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# Clinical and Demographic Correlates of Obstructive Sleep Apnea in Stable Chronic Obstructive Pulmonary Disease Patients - A Cross-Sectional Study

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## ABSTRACT

**Background:** COPD patients often experience disturbed breathing patterns, leading to nocturnal hypoxia and OSA. This study aimed to determine the prevalence of OSA in stable COPD patients and to identify its clinical, demographic and spirometric correlates.

**Methodology:** This cross-sectional study included fifty stable COPD patients. The assessments involved body measurements, spirometry to diagnose and assess COPD severity, Epworth sleepiness scale for daytime sleepiness, and polysomnography for diagnosing and evaluating the severity of OSA.

**Results:** Of the total patients, 22 (44%) were diagnosed with OSA with male predominance (59.09%). Patients with OSA had a significantly higher mean BMI (31.83 kg/m²) compared to those without OSA (24.1 kg/m²), highlighting obesity as a significant risk factor for OSA in COPD patients' Significant associations were found between OSA and hypertension (63.64%) and diabetes mellitus (31.82%), consistent with the impact of OSA on cardiovascular and metabolic health. Most patients with OSA had moderate to severe COPD (GOLD stage II and III), suggesting a correlation between COPD severity and the presence of OSA.

**Conclusions:** OSA was present in 44% of stable COPD patients, with a predominance of severe OSA cases. Higher BMI, larger neck and waist circumference, hypertension, diabetes mellitus, and higher ESS scores were more frequent among patients with OSA.

**Keywords:** COPD, OSA, Overlap syndrome, Spirometry, Polysomnography


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## INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung disorder characterized by chronic respiratory symptoms (dyspnea, cough, sputum production) due to abnormalities of the airways (bronchitis, bronchiolitis) or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction. The diagnosis relies on exposure history, symptomatology along with spirometric confirmation.[1] COPD patients often experience disturbed breathing during REM sleep due to reduced chest wall and intercostal muscle efficiency, leading to nocturnal hypoxia. Obstructive sleep apnea (OSA) is a common sleep related breathing disorder in COPD and its timely recognition is essential for optimizing management and improving patient outcomes. OSA is characterized by recurrent episodes of upper airway obstruction during sleep, leading to pauses in breathing (apneas) or significant reductions in airflow (hypopneas) subsequently leading to nocturnal hypoxemia.[2-4] Classical symptoms include excessive daytime sleepiness, snoring, decreased concentration, fatigability, nocturia, oesophageal reflux with and dry mouth.[5] Risk factors are both modifiable (obesity, smoking and alcohol abuse) and non-modifiable (male gender, advanced age, ethnicity, craniofacial abnormalities etc.). [6-8] Polysomnography (PSG) test remains the gold standard for diagnosing OSA, with severity classified by the Apnea-Hypopnea Index (AHI) as mild (5-15), moderate (15-30) and severe (more than 30 events/hour). [9]

The coexistence of OSA and COPD was first described by David C. Flenley as Overlap syndrome (OS) in year 1985 and is associated with marked nocturnal hypoxemia, hypercapnia, oxidative stress, and heightened sympathetic activity. It has been found to be more prevalent in airway-predominant phenotype of COPD and the underlying pathophysiological mechanisms over the time contribute to pulmonary hypertension, cardiovascular morbidity, and increased mortality in such patients. [10-13] Thus, early detection and management of OSA in patients of COPD helps to decrease morbidity and mortality, improving survival. [14] While global studies have reported variable prevalence, data among Indian COPD patient remain sparse, highlighting the need for region-specific research. This study aimed to determine the prevalence of OSA in stable COPD patients and to identify its clinical, demographic and spirometric correlates.

## MATERIALS AND METHODS

The study is a cross-sectional study was conducted in a tertiary care institute in North India over a one and half year's period. Stable COPD patients who presented to Pulmonary OPD were consecutively enrolled. The diagnosis of COPD was based on clinical symptoms along with spirometry findings as per the latest GOLD guidelines.[1]

The sample size was calculated based on the estimated prevalence of OSA among COPD patients. Using a 95% confidence level, 10% margin of error and an expected prevalence of 10.9% from previous study, [15] the minimum required sample size was calculated using the formula for estimating a proportion formula $n=Z^2pq/d^2$. The calculated sample size was 37. To account for potential non-response, dropouts, and incomplete investigations, an additional 20% was added to the calculated sample size. Finaly the sample size rounded to 50 patients.

