Section: Pulmonary Medicine

Original Research Article

ASSOCIATION OF HYPOXIC BURDEN WITH DISEASE SEVERITY IN OBSTRUCTIVE SLEEP APNOEA: A POLYSOMNOGRAPHY-BASED STUDY

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Received *: 09/11/2025*
Received in revised form *: 16/12/2025*
Accepted *: 03/01/2026*

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DOI: 10.70034/ijmedph.2026.1.164

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 933-938

ABSTRACT

Background: Obstructive sleep apnoea (OSA) is characterized by recurrent upper airway obstruction during sleep, leading to intermittent hypoxaemia and sleep fragmentation. While the apnoea–hypopnoea index (AHI) is the standard metric for grading disease severity, it does not fully capture the physiological impact of nocturnal hypoxia. Oxygen desaturation parameters may better reflect hypoxic burden and associated clinical risk. This study aimed to evaluate the correlation between the degree of oxygen desaturation and the severity of OSA in adults undergoing diagnostic polysomnography.

Materials and Methods: This hospital-based cross-sectional study included 92 adult patients with polysomnography-confirmed OSA. All participants underwent full-night, attended, in-laboratory polysomnography. OSA severity was classified using AHI into mild, moderate, and severe categories. Oxygen desaturation parameters analyzed included minimum and mean nocturnal oxygen saturation (SpO_2), oxygen desaturation index (ODI), and percentage of total sleep time spent with SpO_2 below 90% (T90). Comparisons across severity groups were performed using appropriate parametric or non-parametric tests, and correlations between AHI and desaturation parameters were assessed using correlation coefficients.

Results: The mean age of participants was 52.6 ± 10.8 years, with male predominance (72.8%). Severe OSA constituted 40.2% of cases. A progressive worsening of oxygen desaturation was observed with increasing OSA severity. Minimum SpO_2 decreased from 86.9% in mild OSA to 74.8% in severe OSA, while ODI increased from 9.6 to 38.7 events/hour ($p < 0.001$). Time spent with $SpO_2 < 90\%$ increased significantly across severity categories ($p < 0.001$). AHI showed strong positive correlations with ODI ($r = 0.72$) and T90 ($r = 0.74$), and significant negative correlations with minimum ($r = -0.68$) and mean SpO_2 ($r = -0.61$) (all $p < 0.001$).

Conclusion: Nocturnal oxygen desaturation parameters correlate strongly with the severity of obstructive sleep apnoea. Incorporating hypoxic burden indices alongside AHI may provide a more comprehensive assessment of disease severity and aid in improved risk stratification and clinical management of patients with OSA.

Keywords: Obstructive sleep apnoea; Oxygen desaturation; Hypoxic burden; Apnoea–hypopnoea index; Polysomnography

INTRODUCTION

Obstructive sleep apnoea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, leading to intermittent hypoxaemia, sleep fragmentation, and marked fluctuations in intrathoracic pressure.[1] The global

prevalence of OSA is steadily increasing, paralleling the rise in obesity and ageing populations, with community-based studies suggesting that moderate-to-severe OSA affects nearly 10–20% of middle-aged adults, with higher rates reported among men and individuals with metabolic comorbidities.[2] In the Indian context, epidemiological data indicate a substantial and under-recognized burden of OSA, particularly in urban populations.[3]

The severity of OSA is conventionally graded using the apnoea–hypopnoea index (AHI), which quantifies the number of apnoeic and hypopnoeic events per hour of sleep. While AHI remains the cornerstone for diagnosis and classification, it does not fully capture the physiological impact of repeated respiratory events.[4] Among the downstream consequences of obstructive events, oxygen desaturation—reflecting the depth and duration of hypoxaemia—plays a pivotal role in mediating end-organ dysfunction.[5] Recurrent nocturnal desaturation triggers sympathetic activation, oxidative stress, systemic inflammation, and endothelial dysfunction, thereby contributing to the development of hypertension, coronary artery disease, stroke, insulin resistance, and cognitive impairment.[6]

