



ORIGINAL ARTICLE

**Prevalence of Sleep Related Breathing Disorders (SRBD) and the Assessment of Quality of Sleep Among Patients with Chronic Obstructive Pulmonary Disease (COPD): An Analytical Cross-Sectional Study**

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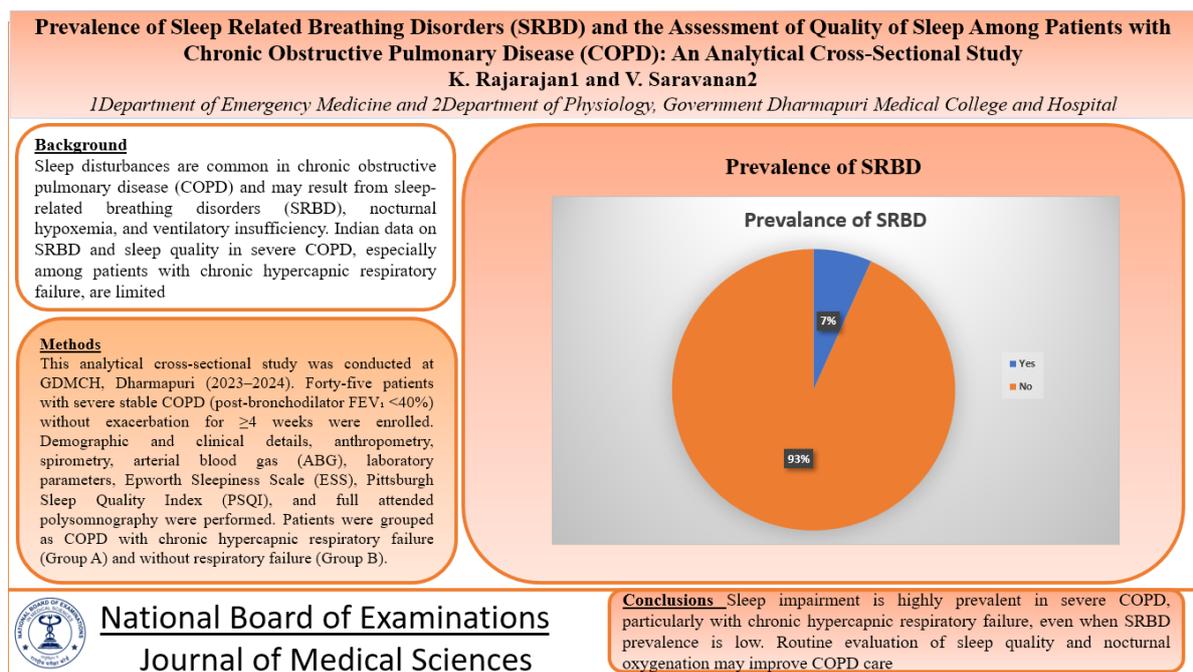
**Abstract**

**Background:** Sleep disturbances are common in chronic obstructive pulmonary disease (COPD) and may result from sleep-related breathing disorders (SRBD), nocturnal hypoxemia, and ventilatory insufficiency. Indian data on SRBD and sleep quality in severe COPD, especially among patients with chronic hypercapnic respiratory failure, are limited. **Objectives:** To estimate the prevalence and risk factors of SRBD in COPD, assess sleep quality, and compare sleep parameters between COPD patients with and without chronic hypercapnic respiratory failure. **Methods:** This analytical cross-sectional study was conducted at GDMCH, Dharmapuri (2023–2024). Forty-five patients with severe stable COPD (post-bronchodilator FEV<sub>1</sub> <40%) without exacerbation for ≥4 weeks were enrolled. Demographic and clinical details, anthropometry, spirometry, arterial blood gas (ABG), laboratory parameters, Epworth Sleepiness Scale (ESS), Pittsburgh Sleep Quality Index (PSQI), and full attended polysomnography were performed. Patients were grouped as COPD with chronic hypercapnic respiratory failure (Group A) and without respiratory failure (Group B). **Results:** The mean age was 57.38 ± 7.48 years and 82.2% were males. SRBD prevalence was low, with obstructive sleep apnea in 6.67% (mean AHI 3.33 ± 3.75). Poor sleep quality was common (PSQI 10.07 ± 3.52) with excessive daytime sleepiness (ESS 11.93 ± 5.34). Polysomnography revealed reduced sleep efficiency (64.15 ± 9.10%), shortened total sleep time (264.71 ± 51.68 min), and elevated arousal index (25.39 ± 7.74/h). Group A (n=32) had significantly worse ABG parameters and poorer sleep indices and nocturnal oxygenation than Group B (n=13). **Conclusion:** Sleep impairment is highly prevalent in severe COPD, particularly with chronic hypercapnic respiratory failure, even when SRBD prevalence is low. Routine evaluation of sleep quality and nocturnal oxygenation may improve COPD care.

