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Original Research Article

PREVALENCE AND CLINICAL CORRELATES OF OBSTRUCTIVE SLEEP APNEA IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Vikas Jaiswal[1], *Davinder Kumar Kundra*[2]

[1]*HOD, Department of Pulmonology, Popular Group of Hospital, Varanasi, India.*
[2]*Consultant - Unit Head, Department of Pulmonology (Respiratory and Sleep Medicine), Manipal Hospital Dwarka, Delhi, India.*

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Corresponding Author:
Dr. Vikas Jaiswal,
HOD, Department of Pulmonology, Popular Group of Hospital, Varanasi, India.
Email: drvikasjai2286@gmail.com

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) and obstructive sleep apnea (OSA) frequently coexist, forming the overlap syndrome, which is associated with increased morbidity and adverse clinical outcomes. Data on the prevalence and clinical impact of co-existing OSA among COPD patients remain limited in the Indian population. This study aimed to determine the prevalence of OSA in patients with COPD and to evaluate its association with disease severity, clinical characteristics, and pulmonary function.

Materials and Methods: This hospital-based cross-sectional study included 436 adult patients with spirometry-confirmed COPD. All participants underwent detailed clinical and anthropometric assessment, pulmonary function testing, and overnight attended polysomnography. OSA was diagnosed using the apnoea–hypopnoea index (AHI), and its severity was classified according to standard criteria. COPD severity was categorized using Global Initiative for Chronic Obstructive Lung Disease (GOLD) spirometric staging. Comparisons were made between COPD patients with and without OSA, and associations between sleep-related parameters and pulmonary function indices were analyzed using appropriate statistical tests.

Results: OSA was diagnosed in 124 patients, yielding a prevalence of 28.4%. The prevalence of OSA increased significantly with worsening COPD severity, from 17.6% in mild COPD to 38.7% in very severe COPD ($p = 0.002$). Compared to patients without OSA, those with overlap syndrome were older and had significantly higher body mass index and neck circumference, greater daytime sleepiness, lower resting oxygen saturation, and a higher frequency of COPD exacerbations (all $p < 0.05$). The apnoea–hypopnoea index showed a significant negative correlation with FEV_1 percent predicted ($r = -0.42$) and resting oxygen saturation ($r = -0.46$), and a positive correlation with body mass index, neck circumference, and Epworth Sleepiness Scale score (all $p < 0.001$).

Conclusion: Co-existing obstructive sleep apnea is common among patients with chronic obstructive pulmonary disease and is associated with greater symptom burden, worse oxygenation, increased exacerbation frequency, and more severe airflow limitation. Routine screening for OSA should be considered in COPD patients, particularly those with advanced disease or obesity, to facilitate early diagnosis and integrated management aimed at improving clinical outcomes.

Keywords: Chronic obstructive pulmonary disease; Obstructive sleep apnea; Overlap syndrome; Polysomnography; Apnoea–hypopnoea index; GOLD staging.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and progressive respiratory disorder characterized by persistent airflow limitation and chronic inflammatory response of the airways to noxious particles or gases.[1] It represents a major global public health burden and is a leading cause of morbidity and mortality worldwide, with a particularly high prevalence in low- and middle-income countries, including India, largely due to tobacco smoking, biomass fuel exposure, and environmental pollution.[2,3] Patients with COPD often experience nocturnal symptoms such as cough, wheeze, and dyspnoea, along with sleep fragmentation and poor sleep quality.[3]

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by recurrent episodes of upper airway collapse during sleep, resulting in intermittent hypoxia, sleep fragmentation, and marked intrathoracic pressure swings.[4] OSA affects approximately 9–38% of the adult population globally, with increasing prevalence among older individuals, males, and those with obesity and metabolic comorbidities.[4] Untreated OSA is associated with significant cardiovascular, metabolic, and neurocognitive consequences.[4]

The coexistence of COPD and OSA in the same individual is referred to as the "overlap syndrome.[5]" This condition is not merely the sum of two diseases but represents a distinct clinical entity with unique pathophysiological interactions and adverse outcomes. Epidemiological studies suggest that OSA may be present in 10–30% of patients with COPD, although prevalence estimates vary widely depending on population characteristics, diagnostic criteria, and study design.[6,7] Despite this, overlap syndrome remains underdiagnosed, especially in resource-limited settings.[7]