Measures of nocturnal oxygenation, such as minimum oxygen saturation (SpO_2 nadir), mean SpO_2, total sleep time spent below a critical saturation threshold (e.g., <90%), and oxygen desaturation index (ODI), provide complementary information beyond AHI.[7] Several studies have suggested that indices of hypoxic burden may correlate more strongly with cardiovascular and metabolic outcomes than AHI alone.[7,8]

Despite growing recognition of the clinical importance of intermittent hypoxaemia, the relationship between the degree of oxygen desaturation and polysomnographic severity of OSA remains incompletely understood, particularly in resource-limited settings.[3] Most available data are derived from Western populations, and there is a paucity of Indian studies systematically evaluating how desaturation parameters vary across different severity categories of OSA. Given differences in craniofacial structure, body habitus, and comorbidity patterns, extrapolation of international data to the Indian population may not always be appropriate.[3,9]

Understanding the correlation between the degree of nocturnal desaturation and OSA severity has important clinical implications. It may help refine risk stratification, identify patients at higher risk of hypoxia-related complications, and support a more comprehensive approach to disease assessment that integrates both frequency-based and hypoxia-based metrics. In this context, the present cross-sectional study was aimed to evaluate the relationship between oxygen desaturation parameters and the severity of obstructive sleep apnoea, as determined by standard polysomnographic indices, in an adult patient population.

MATERIALS AND METHODS

Study design and setting: This hospital-based cross-sectional observational study was conducted in the Department of Pulmonary Medicine in collaboration with the Sleep Laboratory of a tertiary care teaching hospital in India. The study period extended over 12 months, from January 2023 to January 2024. The objective was to evaluate the correlation between the degree of nocturnal oxygen desaturation and the severity of obstructive sleep apnoea (OSA) as assessed by standard polysomnographic parameters.

Study population: Adult patients presenting with symptoms suggestive of sleep-disordered breathing, such as habitual snoring, excessive daytime sleepiness, witnessed apnoeas, non-refreshing sleep, or unexplained fatigue, and referred for diagnostic overnight polysomnography were screened for eligibility. Patients aged ≥18 years who were newly diagnosed with OSA on overnight polysomnography were included in the study. Individuals with predominantly central sleep apnoea, known chronic lung diseases (such as COPD, interstitial lung disease, or bronchiectasis), congestive heart failure, neuromuscular disorders, significant chest wall deformities, or those on long-term oxygen therapy were excluded to avoid confounding effects on nocturnal oxygenation. Patients with acute respiratory illness at the time of study or previously treated OSA were also excluded.

Sample size and sampling technique: A convenience sampling approach was employed, enrolling all eligible consecutive patients undergoing diagnostic polysomnography during the study period. The sample size was calculated to detect a minimum correlation coefficient (r) of 0.40 between oxygen desaturation parameters and apnoea–hypopnoea index (AHI), with a two-sided alpha error of 0.05 and a power of 80%.[10] Based on these assumptions, the minimum required sample size was 84 participants. To account for potential data loss or technically inadequate polysomnography recordings, a total of 92 patients were enrolled in the study. All patients fulfilling the inclusion and exclusion criteria were included until the desired sample size was achieved.

Clinical evaluation and baseline assessment: A detailed clinical evaluation was performed for all participants prior to polysomnography. Demographic data including age, sex, and body mass index (BMI) were recorded. Relevant clinical history focusing on sleep-related symptoms, comorbidities such as hypertension, diabetes mellitus, and cardiovascular disease, and history of smoking or alcohol consumption was obtained. Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS), with a score >10 considered indicative of excessive daytime sleepiness.