**Keywords:** COPD, Sleep quality, Sleep-related breathing disorders, Polysomnography, Chronic hypercapnic respiratory failure

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## Graphical Abstract



### Background

Chronic obstructive pulmonary disease (COPD) represents a major and growing global health challenge, characterised by persistent airflow limitation and chronic respiratory symptoms. COPD contributes substantially to morbidity, mortality, and healthcare burden worldwide and remains one of the leading causes of death and disability-adjusted life years (DALYs) lost globally [1].

In India, the burden of COPD is substantial and under-recognised. Nationwide systematic reviews indicate that the prevalence of COPD among adults ranges between approximately 7% and 13%, with significant variation by study setting, smoking status, age, and exposure to biomass fuel smoke. Meta-analyses of spirometry-based studies among Indian adults demonstrate a pooled prevalence around 13%, highlighting COPD as a common chronic respiratory disease in the Indian population [2].

India's high COPD burden is driven by a combination of traditional risk factors — tobacco smoking, exposure to biomass fuel smoke, air pollution, and occupational dust — as well as non-traditional contributors including prior pulmonary tuberculosis and childhood respiratory infections. These risk factors, prevalent in both urban and rural settings, contribute to high disease prevalence and high DALY loss, underscoring the need for public health prioritisation [3].

In Tamil Nadu, community-based studies also show a high COPD prevalence, with rural populations demonstrating rates exceeding 20% when evaluated using peak flow measures. These local patterns reflect broader regional trends of COPD associated with increasing age, male sex, smoking history, and environmental exposures such as biomass fuel use [4].

Sleep is a vulnerable physiologic state for individuals with COPD. Normal sleep physiology involves reductions in ventilatory drive, reduced respiratory

muscle activity, and increased upper airway resistance, all of which may exacerbate nocturnal hypoxaemia and respiratory instability in COPD patients. Sleep-associated changes in ventilation disproportionately affect patients with COPD, leading to nocturnal hypoventilation, frequent arousals, and fragmented sleep architecture [5].

Sleep-related breathing disorders (SRBD) — including obstructive sleep apnea (OSA), hypoxia events, and sustained nocturnal desaturation — are frequently observed in COPD populations. Recent meta-analytic evidence suggests that OSA and related sleep disorders affect approximately 29% of COPD patients, with a significant portion also experiencing restless legs syndrome and insomnia. These co-morbid sleep disorders contribute to impaired gas exchange, increased symptom burden, and, in overlap syndromes, worse clinical outcomes than COPD alone [6].

Moreover, sleep disturbances among COPD patients extend beyond SRBDs. Studies consistently demonstrate that a large proportion of COPD patients report poor sleep quality, characterised by prolonged sleep latency, reduced sleep efficiency, frequent arousals, and nocturnal symptoms such as dyspnoea or coughing that fragment sleep. These sleep disruptions are multifactorial, related both to underlying pulmonary pathology and to associated co-morbidities such as anxiety and depression [7].

The impact of poor sleep quality in COPD is clinically meaningful. Beyond subjective complaints, impaired sleep has been linked with worsened daytime function, reduced health-related quality of life, higher rates of exacerbations, and increased healthcare utilisation. Recognising and quantifying sleep quality

and SRBD in COPD patients is therefore essential for comprehensive disease management [8].

Despite this evidence, systematic sleep evaluation — particularly with polysomnography — remains under-utilised in routine COPD care, especially in low- and middle-income settings. This study aims to address these gaps by estimating the prevalence of SRBD and assessing sleep quality among patients with severe stable COPD in a tertiary care setting in India, and by comparing sleep parameters between those with and without chronic hypercapnic respiratory failure.