Pathophysiologically, patients with overlap syndrome experience more profound nocturnal oxygen desaturation than those with either COPD or OSA alone. COPD-related ventilation–perfusion mismatch and reduced baseline oxygen reserves, when combined with recurrent apnoeic and hypopnoeic events of OSA, lead to sustained and intermittent hypoxaemia during sleep.[8] This augmented hypoxic burden promotes sympathetic overactivity, systemic inflammation, oxidative stress, and endothelial dysfunction, thereby accelerating the risk of pulmonary hypertension, right heart failure, arrhythmias, and cardiovascular morbidity.[8] Additionally, sleep fragmentation and chronic hypoxia may worsen daytime symptoms, reduce exercise tolerance, and impair quality of life in COPD patients.[9]

Clinically, the recognition of OSA in patients with COPD is challenging, as symptoms such as excessive daytime sleepiness may be less prominent, and nocturnal hypoxaemia is often attributed solely to underlying lung disease. Moreover, routine screening for sleep-disordered breathing is not commonly performed in stable COPD patients.[10] However, timely identification of overlap syndrome is crucial, as evidence suggests that appropriate treatment of OSA with continuous positive airway pressure (CPAP) therapy can significantly reduce nocturnal desaturation, frequency of COPD exacerbations, hospital admissions, and overall mortality in this population.[11]

Given the high burden of COPD in India and the rising prevalence of OSA, understanding the magnitude and clinical relevance of co-existing OSA among COPD patients is essential. There is limited Indian data systematically evaluating the prevalence, clinical characteristics, and sleep-related parameters of overlap syndrome.[7] So, this study aimed to determine the prevalence of OSA in patients with COPD and to evaluate its association with disease severity, clinical characteristics, and pulmonary function. It underscores the need for focused studies to identify OSA among COPD patients, facilitate early diagnosis, and guide integrated management strategies aimed at improving clinical outcomes and reducing long-term complications.

MATERIALS AND METHODS

Study design and setting

This hospital-based observational cross-sectional study was conducted in the Department of Pulmonary Medicine of a tertiary care teaching hospital in India. The study was carried out over a defined period of 24 months, from June 2022 to May 2024. The objective was to assess the prevalence of co-existing obstructive sleep apnea (OSA) among patients diagnosed with chronic obstructive pulmonary disease (COPD) and to evaluate their clinical, anthropometric, and polysomnographic characteristics.

Study population and sample size

Consecutive adult patients with a confirmed diagnosis of COPD attending the outpatient department or admitted to the respiratory wards during the study period were screened for eligibility. The sample size was calculated to estimate the prevalence of co-existing obstructive sleep apnea (OSA) among patients with chronic obstructive pulmonary disease (COPD). Based on previously published systematic review, the reported prevalence of overlap syndrome ranges from 10% to 30%.[11] Assuming an anticipated prevalence of 20% ($p = 0.20$), with a 95% confidence level ($Z = 1.96$) and an absolute precision (d) of 4%, the minimum required sample size was 384. After accounting for an anticipated non-response or incomplete data rate of 15%, the final sample size was inflated to 436 patients. Accordingly, a total of 436 patients

fulfilling the inclusion criteria were enrolled in the study after obtaining written informed consent.

Inclusion and exclusion criteria

Patients aged ≥40 years with a diagnosis of COPD, confirmed by post-bronchodilator spirometry demonstrating a forced expiratory volume in one second to forced vital capacity ratio (FEV_1/FVC) <0.70 as per Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, were included in the study. Both stable COPD patients and those evaluated at least six weeks after an acute exacerbation were eligible.

Patients with previously diagnosed or treated OSA, known neuromuscular disorders, interstitial lung disease, active pulmonary tuberculosis, unstable cardiovascular disease, chronic respiratory failure requiring long-term oxygen therapy, severe obesity (BMI ≥40 kg/m²), or use of sedative-hypnotic drugs were excluded. Patients unwilling to undergo overnight sleep study or unable to provide consent were also excluded.

Clinical and anthropometric assessment

Detailed demographic and clinical data were collected using a predesigned and pretested proforma. Information regarding age, sex, smoking history (pack-years), biomass fuel exposure, duration of COPD, history of exacerbations, comorbidities, and current medications was recorded. Anthropometric measurements included height, weight, body mass index (BMI), neck circumference, waist circumference, and waist-to-hip ratio, measured using standardized techniques.