Stable COPD patients of more than 18 years of age who were willing to participate were included in the study. Any patient with history of acute exacerbation of COPD in the preceding 6 weeks or COPD with other respiratory comorbidities like lung cancer, interstitial lung disease or pneumonia were excluded from the study. Patients with severe cardiac/hemodynamic compromise, or any other issue that may hinder participation of patient were also excluded.

All patients presenting to Pulmonary OPD who met the inclusion criteria and gave informed and written consent were enrolled for the study. Diagnosis of COPD and assessment of severity was done according to the recent GOLD. [1] Demographic and clinical details were recorded from each patient including their duration and type of symptoms, smoking/environmental exposures and comorbidities history of exacerbations and their current treatment. All patients underwent a routine spirometry to measure their lung functions in which parameters like FEV1/FVC, FEV1 and FVC was measured in accordance with the standard guidelines and Epworth sleepiness scale (ESS) score calculation was done.[1]

**Polysomnography:** After initial evaluation, all patients underwent a whole night polysomnography in the departmental sleep laboratory. The level 1 sleep study was performed using Compumedics E-series-44 Channel Polysomnography System. Physiologic sensor leads were placed on patient to record various parameters viz. brain electrical activity (EEG), eye movements (Oculogram), leg muscle movement, jaw muscle movements (EMG), airflow, respiratory efforts, oxygen saturation (SPO2) and ECG etc. Information gathered from all these leads were fed into a computer and recorded as a series of waveform tracings which could be interpreted on screen and on hard copy of the report. By studying each epoch, various sleep stages and abnormalities during sleep could be interpreted. An epoch is a convenient time interval usually equal to one page of recording and conventionally is of 30 seconds. Diagnosis of obstructive sleep apnea was based on accepted criterion of apnea/hypopnea index (AHI)> 05 on polysomnography as per the guidelines of American Academy of Sleep Medicine. [16] The AHI is total number of apneas/hypopneas per hour of sleep and an apneic event being defined as a cessation of oro-nasal

airflow for at least 10 seconds, while hypopnea as a 50% decrease in oro-nasal airflow, associated with oxyhaemoglobin desaturation of more than 3%. Depending upon the AHI, OSA is classified into mild (AHI 5-15), moderate (15-30) and severe (AHI >30). [15,16] Prevalence of OSA was calculated as total number of patients with AHI of more than and equal to 5 over the total COPD patients who underwent polysomnography. Thereafter, different clinical and demographic features was compared between COPD patients with and without OSA. Also, association of OSA with different COPD spirometry stages was evaluated.

The study protocol was approved by the Institutional Ethics committee. (Letter no. GMCH/IEC/776R/2022/167 dated 18/11/2022). Written informed consent was obtained from all the patients to participate.

**Statistical Analysis:** The prevalence was analysed across various parameters such as age, gender, etc. Continuous quantitative variables were expressed as mean ± SD or median, depending on data distribution. Group comparisons for continuous and categorical variables were performed using student T test/Mann-Whitney test and Chi square test/Fischer exact test, respectively. Correlation between AHI with FEV1 was assesses using Pearson correlation. P value <0.05 was considered significant for all statistical tests. Data analysis done by SPSS 25.0 software.

## RESULTS

The study participants ages ranged from 36 to 77 years with mean age of 59.3± 9.3. The mean age of patients with OSA was 59.5 ± 8.8 years, while those without OSA had a mean age of 59.1 ± 9.8 years, this difference was not statistically significant (p = 0.87). The total study population consisted of 38(76%) males & 12(24%) females. Among 22 OSA patients, 13(59.09%) were males & 9 (40.91%) were females and the distribution of two groups was statistically different (P = 0.02). The BMI of participants ranged from 16 kg/m2 to 51 kg/$m^2$, with an overall mean of 27.5 ± 7.03 kg/$m^2$, whereas, mean BMI among patients of OSA with COPD was 31.83 ± 7.82 kg/$m^2$. The distribution of BMI between the two groups was statistically different with P = 0.003. (Table 1)