### **Objectives**

1. To estimate the prevalence of Sleep Related Breathing Disorders (SRBD) and its risk factors among patients with COPD
2. To assess the quality of sleep among patients with COPD.
3. To compare the sleep parameters between patients with and without Chronic Hypercapnic Respiratory Failure

### **Methodology**

This analytical cross-sectional study was conducted at a tertiary care hospital in Dharmapuri, Tamil Nadu, India, between 2023 and 2024 among patients with Chronic Obstructive Pulmonary Disease (COPD) attending regular follow-up. Patients with a clinical history consistent with severe stable COPD and no exacerbation of airway disease for at least four weeks prior to evaluation were considered eligible. Individuals aged more than 80 years, those with known cardiac, hepatic, or renal diseases, and patients with respiratory acidosis were excluded to avoid confounding effects on gas exchange and

sleep parameters. Patients diagnosed with COPD or COPD with pulmonary tuberculosis sequelae were consecutively screened, and those demonstrating severe airflow limitation (post-bronchodilator  $FEV_1 < 40\%$ ) on spirometry were invited to participate. 45 study participants were recruited by purposive sampling and written informed consent was obtained from all eligible participants before enrolment.

All enrolled patients were admitted and underwent detailed clinical evaluation including comprehensive history and physical examination. Anthropometric measurements such as height, weight, body mass index (BMI), neck circumference, and waist circumference were recorded using standard methods. Vital parameters including respiratory rate, pulse rate, blood pressure, and daytime peripheral oxygen saturation ( $SpO_2$ ) were measured at rest. Arterial Blood Gas (ABG) analysis was performed to assess gas exchange status. Laboratory investigations including fasting and postprandial blood glucose, blood urea, serum creatinine, and liver function tests were conducted to exclude systemic comorbidities. Subjective sleep assessment was carried out using validated questionnaires, namely the Epworth Sleepiness Scale (ESS) for daytime sleepiness and the Pittsburgh Sleep Quality Index (PSQI) for sleep quality.

Overnight full-attended polysomnography (PSG) was performed for a minimum recording duration of six hours. Patients were instructed to retire to bed approximately one hour before their usual sleep time, and recordings were initiated at habitual lights-off and terminated upon spontaneous awakening.

The monitored parameters included electroencephalography (EEG), bilateral electro-oculography (EOG), thoracoabdominal movements using inductance bands, nasal airflow via pressure cannula, body position, leg movements, and continuous arterial oxygen saturation. Sleep staging and respiratory events were scored according to the American Academy of Sleep Medicine (AASM) criteria. The derived variables included Total Bed Time (TBT), Total Sleep Time (TST), sleep latency, sleep efficiency, sleep stages (in minutes and percentage of TST), arousal index, apnea-hypopnea index (AHI), mean and minimal nocturnal oxygen saturation, and the presence of nocturnal desaturation.

## Results

### *Study Population and Baseline Characteristics*

The study included a total of 45 patients with Chronic Obstructive Pulmonary Disease (COPD). The mean age of the study population was  $57.38 \pm 7.48$  years, ranging from 39 to 72 years. There was a significant male predominance, with 37 (82.2%) males and 8 (17.8%) females.

Regarding lifestyle factors and comorbidities, the mean smoking history was  $29.09 \pm 19.09$  pack years. Alcohol consumption was reported by 22.2% of the participants. Diabetes was present in 17.8% of the cohort. Anthropometric measurements revealed a mean BMI of  $21.83 \pm 3.14$   $kg/m^2$ , a mean neck circumference of  $33.76 \pm 2.96$  cm, and a mean waist circumference of  $80.20 \pm 8.19$  cm. Table 1 shows the distribution of baseline characters of the study sample.

Table 1. Baseline Characters

Variable		Distribution	
Age		57.38 $\pm$ 7.5 years	
Gender	Male	37	82.2 %
	Female	8	17.8 %
Smoking pack years		29.09 + 19.1	
Alcoholic	Yes	10	22.2
	No	35	77.8
Diabetes	Yes	8	17.8
	No	37	82.2
BMI		21.83 + 3.13	
Waist Circumference		33.76 $\pm$ 2.95	
Neck Circumference		80.2 $\pm$ 8.19	

***Prevalence of Sleep Related Breathing Disorders (SRBD) and Clinical Symptoms***

The prevalence of Sleep Related Breathing Disorders (SRBD) among the 45 patients with COPD in this study was low, primarily manifesting as Obstructive Sleep Apnea (OSA). Only 3 out of 45 patients (6.67%) were diagnosed with Obstructive

Sleep Apnea. No cases of Central Sleep Apnea or Mixed Sleep Apnea were identified. The mean Apnea-Hypopnea Index (AHI) for the total cohort was 3.33  $\pm$  3.75. Figure 1 shows the prevalence of Sleep Related Breathing Disorder (SRBD) among COPD patients in our study.