Polysomnography protocol: All participants underwent full-night, attended, in-laboratory polysomnography using a standardized digital polysomnography system. Recordings included

electroencephalography (EEG), electrooculography (EOG), submental and tibial electromyography (EMG), electrocardiography (ECG), airflow measurement using nasal pressure transducer and oronasal thermal sensor, respiratory effort using thoracic and abdominal belts, body position sensors, and continuous pulse oximetry for oxygen saturation (SpO_2). Sleep stages and respiratory events were scored manually by a trained sleep technologist and reviewed by a qualified sleep physician, in accordance with the American Academy of Sleep Medicine (AASM) guidelines.

Apnoea was defined as a ≥90% reduction in airflow lasting at least 10 seconds, while hypopnoea was defined as a ≥30% reduction in airflow lasting at least 10 seconds associated with either ≥3% oxygen desaturation or an arousal. The apnoea–hypopnoea index (AHI) was calculated as the total number of apnoeas and hypopnoeas per hour of sleep.

Classification of OSA severity: Based on AHI values, OSA severity was classified as mild (AHI 5–14.9 events/hour), moderate (AHI 15–29.9 events/hour), and severe (AHI ≥30 events/hour). Patients with AHI <5 events/hour were excluded from the analysis.

Assessment of oxygen desaturation parameters: Nocturnal oxygenation parameters were derived from continuous pulse oximetry recordings obtained during polysomnography. The degree of desaturation was assessed using minimum SpO_2 (nadir saturation), mean SpO_2 during total sleep time, oxygen desaturation index (ODI; defined as the number of ≥3% desaturation events per hour of sleep), and cumulative time spent with SpO_2 below 90% (T90), expressed as both minutes and percentage of total sleep time. These parameters were analyzed individually and in relation to AHI-based OSA severity categories.

Statistical analysis: Data were entered into a predesigned proforma and analysed using the Statistical Package for the Social Sciences (SPSS) software, version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on data distribution, while categorical variables were expressed as frequencies and percentages. Normality of data was assessed using the Shapiro–Wilk test. Comparisons of oxygen desaturation parameters across OSA severity groups were performed using one-way analysis of variance (ANOVA) or Kruskal–Wallis test, as appropriate. Correlation between AHI and oxygen desaturation parameters was evaluated using Pearson's or Spearman's correlation coefficient. A p-value <0.05 was considered statistically significant.

Ethical considerations: The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee prior to commencement of the study. Written informed consent was obtained from all participants before enrollment, and confidentiality of patient data was strictly maintained.

RESULTS

The study included 92 adults with polysomnography-confirmed obstructive sleep apnoea. The mean age of participants was 52.6 ± 10.8 years, with a predominance of males (72.8%). The majority belonged to the middle-aged and elderly age groups, with 65.2% aged ≥50 years. The mean body mass index (BMI) was 29.4 ± 4.2 kg/m², with 85.9% of participants being overweight or obese. The mean Epworth Sleepiness Scale (ESS) score was 13.8 ± 4.2, and excessive daytime sleepiness (ESS > 10) was observed in 66.3% of patients. Hypertension and diabetes mellitus were present in 42.4% and 30.4% of participants, respectively [Table1].

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 92).

Variable	Frequency (%)/mean ± SD
Age (years)	52.6 ± 10.8
Age group (years)	
30–39	8 (8.7)
40–49	24 (26.1)
50–59	34 (37.0)
≥60	26 (28.2)
Gender	
Male	67 (72.8)
Female	25 (27.2)
Body Mass Index (kg/m²)	29.4 ± 4.2
BMI category (kg/m²)	
Overweight (25–29.9)	41 (44.6)
Obese (≥30)	38 (41.3)
Neck circumference (cm)	39.6 ± 3.1
Epworth Sleepiness Scale score	13.8 ± 4.2
ESS >10	61 (66.3)
Hypertension	39 (42.4)
Diabetes mellitus	28 (30.4)

BMI – Body mass index; ESS – Epworth Sleepiness Scale

Based on apnoea–hypopnoea index (AHI) values, 26 patients (28.3%) were classified as having mild OSA, 29 (31.5%) as moderate OSA, and 37 (40.2%) as severe OSA. Severe OSA constituted the largest proportion of cases, indicating a substantial burden of advanced disease among patients referred to the tertiary care sleep laboratory [Table 2].

