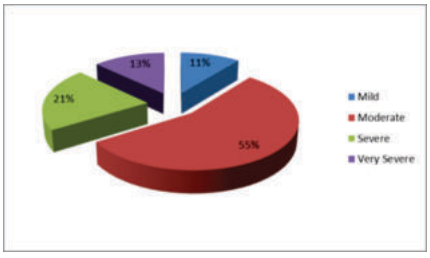

Figure 1

74 (79%) patients of our study had pulmonary arterial hypertension with mean PASP of 37.12 ± 16.36 (Mean ± SD).

The mean CRP of patients without pulmonary hypertension was 4.95 ± 1.95 and with pulmonary hypertension was 5.39 ± 2.16. Distribution of mean CRP with pulmonary hypertension was not statistically significant (p=0.4081). Figure 2 shows distribution of mean CRP with pulmonary hypertension.

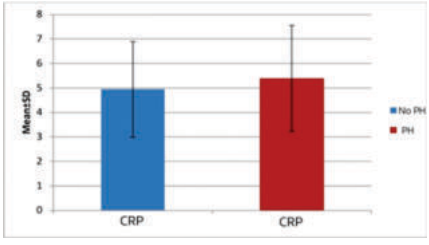

Figure 2

Among patients with pulmonary hypertension; 42 (56.8%) patients had moderate airflow obstruction, 20 (27.0%) had severe airflow obstruction and 12 (16.2) patients were very severe airflow obstruction. Among patients without pulmonary hypertension; 10 (50%) patients had mild airflow obstruction and 10 (50%) patients had moderate airflow obstruction. Association of airflow obstruction severity with pulmonary hypertension was statistically significant (p<0.0001). Figure 3 shows distribution of airflow obstruction severity with pulmonary hypertension.

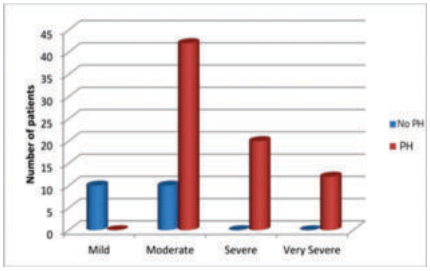

Figure 3

DISCUSSION

The present study was a Cross sectional study. This study was conducted from April 2021 to September 2022 at Dept. of Respiratory Medicine N. R. S. Medical College& Hospital, Kolkata. 94 patients were included in this study.

In our study, 23 (24.5%) patients were ≤50 years of age, 41 (43.6%) patients were 51-60 years of age, 30 (31.9%) patients were ≥61 years of age. The mean age of our study population was 56.12 ± 6.62 (Mean ± SD). The patient population consisted of 63 (67.0%) male and 31 (33.0%) female. The mean FEV1 of our study population was 55.80 ± 18.73 (Mean ± SD). The mean CRP of our study population was 5.30 ± 2.11 (Mean ± SD).

National Emphysema Treatment Trial[10] revealed mean age 66.4 ± 6.1 with male preponderance (61.2%). This study also revealed mean FEV1 26.9 ± 7.1.

Among 94 patients, 10 (10.6%) patients had mild airflow obstruction, 52 (55.3%) patients had moderate airflow obstruction, 20 (21.3%) patients had severe airflow obstruction and 12 (12.8%) patients had very severe airflow obstruction as per gold severity criteria.

74 (79%) patients of our study had pulmonary arterial hypertension with mean PASP of 37.12 ± 16.36 (Mean ± SD).

M.R. Fisher et al demonstrated mean pulmonary artery systolic pressure 35.7 ± 7.8 in their study.[9]

The mean CRP of patients without pulmonary hypertension was 4.95 ± 1.95 and with pulmonary hypertension was 5.39 ± 2.16. Distribution of mean CRP with pulmonary hypertension was not statistically significant (p=0.4081).

Gan and colleagues aggregated data from five cross-sectional studies and estimated an average mean increase in serum CRP of 1.85 mg/L in individuals with stable COPD.[6]

Morten Dahl et al revealed that increased serum CRP is a strong long-term predictor of COPD hospitalization and death, independent of smoking and lung function.[7]

Among patients with pulmonary hypertension; 42 (56.8%) patients had moderate airflow obstruction, 20 (27.0%) had severe airflow obstruction, 12 (16.2) patients were very severe airflow obstruction. Among patients without pulmonary hypertension; 10 (50%) patients had mild airflow obstruction and 10 (50%) patients had moderate airflow obstruction. Association of airflow obstruction severity with pulmonary hypertension was statistically significant (p<0.0001).

Schunemann HJ et al demonstrated that airflow limitation doubles the risk of cardiovascular mortality independent of smoking.[11]

He H et al showed that pulmonary hypertension (PH) is a common complication of chronic obstructive pulmonary disease (COPD). Although alveolar hypoxia is considered as a main cause of PH in COPD, structural and functional changes of pulmonary circulation are apparent at the initial stage of COPD. We hypothesized that an inflammatory response and oxidative stress might contribute to the formation of PH in COPD.[12]

Despite every sincere effort our study has limitations. The notable short comings of this study are small sample size and the study has been done in a single center.

CONCLUSION

Increased serum CRP level is associated with lung inflammation in stable COPD. Airflow obstruction severity correlates with the pathogenesis of pulmonary hypertension in COPD.

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