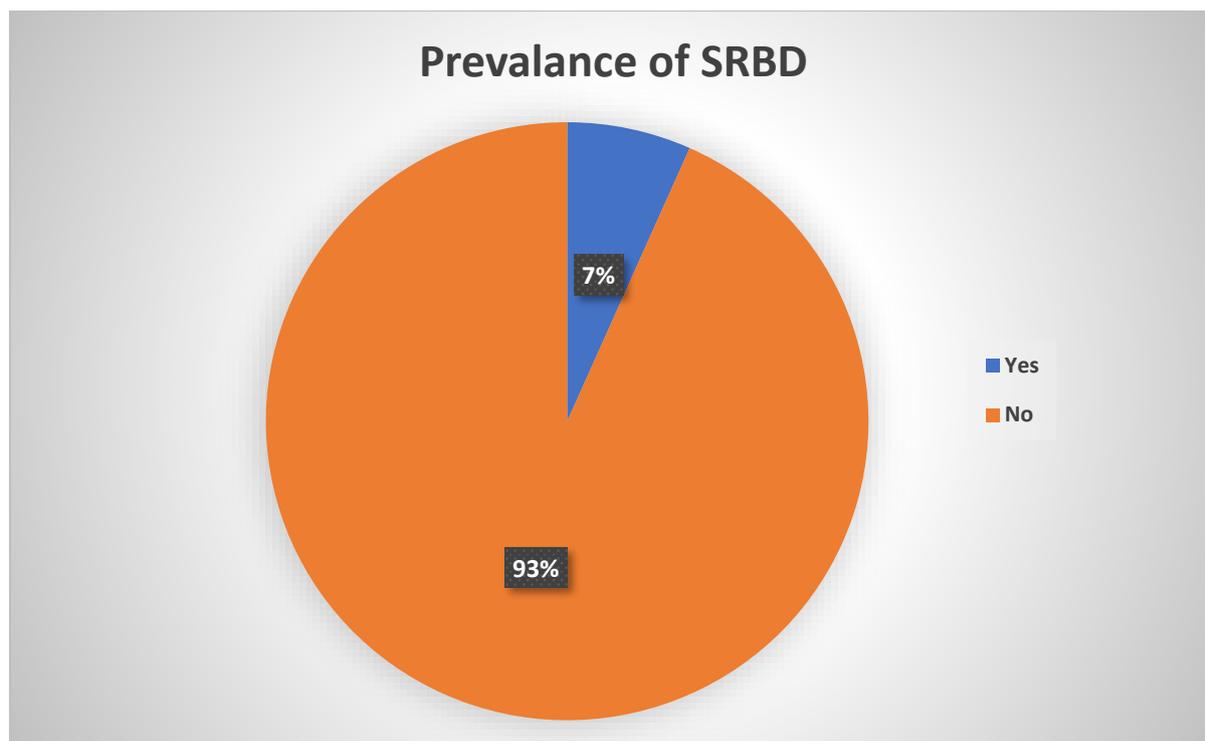


Figure 1. Prevalence of SRBD

Clinical symptoms related to sleep and respiration were common. Nocturnal awakening was the most frequent symptom, reported by 75.6% of patients. Other symptoms included snoring (35.6%), morning headache (33.3%), and choking episodes (6.7%). Predominant respiratory symptoms included breathlessness (62.2%) and a combination of breathlessness and wheeze (37.8%).

#### ***Assessment of Sleep Quality***

The study utilized both subjective and objective measures to assess sleep quality. The mean Pittsburgh Sleep Quality Index (PSQI) score was  $10.07 \pm 3.52$ ,

indicating overall poor sleep quality in the study population. Excessive daytime sleepiness, as measured by the Epworth Sleepiness Scale (ESS), showed a mean score of  $11.93 \pm 5.34$ .

