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# ORIGINAL ARTICLE

## Assessment of right ventricular changes in COPD patients and its correlation with severity of disease

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***ABSTRACT:***
**Background:** Chronic obstructive pulmonary disease (COPD) includes emphysema, an anatomically defined condition characterized by destruction and enlargement of the lung alveoli. The present study was conducted to assess right ventricular changes in COPD patients and its correlation with severity of disease. **Materials & Methods:** 65 patients of COPD of both genders. Pulmonary function tests were done in all patients and patients were graded according to the severity of COPD with guidelines given by Global initiative for obstructive lung disease (GOLD). **Results:** Severity of COPD was mild in 8, moderate in 12, severe in 20 and very severe in 25 cases. The difference was significant (P< 0.05). Echo findings such as cor pulmonale PAH, LVDD, RVSD, LVH correlated with disease severity (P< 0.05). **Conclusion:** Authors found that right ventricular changes in COPD patients correlated with severity of disease.
**Key words:** COPD, Pulmonary function, right ventricular.

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## INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by airflow limitation that is not fully reversible. Chronic obstructive pulmonary disease (COPD) includes emphysema, an anatomically defined condition characterized by destruction and enlargement of the lung alveoli; chronic bronchitis, a clinically defined condition with chronic cough and phlegm; and small airway disease, a condition in which small bronchioles are narrowed.[1]

Hypertrophy of the RV with preserved systolic function is the predominant effect of chronic pulmonary disease. Chronic pulmonary disease results in relatively slow increases in PA pressure (about 3 mm Hg/year), allowing time for adequate compensation.[2] The normally thin-walled, compliant RV is hypertrophied to mitigate intraluminal pressure increases, and ultimately minimizes wall stress. Increased RV thickness is accompanied by hypertrophy of individual myocytes, remodeling of the myocardial extracellular matrix, alterations in glucose metabolism, and in some models, compensatory increases in capillary density.[3]

Concentric RV hypertrophy can precede resting hypoxia in stable COPD patients, and has been demonstrated at autopsy in 76% of patients with advanced COPD. RV hypertrophy was estimated to be present in 50% of patients with restrictive lung disease. In OSA, one study showed that 71% of patients had RV hypertrophy by echocardiography. However, it remains unclear whether RV hypertrophy results from isolated OSA or other co-morbidities. While RV wall thickness was increased in subjects with SDB in the Framingham cohort, a more recent study of OSA patients without pulmonary disease or evidence of LV dysfunction showed no increase in RV wall thickness.[4] The present study was conducted to assess right ventricular changes in COPD patients and its correlation with severity of disease.

## MATERIALS & METHODS

The present study comprised of 65 patients of COPD of both genders. All were enrolled after obtaining their written consent.

Particulars of patients such as name, age, gender etc. was recorded. All patients were diagnosed by physical, radiographic and pulmonary function test (PFT). All patients were subjected to routine investigations such as complete blood count, lipid profile, blood sugar, blood urea, serum creatinine, electrocardiography. Pulmonary function tests were done in all patients and patients were graded according to the severity of COPD with guidelines given by Global initiative for obstructive lung disease (GOLD). The patients were subjected to radiological examination and 2D echo were done to assess the severity of right ventricular dysfunction. Results were clubbed and subjected to statistical analysis. P value less than 0.05 was considered significant.

## RESULTS

**Table I Distribution of patients**

| Total- 65 | | |
|---|---|---|
| **Gender** | **Males** | **Females** |
| Number | 35 | 30 |

Table I shows that out of 65 patients, males were 35 and females were 30.

**Table II Severity of COPD**

| Severity | Number | P value |
|---|---|---|
| Mild | 8 | 0.01 |
| Moderate | 12 | |
| Severe | 20 | |
| Very severe | 25 | |

Table II, graph I shows that severity of COPD was mild in 8, moderate in 12, severe in 20 and very severe in 25 cases. The difference was significant (P< 0.05).