All participants were evaluated for symptoms suggestive of sleep-disordered breathing, including loud snoring, witnessed apnoea, nocturnal choking, unrefreshing sleep, and excessive daytime sleepiness. Daytime sleepiness was quantified using the Epworth Sleepiness Scale (ESS), with a score >10 considered indicative of excessive daytime sleepiness.

Pulmonary function testing

Spirometry was performed in all participants using a calibrated computerized spirometer in accordance with American Thoracic Society/European Respiratory Society guidelines. Post-bronchodilator values were used for analysis. COPD severity was classified based on percent predicted FEV_1 into mild, moderate, severe, and very severe categories as per GOLD criteria. Oxygen saturation at rest was measured using pulse oximetry after at least five minutes of seated rest.

Sleep study and diagnosis of OSA

All enrolled patients underwent overnight attended polysomnography in the sleep laboratory. The study included continuous monitoring of electroencephalogram, electrooculogram, electromyogram, electrocardiogram, nasal and oral airflow, thoracoabdominal movements, oxygen saturation (SpO_2), body position, and snoring sounds. Sleep stages and respiratory events were scored manually by a trained sleep technician and reviewed by a pulmonologist experienced in sleep medicine, who was blinded to the spirometric severity of COPD.

Apnoea was defined as a complete cessation of airflow lasting at least 10 seconds, while hypopnoea was defined as a reduction in airflow of at least 30% lasting ≥10 seconds, associated with a ≥3% oxygen desaturation or arousal. The apnoea–hypopnoea index (AHI) was calculated as the number of apnoeas and hypopnoeas per hour of sleep. OSA was diagnosed when AHI was ≥5 events per hour in the presence of symptoms or ≥15 events per hour irrespective of symptoms. OSA severity was classified as mild (AHI 5–14.9), moderate (AHI 15–29.9), or severe (AHI ≥30 events/hour).

Definition of overlap syndrome

Patients with spirometry-confirmed COPD and polysomnography-confirmed OSA were classified as having COPD–OSA overlap syndrome. Comparisons were made between COPD patients with and without co-existing OSA to identify associated clinical and physiological factors.

Statistical analysis

Data were entered into a predesigned database and analyzed using Statistical Package for the Social Sciences (SPSS) software, version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation or median (interquartile range), depending on data distribution, while categorical variables were expressed as frequencies and percentages. Comparisons between COPD patients with and without obstructive sleep apnea were performed using the independent samples t-test for normally distributed continuous variables and the Mann–Whitney U test for non-normally distributed variables, while categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Trends in the prevalence of obstructive sleep apnea across GOLD stages were analyzed using the chi-square test for trend. Correlations between the apnoea–hypopnoea index and clinical, anthropometric, and spirometric parameters were assessed using Spearman's rank correlation coefficient (ρ). A p-value <0.05 was considered statistically significant.

Ethical considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment. Confidentiality of patient data was maintained throughout the study, and all procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

The study included 436 patients with spirometry-confirmed COPD, with a mean age of 58.9 ± 9.6 years, and nearly half (45.4%) aged ≥60 years. Males constituted 79.8% of the study population. The mean BMI was 24.8 ± 4.1 kg/m², with 44.0% of

patients being overweight (BMI ≥25 kg/m²). A history of smoking was present in 87.6% of participants, while 30.3% reported biomass fuel exposure. The median duration of COPD was 6 years (IQR 3–10), and 35.3% had experienced two or more exacerbations in the preceding year. The mean resting SpO_2 was 93.1 ± 2.6%, and excessive daytime sleepiness (ESS >10) was observed in 37.6% of patients. [Table 1]

Table 1: Baseline demographic and clinical characteristics of the study population (n = 436)

Variable	Frequency (%) / Mean ± SD / Median (IQR)
Age (years)	58.9 ± 9.6
Age group	
≥60 years	198 (45.4)
<60 years	238 (54.6)
Gender	
Male	348 (79.8)
Female	88 (20.2)
BMI (kg/m²)	24.8 ± 4.1
BMI category	
≥25 kg/m²	192 (44.0)
<25 kg/m²	244 (56.0)
Neck circumference (cm)	38.2 ± 3.4
Smoking status	
Current smokers	214 (49.1)
Ex-smokers	168 (38.5)
Never smokers	54 (12.4)
Biomass fuel exposure	132 (30.3)
Duration of COPD (years)	6 (3–10)
≥2 exacerbations in past year	154 (35.3)
Resting SpO_2 (%)	93.1 ± 2.6
Epworth Sleepiness Scale (ESS) score	9.4 ± 4.1
ESS category	
>10	164 (37.6)
≤10	272 (62.4)

BMI = body mass index; SpO_2 = peripheral oxygen saturation; ESS = Epworth Sleepiness Scale; SD = standard deviation; IQR = interquartile range.