**Table 1: Distribution of various demographic, anthropometric and clinical variables with or without OSA**

| Variables | With OSA (n=22) | Without OSA (n=28) | P value |
|---|---|---|---|
| **Age (years) (Mean ± SD)** | 59.55 ± 8.79 | 59.11 ± 9.79 | 0.87 |
| **Gender (n(%))** | | | |
| Female | 9 (40.91) | 3 (10.71) | 0.02 |
| Male | 13 (59.09) | 25 (89.29) | |
| **Weight (kg) (Mean ± SD)** | 84.41 ± 18.71 | 67.18 ± 12.52 | 0.0007 |
| **Height (cm) (Mean ± SD)** | 163.77 ± 8.72 | 166.82 ± 6.96 | 0.175 |
| **Body mass index (kg/$m^2$) (n(%))** | | | |
| <18.5 {Underweight} | 0 (0) | 3 (10.71) | 0.003 |
| 18.5 to 24.99{Normal BMI} | 5 (22.73) | 16 (57.14) | |
| 25 to 29.99 {Overweight} | 5 (22.73) | 7 (25) | |
| 30 to 34.99 {Obese class 1} | 6 (27.27) | 2 (7.14) | |
| 35 to 39.99 {Obese class 2} | 2 (9.09) | 0 (0) | |
| >=40 {Obese class 3} | 4 (18.18) | 0 (0) | |
| **BMI (Mean ± SD)** | 31.83 ± 7.82 | 24.1 ± 3.85 | 0.0002 |
| **Neck circumference(cm) (Mean ± SD)** | 36.77 ± 3.79 | 31.89 ± 4.21 | <.0001 |
| **Waist circumference (cm) (Mean ± SD)** | 108.64 ± 28.15 | 83.04 ± 12.23 | 0.0005 |
| **Hip circumference (cm) (Mean ± SD)** | 111.18 ± 29.6 | 88.04 ± 13.96 | 0.002 |
| **Waist Hip ratio** | 0.98 ± 0.13 | 0.94 ± 0.09 | 0.171 |
| **History of past illness (n(%))** | | | |
| Hypertension | 14 (63.64) | 6 (21.43) | 0.002 |
| Hypothyroid | 4 (18.18) | 1 (3.57) | 0.155 |
| Coronary artery disease | 2 (9.09) | 0 (0) | 0.189* |
| Previous history of COPD exacerbation | 11 (50) | 11 (39.29) | 0.449 |
| **Chief Complaints (n(%))** | | | |
| Cough | 2 (9.09) | 7 (25) | 0.266 |
| Dyspnea | 7 (31.82) | 5 (17.86) | 0.251 |
| Snoring | 16 (72.73) | 3 (10.71) | <.0001 |
| Tiredness | 8 (36.36) | 0 (0) | 0.0006 |
| Daytime sleepiness | 13 (59.09) | 1 (3.57) | <.0001 |
| Apnea | 2 (9.09) | 1 (3.57) | 0.576 |
| **ESS score (Mean ± SD)** | 7.68 ± 1.67 | 4.5 ± 1.75 | <.0001 |
| **GOLD stage (n(%))** | | | |
| I | 0 (0) | 4 (14.29) | <.0001 |
| II | 21 (95.45) | 11 (39.29) | |
| III | 0 (0) | 12 (42.86) | |
| IV | 1 (4.55) | 1 (3.57) | |

The neck circumference of the participants ranged from 24 to 44 cm, with mean of 42.98 ± 2.31 cm. Among OSA patients, the mean neck circumference was 34.04 ± 4.68cm showing a statistically significant difference between the two groups (p <0.05). Among the participants, waist circumference varied between 60 and 180 cm with an average measurement of 94.3 ± 24.23 cm. The distribution of waist circumference between 2 groups was statistically different with P <0.05. Among the participants, the waist hip ratio was observed to range from 0.76-1.25, with mean of 0.96 ± 0.11 and this distribution was statistically not different (P = 0.171). (Table 1)

There were different comorbid conditions in all the 50 patients taken. Among these patients, Chi - square test was used to identify the association of history of past illness with OSA. Out of 22 patients with OSA, hypertension was present in 14(63.64%), diabetes mellitus 7(31.82%), hypothyroid 4(18.18%), previous history of COPD exacerbation 11(50%), coronary artery disease 2(9.09%). Patient with past history of hypothyroidism, previous history exacerbation, coronary artery disease had no significant difference among both the groups with or without OSA with P >0.05. (Table 1)

There are variety of symptoms with which COPD patients present to hospital, such as cough, dyspnea, snoring, tiredness, daytime sleepiness etc. However, snoring (38%) was the most prevalent symptom presenting along with other complaints of COPD in our study population. Other presenting complaints were day time sleepiness (28%), dyspnea (24%), cough (18%), tiredness (16%) and apnea (6%). The symptoms of snoring, tiredness, and daytime sleepiness were significantly more common in patients with OSA (P <0.05). ESS ranging from 2-11 in 50 study population with mean of 5.9 ± 2.33. Among 22 patients with OSA, there was range 5-11 with mean of 7.68 ± 1.67. The distribution of ESS with or without OSA was statistically different (P <0.05). (Table 1)

## SPIROMETRY

**FEV1:** FEV1 ranged from 26-106% with mean of 61.58 ± 17.71 %with showing 21(95.45%) patients with GOLD 2 and 1 (4.55%) with GOLD 4 among 22 patients with OSA.