Objective polysomnographic parameters for the total cohort showed a mean Total Sleep Time (TST) of  $264.71 \pm 51.68$  minutes and a mean sleep efficiency of  $64.15 \pm 9.10\%$ . The mean arousal index was  $25.39 \pm 7.74$  per hour. Sleep architecture was characterized by a mean NREM 1,2 percentage of 73.69%, NREM 3 of 10.19%, and REM of 15.75% of TST. Table 2 shows the distribution of quality of sleep variables.

Table 2. Quality of Sleep related variables

Variable	Distribution (Mean $\pm$ SD)
Total bed (TBT) (in minutes)	410.1 $\pm$ 42.3
Total Sleep Time (TST)	264.7 $\pm$ 51.7
WASO (Wake after sleep onset) (in minutes)	91.5 $\pm$ 27.4
WASO (% of TBT)	22.4 $\pm$ 6.5
Sleep latency (in minutes )	51.8 $\pm$ 17.6
Sleep efficiency %	64.2 $\pm$ 9.1
Arousal index (per hour)	25.4 $\pm$ 7.7
AHI (Apnea hypopnea Index)	3.33 $\pm$ 3.7
PSQI	10.07 $\pm$ 3.52
ESS	11.93 $\pm$ 5.34

***Comparison Between Patients with Respiratory Failure (Group A) and Without Respiratory Failure (Group B)***

For the purpose of comparative analysis, the total cohort was categorized into two groups based on the presence of chronic hypercapnic respiratory failure. Patients with Chronic Hypercapnic Respiratory Failure is defined by daytime awake PaCO<sub>2</sub> > 45 mmHg while in a stable condition with PaO<sub>2</sub> > 60 mm Hg and pH > 7.350 and those with awake PaCO<sub>2</sub> < 45 mmHg with PaO<sub>2</sub> > 60 mm Hg and pH > 7.350 are considered as without Hypercapnic Respiratory Failure. It is also noted that none of the patients in our study were on home non-invasive ventilation

(NIV) or long-term home oxygen therapy at the time of enrolment.

- **Group A:** Patients with COPD and Hypercapnic Respiratory Failure (n=32; 71.1%).
- **Group B:** Patients with COPD without Hypercapnic Respiratory Failure (n=13; 28.9%)

Clinical and Laboratory Parameters: Patients with respiratory failure (Group A, n=32) had a significantly higher pulse rate compared to Group B (90.66 vs. 82.54 bpm; p=0.000). Arterial Blood Gas (ABG) analysis confirmed the categorization, with Group A showing

significantly higher PaCO<sub>2</sub> (50.90 vs. 38.56 mmHg; p=0.000) and HCO<sub>3</sub> (30.03 vs. 25.66 mmol/L; p=0.000), and significantly lower PaO<sub>2</sub> (65.04 vs. 74.01 mmHg; p=0.000). There were no significant differences between the groups regarding age, BMI, or spirometric values (FEV1, FVC).

### ***Sleep Parameters and Quality***

Significant differences were observed in sleep parameters between the two groups:

- Sleep Duration and Efficiency: Group A had a significantly shorter TST (234.7 vs. 290.9 min; p=0.035) and lower sleep efficiency (58.5% vs. 69.1%; p=0.042).
- Sleep Latency and Arousals: Group A exhibited significantly prolonged sleep latency (62.7 vs. 42.4 min; p=0.003) and a higher arousal index (31.1 vs. 20.4; p=0.003).
- Subjective Sleep Quality: The PSQI score was significantly higher in Group A (13.2 vs. 7.3; p=0.018), reflecting worse sleep quality.
- Sleep Architecture: Group A showed a significantly higher percentage of NREM 1,2 (76.8% vs. 71%; p=0.019) and significantly lower NREM 3 duration (20.7 vs. 33.9 min; p=0.002) and REM duration (34.7 vs. 48.6 min; p=0.030).

Oxygen Saturation: Oxygenation was significantly more impaired in Group A across all parameters:

- Daytime SpO<sub>2</sub>: 92.6% vs. 93.8% (p=0.03).
- Nocturnal SpO<sub>2</sub>: 85.3% vs. 88.9% (p=0.001).
- Minimal Nocturnal SpO<sub>2</sub>: 75.2% vs. 79.4% (p=0.001).
- Significant Nocturnal Desaturation was more prevalent in Group A (68.8%) compared to Group B (38.5%).