Based on post-bronchodilator spirometry, the majority of patients had moderate (39.9%) or severe COPD (30.3%). Mild COPD was observed in 15.6% of participants, while 14.2% were classified as having very severe disease. This distribution reflects a predominance of moderate-to-severe airflow limitation in the hospital-based cohort. [Table 2]

Table 2: Distribution of COPD severity according to GOLD spirometric classification

GOLD stage	Frequency (%)
Mild (FEV_1 ≥80%)	68 (15.6)
Moderate (FEV_1 50–79%)	174 (39.9)
Severe (FEV_1 30–49%)	132 (30.3)
Very severe (FEV_1 <30%)	62 (14.2)

GOLD = Global Initiative for Chronic Obstructive Lung Disease; FEV_1 = forced expiratory volume in one second.

OSA (AHI ≥5 events/hour) was diagnosed in 124 patients, yielding a prevalence of 28.4%. Among these, mild OSA was present in 11.9%, moderate OSA in 10.1%, and severe OSA in 6.4% of the total cohort. The mean AHI among patients with OSA was 17.6 ± 12.9 events/hour. Significant nocturnal hypoxaemia was observed, with a mean minimum SpO_2 of 78.6 ± 8.1% and a median time spent with SpO_2 <90% of 18.4% (IQR 9.2–32.6) of total sleep time. [Table 3]

Table 3: Prevalence and severity of obstructive sleep apnea among COPD patients (n = 436)

Variable	Frequency (%)/mean ± SD/median (IQR)
OSA present (AHI ≥5/hr)	124 (28.4)
Mild OSA (AHI 5–14.9)	52 (11.9)
Moderate OSA (AHI 15–29.9)	44 (10.1)
Severe OSA (AHI ≥30)	28 (6.4)
Mean AHI (events/hr)	17.6 ± 12.9
Minimum nocturnal SpO_2 (%)	78.6 ± 8.1
Time SpO_2 <90% (% of TST)	18.4 (9.2–32.6)

OSA = obstructive sleep apnea; AHI = apnoea–hypopnoea index; SpO_2 = oxygen saturation; TST = total sleep time.

Compared to patients without OSA, those with co-existing OSA were significantly older (61.2 ± 8.4 vs. 58.0 ± 9.9 years; p = 0.003) and more likely to be male (87.1% vs. 76.9%; p = 0.02). Patients with overlap syndrome had higher BMI (26.7 ± 3.9 vs. 24.1 ± 4.0 kg/m²; p <0.001) and larger neck circumference (40.1 ± 3.1 vs. 37.4 ± 3.2 cm; p <0.001). Excessive daytime sleepiness was more pronounced in the OSA group, reflected by higher ESS scores (12.6 ± 4.2 vs. 8.1 ± 3.5; p <0.001). Additionally, patients with OSA had a higher frequency of ≥2 COPD exacerbations in the previous year (45.2% vs. 31.4%; p = 0.007) and lower resting SpO_2 levels (92.1 ± 2.9% vs. 93.5 ± 2.4%; p <0.001). [Table 4]

Table 4: Comparison of clinical and physiological characteristics between COPD patients with and without OSA

Variable	COPD with OSA (n = 124)	COPD without OSA (n = 312)	p-value
	Frequency (%)/mean ± SD		
Age (years)	61.2 ± 8.4	58.0 ± 9.9	0.003
Gender			
Male	108 (87.1)	240 (76.9)	0.02
Female	16 (12.9)	72 (23.1)	
BMI (kg/m²)	26.7 ± 3.9	24.1 ± 4.0	<0.001
Neck circumference (cm)	40.1 ± 3.1	37.4 ± 3.2	<0.001
Epworth Sleepiness Scale (ESS) score	12.6 ± 4.2	8.1 ± 3.5	<0.001
Exacerbations per year			
≥2	56 (45.2)	98 (31.4)	0.007
<2	68 (54.8)	214 (68.6)	
Resting SpO_2 (%)	92.1 ± 2.9	93.5 ± 2.4	<0.001

BMI = body mass index; ESS = Epworth Sleepiness Scale; SpO_2 = oxygen saturation; SD = standard deviation.