Table 2: Distribution of Obstructive Sleep Apnoea Severity Based on Apnoea–Hypopnoea Index.

OSA Severity	AHI (events/hour)	Frequency (%)
Mild	5.0–14.9	26 (28.3)
Moderate	15.0–29.9	29 (31.5)
Severe	≥30.0	37 (40.2)

OSA – Obstructive sleep apnoea; AHI – Apnoea–hypopnoea index

A progressive and statistically significant worsening of oxygen desaturation parameters was observed with increasing OSA severity. Minimum SpO_2 declined significantly from mild (86.9 ± 3.8%) to moderate (81.4 ± 4.6%) and severe OSA (74.8 ± 6.2%) ($p < 0.001$). Similarly, mean nocturnal SpO_2 decreased across severity groups ($p < 0.001$). Oxygen desaturation index (ODI) showed a marked increase from mild to severe OSA (9.6 ± 4.1 vs 38.7 ± 11.4 events/hour; $p < 0.001$). The proportion of total sleep time spent with SpO_2 < 90% (T90) increased substantially with disease severity, with severe OSA patients exhibiting a median T90 of 26.3% compared to 2.4% in mild OSA ($p < 0.001$) [Table 3].

Table 3: Oxygen Desaturation Parameters Across OSA Severity Categories.

Parameter	Mild OSA (n=26)	Moderate OSA (n=29)	Severe OSA (n=37)	p-value
	mean ± SD/median (IQR)			
Minimum SpO_2 (%)	86.9 ± 3.8	81.4 ± 4.6	74.8 ± 6.2	<0.001
Mean SpO_2 (%)	94.1 ± 1.6	92.3 ± 2.1	89.6 ± 2.8	<0.001
ODI (events/hour)	9.6 ± 4.1	19.8 ± 6.3	38.7 ± 11.4	<0.001
Time with SpO_2 <90% (T90, % of TST)	2.4 (1.1–4.6)	8.7 (5.2–14.1)	26.3 (18.4–41.7)	<0.001

SpO_2 – Peripheral oxygen saturation; ODI – Oxygen desaturation index; T90 – Percentage of total sleep time with SpO_2 < 90%; TST – Total sleep time

Correlation analysis demonstrated strong and statistically significant associations between AHI and indices of nocturnal hypoxaemia. AHI showed a strong positive correlation with ODI ($r = 0.72$, $p < 0.001$) and T90 ($r = 0.74$, $p < 0.001$). Conversely, significant negative correlations were observed between AHI and minimum SpO_2 ($r = −0.68$, $p < 0.001$) as well as mean SpO_2 ($r = −0.61$, $p < 0.001$), indicating worsening oxygenation with increasing OSA severity [Table 4].

Table 4: Correlation Between Apnoea–Hypopnoea Index and Oxygen Desaturation Parameters.

Parameter	Correlation coefficient (r)	p-value
Minimum SpO_2 (%)	−0.68	<0.001
Mean SpO_2 (%)	−0.61	<0.001
ODI (events/hour)	0.72	<0.001
T90 (% of TST)	0.74	<0.001

AHI – Apnoea–hypopnoea index; SpO_2 – Peripheral oxygen saturation

When patients were dichotomized into non-severe (mild–moderate) and severe OSA groups, severe OSA patients demonstrated significantly worse nocturnal oxygenation across all parameters. Minimum and mean SpO_2 levels were significantly lower in severe OSA, while ODI and T90 were markedly higher compared to non-severe OSA (all $p < 0.001$). These findings highlight the substantially greater hypoxic burden associated with severe disease [Table 5].

Table 5: Comparison of Oxygen Desaturation Parameters Between Non-Severe and Severe OSA.