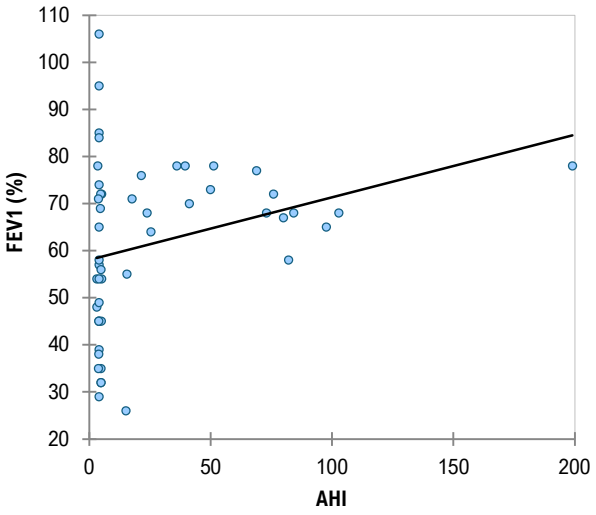


**Figure 1: Scatterplot of AHI with FEV1 (%)**

**Table 2: Multivariate logistic regression to find out independent predictors of OSA among COPD patients**

| Variables | Adjusted Odds ratio | 95% Confidence Interval | P value |
|---|---|---|---|
| **ESS score** | 1.399 | 0.701 - 2.789 | 0.341 |
| **Weight(kg)** | 0.928 | 0.795 - 1.084 | 0.347 |
| **Neck circumference(cm)** | 0.977 | 0.679 - 1.405 | 0.899 |
| **Waist circumference(cm)** | 1.039 | 0.916 - 1.179 | 0.552 |
| **Hip circumference(cm)** | 0.94 | 0.832 - 1.062 | 0.322 |
| **FEV1 (%)** | 1.007 | 0.89 - 1.139 | 0.914 |
| **Gender** | | - | |
| Female | 1 | - | |
| Male | 2.381 | 0.014 - 396.796 | 0.74 |
| **Snoring** | 2.626 | 0.133 - 51.842 | 0.526 |
| **Tiredness** | 0.805 | 0.013 - 48.414 | 0.917 |
| **Daytime illness** | 1.016 | 0.032 - 32.323 | 0.993 |
| **Hypertension** | 1.292 | 0.174 - 9.591 | 0.802 |
| **Diabetes mellitus** | 10.361 | 0.16 - 672.893 | 0.272 |
| **Smoker** | 0.214 | 0.004 - 11.432 | 0.448 |
| **Body mass index (kg/m²) distribution** | | - | |
| 18.5 to 24.99 {Normal BMI} | 1 | - | |
| <18.5 {Underweight} | 0.869 | 0.008 - 97.633 | 0.953 |
| 25 to 29.99 {Overweight} | 4.456 | 0.097 - 204.016 | 0.444 |
| 30 to 34.99 {Obese class 1} | 8.273 | 0.059 - 1160.461 | 0.402 |
| 35 to 39.99 {Obese class 2} | 79.066 | 0.009 - 724542.93 | 0.348 |
| >=40{Obese class 3} | 249.26 | 0.005 - 11778199 | 0.315 |
| **GOLD stage** | | - | |
| I | 1 | - | |
| II | 9.92 | 0.038 - 2599.704 | 0.786 |
| III | 1.813 | 0.001 - 3423.019 | 0.384 |
| IV | 22.666 | 0.002 - 232874.77 | 0.977 |

**POLYSOMNOGRAPHY:** AHI ranged from 3-199 with median AHI of 4.8(4-38.55). Among 22 patients of OSA, there are 3(13.64%) patients with mild OSA, 5(22.73%) with moderate OSA and 14(63.64%) with severe OSA.

The scatterplot in Figure 1 depicts the correlation between AHI and FEV1. Each plotted point corresponds to a single case, whereas the black trend line demonstrates the overall relationship between the two variables. The Pearson correlation between AHI with FEV1 is 0.281, having significant positive association between AHI and FEV1 with P= 0.04.

Multivariate logistic regression suggests no significant risk factors with P >0.05, depicting that there is no independent risk factor (Table 2).