Table 3 shows the distribution of quality of sleep variables among Group A (Patients with COPD and Respiratory Failure) and Group B (Patients with COPD without Respiratory Failure).

Table 3. Quality of Sleep related variables among the study groups

Variable	Group A (n=32)	Group B (n=13)	p-value
<b>Total bed (TBT) (in minutes)</b>	399.9 ± 35.3	419 ± 46.5	0.272
<b>Total sleep Time (TST) (in minutes)</b>	234.7 ± 33.6	290.9 ± 50.9	<b>0.035*</b>
<b>Sleep latency (in minutes )</b>	62.7 ± 18.5	42.4 ± 9.7	<b>0.003*</b>
<b>Sleep efficiency %</b>	58.5 ± 6.3	69.1 ± 8.4	<b>0.042*</b>
<b>Arousal index (per hour)</b>	31.1 ± 5.6	20.4 ± 5.6	<b>0.003*</b>
<b>WASO ( Wake after sleep onset ) (in minutes)</b>	100 ± 23.9	84 ± 28.6	0.599

<b>WASO (% of TBT)</b>	24.9 ± 5.3	20.1 ± 6.6	0.295
<b>AHI (Apnea hypopnea Index)</b>	3.19 ± 2.13	2.4 ± 1.7	0.987
<b>ESS score (Epworth Sleepiness Score)</b>	11.5 ± 5.5	9.7 ± 4.2	0.219
<b>PSQI score (Pittsburgh Sleep Quality Index)</b>	13.2 ± 1.9	7.3 ± 2.1	<b>0.018*</b>
<b>NREM1,2 (in Minutes )</b>	180.1 ± 29	208.1 ± 34.2	0.322
<b>NREM 1,2 (%of TST)</b>	76.8 ± 5.7	71 ± 6	<b>0.019*</b>
<b>NREM3 (Min )</b>	20.7 ± 8.5	33.9 ± 15.4	<b>0.002*</b>
<b>NREM3 (% of TST)</b>	8.8 ± 3.4	11.4 ± 3.9	<b>0.002*</b>
<b>REM (min )</b>	34.7 ± 7.8	48.6 ± 13.7	<b>0.03*</b>
<b>REM (% of TST)</b>	14.7 ± 3.2	16.7 ± 3.3	0.167
<b>Daytime SpO<sub>2</sub> %</b>	92.6 ± 1.6	93.8 ± 1.3	<b>0.03*</b>
<b>Nocturnal SpO<sub>2</sub> %</b>	85.3 ± 2.8	88.9 ± 2.9	<b>0.001*</b>
<b>Minimal nocturnal SpO<sub>2</sub> %</b>	75.2 ± 5.4	79.4 ± 6.2	<b>0.001*</b>
<b>Obstructive Sleep Apnea</b>	2 (6.25%)	1 (6.66%)	0.86

\*p-value<0.05-Statistically significant

## Discussion

In this study of 45 COPD patients, the mean age was  $57.38 \pm 7.48$  years with a predominance of males (82.2%), consistent with demographic patterns reported in Indian and global COPD cohorts where males often predominate due to higher smoking and environmental exposure. COPD continues to be a major public health issue in India with a high disease burden, particularly in populations with tobacco use and biomass exposure [3]. Moreover, studies have noted regional variations in COPD prevalence, emphasizing the influence of local environmental and lifestyle factors [9,10].

The prevalence of Sleep Related Breathing Disorders (SRBD) in our cohort was low (6.67% for OSA), with a mean AHI of  $3.33 \pm 3.75$ , and no central or mixed apnea identified. While some reports show higher rates of overlap syndrome, prevalence estimates vary widely across settings and diagnostic approaches [11,12].

The relatively low OSA prevalence in our sample may reflect the predominance of normal BMI and exclusion of major comorbidities. Nevertheless, clinical symptoms of sleep disturbance were frequently reported — most notably nocturnal awakenings (75.6%), snoring, and morning headache — aligning with literature indicating high rates of

symptomatic sleep disruption in COPD even when polysomnographic OSA is uncommon [5,13,14].

Objective and subjective assessments of sleep quality further highlighted significant impairment in this cohort. The mean PSQI score of  $10.07 \pm 3.52$  reflects poor overall sleep quality, and the mean ESS score of  $11.93 \pm 5.34$  indicates excessive daytime somnolence. These results are consistent with prior studies showing that COPD patients often experience poor sleep quality due to nocturnal dyspnea, cough, and frequent arousals, and that poor PSQI scores correlate with lower quality of life in COPD [7,15].