**Graph I Severity of COPD**

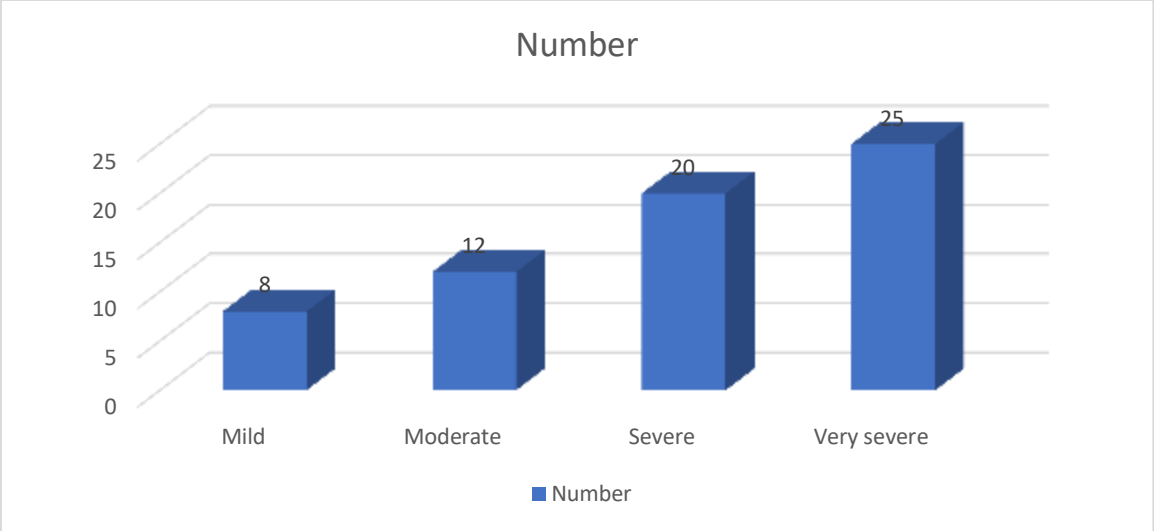


**Table III ECHO findings with disease severity**

| ECHO | Mild | Moderate | Severe | Very severe | P value |
|---|---|---|---|---|---|
| RA dilatation | 1 | 2 | 1 | 3 | 0.14 |
| RVH | 1 | 1 | 2 | 2 | 0.122 |
| Cor pulmonale | 2 | 3 | 3 | 6 | 0.05 |
| PAH | 2 | 3 | 0 | 7 | 0.02 |
| LVDD | 1 | 0 | 0 | 5 | 0.01 |
| RVSD | 1 | 1 | 4 | 1 | 0.05 |
| LVH | 0 | 2 | 10 | 1 | 0.02 |
| Total | 8 | 12 | 20 | 25 | |

Table III shows that echo findings such as cor pulmonale PAH, LVDD, RVSD, LVH correlated with disease severity (P< 0.05).

## DISCUSSION

Chronic obstructive pulmonary disease is one of the leading cause of chronic morbidity and mortality worldwide. It is seen that myocardial systolic function is generally preserved in PH associated with chronic lung disease. Although prior studies have demonstrated that slight reductions in RV ejection fraction occur commonly in chronic lung disease, the dependence of ejection fraction on ventricular pre-load, after-load, and myocardial contractility makes this observation difficult to interpret.[5] Intrinsic myocardial contractility appears to be preserved in COPD patients, as demonstrated by the normal RV end-systolic pressure-volume relationship measured at

rest and with exercise. RV diastolic function may be impaired in chronic lung disease patients with PH, as demonstrated by the direct associations between PH and reduced early to late ventricular filling velocity ratio (E/A ratio) and prolonged myocardial relaxation time in COPD patients. Impaired RV diastolic function can also be demonstrated in healthy individuals exposed to acute hypoxia.[6] The present study was conducted to assess right ventricular changes in COPD patients and its correlation with severity of disease.

In present study, out of 65 patients, males were 35 and females were 30. Severity of COPD was mild in 8, moderate in 12, severe in 20 and very severe in 25 cases. Jatav et al[7] in their study 100 patients of COPD fulfilling the inclusion criteria coming to OPD/wards of NMCH, Kota were recruited. They were staged by pulmonary function test (PFT) and evaluated by echocardiography. Statistical analysis of correlation was done with chi square test and statistical significance was taken ($p<0.05$).

We found that echo findings such as cor pulmonale PAH, LVDD, RVSD, LVH correlated with disease severity. COPD is a chronic inflammatory disorder of small airways that leads to airflow limitation, impaired gas exchange, and parenchymal loss in the case of emphysema. Hypoxemia results from impaired V/Q matching, and is compounded by the loss of alveolar surface area for diffusion in emphysema.[8] Interstitial lung diseases represent a more heterogeneous group of disorders, characterized by inflammatory or fibrotic destruction of lung parenchyma, generally at the level of the alveolar interstitium. These changes may be associated with autoimmune disease, exposure to cigarette smoke and other respiratory irritants, granulomatous diseases, or may be idiopathic. Interstitial changes result in impaired diffusing capacity, which may be exacerbated by poor V/Q matching in some patients. In OSA, hypoxia results from hypoventilation during obstructive episodes, and is by definition intermittent.[9] However, in patients with OHS or an overlap syndrome of COPD and OSA, diurnal hypoxemia may be present. At altitude, pulmonary vascular remodeling induced by chronic alveolar hypoxia can be exacerbated by hypoventilation, resulting in chronic mountain sickness, which is frequently associated with PH and right heart dysfunction.[10]

Another common factor in the increased PVR associated with chronic lung disease is disruption of pulmonary capillary beds. In COPD, loss of alveolated lung tissue, as occurs in emphysema, may result in loss of pulmonary capillary beds.[11] This is supported by the negative correlation observed between diffusing capacity for carbon monoxide ($D_LCO$) and mean PA pressure in patients with severe COPD. Mechanical compression of extra-alveolar vessels by lung hyperinflation had previously been assumed to contribute to PVR, though recent evidence does not support this hypothesis.[12]

## CONCLUSION

Authors found that right ventricular changes in COPD patients correlated with severity of disease.

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