## DISCUSSION

This comprehensive study endeavours to elucidate the prevalence of OSA among stable COPD patients and delve into its multifaceted associations with COPD severity, comorbidities, symptoms, and risk factors. In our study population, age was not significantly associated with OSA (p= 0.87), in contrast to prior studies where advancing age increased risk. [2] This may be explained by the relatively small sample size of our study. Male gender was significantly associated with OSA, consistent with findings observed in a study by Gallego et al, where they found that prevalence of OSA was higher in males than females. [14] This can be due to the anatomical differences in the upper airway and pharyngeal structures of the two.

Obesity related indices showed a strong relationship with OSA. In our study, OSA patients had significantly higher BMI and neck circumference than those without OSA, findings supported by other studies as well. [14,17] Obesity increases soft tissue deposition in the upper airway, predisposing to collapse during sleep. Our results reinforce the role of anthropometric measures, particularly BMI and neck circumference, as key screening markers for OSA in COPD patients. [18]

Comorbidities were frequent in our study participants. There was a significant association between hypertension and OSA, similar to findings by Georgine et al., and Pinto et al., who reported hypertension in 39% of OSA patients. [19,20] This link is likely mediated by sympathetic activation and RAAS upregulation secondary to intermittent hypoxia. [21] All patients with diabetes in our study also had OSA (p=0.002), supporting prior observations that overlap syndrome increases metabolic risk. The mechanism includes sympathetic activation and insulin resistance induced by sleep fragmentation. [22] In our study, there was no significant association between OSA and CAD (p = 0.189) or hypothyroidism (p = 0.155). The lack of significance may be attributed to the small number of cases in these subgroups, which limited statistical power. While OSA has been implicated in cardiovascular dysfunction through mechanisms such as negative intrathoracic pressure swings, sympathetic activation, and vascular remodeling, [23,24] our findings suggest that the relationship may not be evident in smaller samples. Similarly, although most of our hypothyroid patients also had OSA, the association was not statistically significant, in line with the observations from Turkan Mete et al. and another by Carratù P et al, who reported no consistent link between OSA and hypothyroidism. [25,26]

Subjective sleepiness, as measured by ESS, was significantly higher in OSA patients, consistent with previous studies. [27] This supports the clinical utility of ESS as a simple screening tool in COPD population, though it cannot replace polysomnography. 44% of COPD patients in our study had coexistent OSA, most of them with severe disease. This prevalence is comparable to previous reports ranging from 3-66%, with Soler et al. reporting 65.9% and Mohammad et al. 50% among COPD cohorts. [27,28] This much variation across the studies likely reflects differences in patient selection, COPD severity and diagnostic methods. Pulmonary function findings were noteworthy in our study. Patients with OSA-COPD overlap had better FEV1 than those with COPD alone. This has been observed previously also and hypothesized to reflect a protective effect of lung hyperinflation on airway collapsibility. [29,30] This counterintuitive observation warrants further mechanistic exploration. Importantly, no independent predictor of OSA were identified on multivariate analysis, again likely reflecting limited sample size. Despite this, the study provides valuable insight into the interplay between anthropometric, comorbid, and pulmonary variables in COPD patients with OSA.

The strengths of our study include systematic evaluation of multiple clinical and functional parameters in a relatively underexplored patient group, and use of polysomnography as the diagnostic standard. However, some limitations must be acknowledged in this study. The sample size was small, limiting statistical power. Only stable COPD patients were included, which may underestimate the true prevalence of OSA across the disease spectrum. The absence of a control group also precluded adjustment for confounders. Larger, multicenter studies are required to validate these findings and clarify mechanistic pathways.

## CONCLUSION

In conclusion, nearly half of our stable COPD patients had coexistent OSA, most with severe disease. Male gender, higher BMI, larger neck and waist circumference, hypertension, diabetes mellitus, and higher ESS scores were significant associations. The observation of better lung function among overlap patients highlights the complex interaction between COPD physiology and sleep-disordered breathing. These findings underscore the importance of systematic screening for OSA in COPD patients, particularly those

with obesity or cardiometabolic comorbidities, to enable earlier intervention and potentially improve long-term outcomes.

**Individual Author's Contribution: SB** and **KK** contributed to study design, data collection, analysis, and manuscript preparation. **CB** assisted in study design, data collection and manuscript preparation. **MKS** assisted in study design, data analysis and manuscript preparation. **DA** and **VS** contributed to study conception, study design, analysis, and manuscript preparation.

**Availability of data:** The data that support the findings of this study are available from the corresponding author on reasonable request.

**Declaration of Non-use of generative AI Tools:** This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and expertise.

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