The prevalence of OSA increased progressively with worsening COPD severity. OSA was present in 17.6% of patients with mild COPD, 24.1% with moderate COPD, 34.8% with severe COPD, and 38.7% with very severe COPD. Trend analysis demonstrated a statistically significant increase in OSA prevalence with advancing GOLD stage (χ^2 for trend, p = 0.002). [Table 5]

Table 5: Association between COPD severity and prevalence of obstructive sleep apnea

GOLD stage	OSA present	OSA absent	p-value
	Frequency (%)		
Mild (FEV_1 ≥80%)	12/68 (17.6%)	56/68 (82.4%)	0.002
Moderate (FEV_1 50–79%)	42/174 (24.1%)	132/174 (75.9%)	
Severe (FEV_1 30–49%)	46/132 (34.8%)	86/132 (65.2%)	
Very severe (FEV_1 <30%)	24/62 (38.7%)	38/62 (61.3%)	

GOLD = Global Initiative for Chronic Obstructive Lung Disease; χ^2 = chi-square test.

AHI showed a significant negative correlation with FEV_1 percent predicted (r = −0.42; p <0.001) and resting SpO_2 (r = −0.46; p <0.001), indicating worsening sleep-disordered breathing with declining pulmonary function. Positive correlations were observed between AHI and BMI (r = 0.51), neck circumference (r = 0.48), and ESS score (r = 0.57), all of which were statistically significant (p <0.001). [Table 6]

Table 6: Correlation between sleep-related parameters and pulmonary function indices

Parameter	AHI (r)	p-value
FEV_1 % predicted	−0.42	<0.001
Resting SpO_2 (%)	−0.46	<0.001
BMI (kg/m²)	0.51	<0.001
Neck circumference (cm)	0.48	<0.001
ESS score	0.57	<0.001

AHI = apnoea–hypopnoea index; FEV_1 = forced expiratory volume in one second; SpO_2 = oxygen saturation; ESS = Epworth Sleepiness Scale.

DISCUSSION

In the present hospital-based cross-sectional study, the prevalence of co-existing obstructive sleep apnea (OSA) among patients with chronic obstructive pulmonary disease (COPD) was 28.4%, confirming that overlap syndrome is a frequent clinical entity rather than an incidental association. This prevalence is comparable to that reported in earlier international studies by Sami et al., and Fanaridis et al., which have documented OSA in 10–30% of COPD patients using polysomnography-based diagnosis.[12,13] Indian hospital-based studies by Deshmukh et al., and Sanjay et al., have similarly reported prevalence ranging from 20% to 35%, particularly in cohorts with moderate-to-severe COPD, suggesting that overlap syndrome may be under-recognized in routine clinical practice.[14,15]

A significant and clinically important finding of this study is the stepwise increase in OSA prevalence

with worsening COPD severity, rising from 17.6% in mild COPD to 38.7% in very severe COPD. This observation is consistent with prior studies that have demonstrated higher OSA prevalence in patients with advanced airflow limitation.[14,15] McNicholas et al. reported that severe COPD patients exhibit greater upper airway collapsibility during sleep due to lung hyperinflation and reduced caudal traction, thereby predisposing them to obstructive events.[16] Our findings reinforce this mechanistic explanation and provide quantitative evidence from an Indian cohort.[15]

Patients with overlap syndrome in the present study were older, predominantly male, and had significantly higher BMI and neck circumference compared to COPD patients without OSA. Similar demographic and anthropometric associations have been reported consistently across studies by Gunduz et al., and Sarawade et al.[17,18] Notably, although the mean BMI in our overlap group (26.7 kg/m²) would be considered only mildly elevated by Western standards, it was strongly associated with OSA severity, highlighting the relevance of lower BMI thresholds for OSA risk in Indian populations.[15,19] The strong positive correlations observed between AHI and BMI ($r = 0.51$) as well as neck circumference ($r = 0.48$) further substantiate the role of upper airway anatomy and adiposity in the pathogenesis of overlap syndrome.[19]

Excessive daytime sleepiness was significantly more pronounced in patients with overlap syndrome, reflected by higher Epworth Sleepiness Scale (ESS) scores, and ESS showed the strongest correlation with AHI ($r = 0.57$). This finding is in agreement with previous studies Lan et al., and Sartori et al., suggesting that sleep fragmentation and intermittent hypoxia due to OSA contribute substantially to symptom burden in COPD patients.[20,21]

However, several studies by Sartori et al., and Jain et al., have noted that daytime sleepiness may be underreported in COPD due to attribution of fatigue to respiratory disease alone.[21,22] The present findings emphasize the importance of systematic sleep-related symptom screening in COPD clinics.