Polysomnographic parameters in our study showed reduced total sleep time, low sleep efficiency, and a high arousal index, similar to findings from polysomnography studies reporting fragmented sleep and reduced restorative stages in COPD patients [5].

When comparing patients with chronic hypercapnic respiratory failure (Group A) to those without (Group B), Group A demonstrated significant gas exchange abnormalities — higher  $\text{PaCO}_2$  and  $\text{HCO}_3^-$ , and lower  $\text{PaO}_2$  — reflecting greater ventilatory insufficiency. These findings are in line with evidence showing that chronic hypercapnia is associated with impaired ventilatory response and poorer outcomes in COPD [16,17].

Notably, spirometric values ( $\text{FEV}_1$ , FVC) were similar between groups, underscoring that hypercapnia and nocturnal respiratory impairment may not be fully explained by traditional spirometric severity alone. This observation has been reported in other studies where nocturnal desaturation and hypercapnia occur

independent of airflow limitation severity [5,18].

Group A also had significantly poorer sleep parameters and quality than Group B: shorter total sleep time, lower sleep efficiency, longer sleep latency, higher arousal index, and worse PSQI scores, corroborating evidence that nocturnal hypoxemia and gas exchange dysfunction in COPD adversely impact sleep [5,19].

Additionally, Group A showed altered sleep architecture with higher proportions of lighter NREM stages and reduced deep and REM sleep, consistent with studies demonstrating that hypoxia and ventilatory instability in COPD fragment sleep and reduce restorative sleep stages [7,20].

Measures of oxygenation, including daytime and nocturnal  $\text{SpO}_2$  and minimal nocturnal saturation, were significantly worse in Group A, further confirming the close relationship between nocturnal hypoxemia and sleep disruption in COPD [5].

## Conclusion

This study demonstrates that sleep impairment is highly prevalent among patients with severe stable COPD, even though the prevalence of polysomnography-confirmed sleep related breathing disorders (SRBD), particularly OSA, was low in the present cohort. The majority of patients reported significant sleep-related symptoms, and both subjective (PSQI, ESS) and objective polysomnographic findings confirmed poor sleep quality, reduced sleep efficiency, shortened total sleep time, and increased sleep fragmentation.

Patients with chronic hypercapnic respiratory failure exhibited significantly

worse sleep outcomes than those without respiratory failure, including poorer sleep efficiency, prolonged sleep latency, higher arousal index, altered sleep architecture with reduced restorative sleep stages (NREM3 and REM), and more severe nocturnal oxygen desaturation. These findings highlight that gas exchange impairment and nocturnal hypoxemia, rather than OSA alone, may be major contributors to disturbed sleep in severe COPD. Routine assessment of sleep quality and nocturnal oxygenation in COPD—especially among patients with chronic hypercapnia—may therefore be clinically valuable for comprehensive disease management and improving overall patient outcomes.

### **Limitations**

The relatively small sample size and recruitment from a single tertiary care center may limit the generalizability of the findings across wider COPD population. Potential confounding factors such as medication use, coexisting psychiatric conditions, and socioeconomic influences on sleep were not comprehensively assessed. Although full attended polysomnography was performed, apnea–hypopnea index (AHI) values were based on automated scoring and were not independently verified through manual rescoring, which may introduce measurement variability. Additionally, the single-night polysomnographic assessment may not account for night-to-night variability in sleep parameters.

### **Statements and Declarations**

#### **Author Contributions**

KR has contributed to the conceptualization and design of the study, literature search, data acquisition,

manuscript editing and review. VS contributed towards data acquisition Statistical analysis, Manuscript review and editing. KR acted as the corresponding author for this manuscript

### **Conflicts of interest**

The authors declare that they do not have conflict of interest.

### **Funding**

No funding was received for conducting this study.

### **Data availability statement**

The datasets generated and analyzed are not publicly available due to the presence of sensitive information that could potentially lead to indirect identification of participants and are available from the corresponding author upon reasonable request.

### **Ethical Consideration**

This study has been approved by the Institution Ethics Committee and Written informed consent was obtained from all participants

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*Use of AI:* Authors declare the usage of Chat-GPT 4.0 for content and language moderation alone.

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