Importantly, overlap syndrome was associated with worse resting oxygenation and a higher frequency of COPD exacerbations. Nearly 45% of patients with OSA experienced two or more exacerbations per year, compared to 31% in those without OSA ($p = 0.007$). This association has been previously documented by Czerwaty et al., who reported a significantly increased risk of COPD-related hospitalizations among patients with untreated OSA.[23] The underlying mechanisms include repetitive nocturnal hypoxaemia, heightened sympathetic activity, oxidative stress, and systemic inflammation, all of which may increase susceptibility to exacerbations and accelerate disease progression.[24,25]

Correlation analysis in this study demonstrated a moderate negative correlation between AHI and FEV_1 percent predicted ($r = -0.42$) and resting SpO_2 ($r = -0.46$), indicating that worsening pulmonary function is associated with increasing severity of sleep-disordered breathing. Similar inverse relationships between lung function and AHI have been reported in earlier studies by Tondo et al., and Zhu et al., supporting the concept that declining ventilatory reserve and impaired gas exchange amplify nocturnal respiratory instability in overlap syndrome.[26,27] These findings are clinically significant, as combined nocturnal and daytime hypoxaemia has been shown to increase the risk of pulmonary hypertension, right ventricular dysfunction, and cardiovascular mortality in overlap syndrome patients.[28]

Overall, the present study provides robust evidence that COPD–OSA overlap syndrome is associated with greater symptom burden, worse oxygenation, increased exacerbation frequency, and more severe physiological impairment compared to COPD alone. Consistent with prior longitudinal studies, these findings underscore the importance of early identification and treatment of OSA, as continuous positive airway pressure (CPAP) therapy has been shown to reduce exacerbations, hospitalizations, and mortality in overlap syndrome.[29,30] Routine screening for OSA should therefore be considered in COPD patients, particularly those with advanced disease, obesity, frequent exacerbations, or unexplained nocturnal desaturation.

Limitations

This study has certain limitations that should be acknowledged. First, the cross-sectional design precludes establishing a causal relationship between chronic obstructive pulmonary disease severity and the development of obstructive sleep apnea. Although significant associations were observed between airflow limitation, anthropometric parameters, and sleep-disordered breathing, longitudinal studies are required to clarify temporal relationships and disease progression. Second, as this was a single-center, tertiary care hospital–based study, the findings may not be fully generalizable to the broader community, particularly to patients with milder COPD who may not seek hospital-based care. Third, although attended polysomnography was used for the diagnosis of OSA, night-to-night variability in sleep parameters could not be accounted for, as only a single-night study was performed. Fourth, information on exacerbation frequency and symptom history was partly based on patient recall, which may be subject to recall bias. Fifth, while several important confounders were evaluated, multivariable regression analysis was not performed, limiting the ability to identify independent predictors of overlap syndrome after adjusting for potential confounders such as age, sex, obesity, and smoking status. Finally, cardiovascular outcomes, pulmonary hypertension, and long-term mortality were not assessed, and the impact of OSA treatment, particularly continuous positive airway pressure therapy, could not be evaluated. These

outcomes warrant further investigation through prospective interventional studies.

CONCLUSION

The present study demonstrates that co-existing obstructive sleep apnea is highly prevalent among patients with chronic obstructive pulmonary disease, affecting nearly one-third of the study population. The prevalence of OSA increased significantly with worsening COPD severity, and overlap syndrome was associated with older age, male gender, higher body mass index, increased neck circumference, greater daytime sleepiness, lower oxygen saturation, and a higher frequency of COPD exacerbations. The observed correlations between apnoea–hypopnoea index and pulmonary function parameters highlight the compounded physiological burden imposed by the coexistence of these two disorders. These findings emphasize that COPD–OSA overlap syndrome represents a clinically important and potentially modifiable condition rather than a coincidental coexistence. Given the availability of effective therapeutic interventions for OSA, routine screening for sleep-disordered breathing should be considered in COPD patients, particularly those with advanced disease, obesity, frequent exacerbations, or unexplained hypoxaemia. Early diagnosis and integrated management of overlap syndrome may lead to improved symptom control, reduced exacerbation burden, and better long-term outcomes in this high-risk population.

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