Parameter	Mild–Moderate OSA (n=55)	Severe OSA (n=37)	p-value
	mean ± SD/median (IQR)		
Minimum SpO_2 (%)	84.0 ± 4.9	74.8 ± 6.2	<0.001
Mean SpO_2 (%)	93.2 ± 2.0	89.6 ± 2.8	<0.001
ODI (events/hour)	15.1 ± 7.8	38.7 ± 11.4	<0.001
T90 (% of TST), median (IQR)	5.8 (2.4–10.7)	26.3 (18.4–41.7)	<0.001

OSA – Obstructive sleep apnoea; ODI – Oxygen desaturation index; T90 – Percentage of total sleep time with SpO_2 < 90%.

DISCUSSION

The present cross-sectional study demonstrates a strong and graded association between the degree of nocturnal oxygen desaturation and the severity of obstructive sleep apnoea (OSA), as assessed by the apnoea–hypopnoea index (AHI). With increasing OSA severity, there was a progressive decline in

minimum and mean nocturnal oxygen saturation, accompanied by a marked rise in oxygen desaturation index (ODI) and the proportion of total sleep time spent with SpO_2 below 90% (T90).[11] Furthermore, correlation analysis revealed strong positive correlations of AHI with ODI and T90 and significant negative correlations with minimum and mean SpO_2, underscoring the close pathophysiological link between event frequency and hypoxic burden.[12]

The observed demographic profile, characterized by male predominance, middle-to-older age distribution, and a high prevalence of overweight and obesity, is consistent with the established epidemiology of OSA in Indian populations.[13,14] Studies by Agrawal et al., and Srivastava et al., have similarly reported male predominance (65–75%) and mean BMI values in the overweight or obese range, reflecting changing lifestyle patterns and increasing metabolic risk factors in the Indian population.[15,16] The high prevalence of hypertension and diabetes mellitus in the present cohort further supports the close association between OSA and cardiometabolic comorbidities.[17]

A key finding of this study is the progressive worsening of nocturnal hypoxaemia across OSA severity categories. Minimum SpO_2 showed a steep decline from mild to severe OSA, highlighting deeper nadirs of oxygenation in patients with higher AHI. This is pathophysiologically explained by prolonged and more frequent upper airway collapse in severe OSA, leading to sustained apnoeic events, reduced alveolar ventilation, and delayed reoxygenation during sleep.[18] Mean nocturnal SpO_2, which reflects cumulative hypoxic exposure, also decreased significantly with increasing severity, indicating that severe OSA is characterized not only by deeper desaturation but also by a greater overall hypoxic burden.[19,20]

The oxygen desaturation index emerged as one of the strongest correlates of OSA severity in the present study. ODI reflects the frequency of intermittent hypoxia episodes and has been increasingly recognized as a clinically meaningful marker of disease burden. International studies by De Chazal P et al., and Hui et al., have demonstrated that ODI correlates closely with AHI and may even outperform AHI in predicting cardiovascular outcomes such as hypertension, coronary artery disease, and stroke.[21,22] Similar to our findings, prior Indian studies by Varghese et al., and Sharma et al., have reported significantly higher ODI values in severe OSA compared to mild and moderate disease, emphasizing the relevance of intermittent hypoxia in disease progression.[23,24]

Time spent with oxygen saturation below 90% (T90) showed the strongest positive correlation with AHI in this study. T90 represents the cumulative duration of hypoxaemia and is considered a robust indicator of hypoxic load. Prolonged nocturnal hypoxaemia has been linked to sympathetic nervous system activation, oxidative stress, systemic inflammation, endothelial dysfunction, and metabolic dysregulation.[25] These mechanisms provide a plausible biological explanation for the strong association between higher T90 values and severe OSA observed in our cohort. International literature by Zeng et al., and He et al., increasingly suggest that hypoxic burden indices such as T90 may better predict long-term cardiovascular and neurocognitive consequences than AHI alone.[26,27]

The strong inverse correlations observed between AHI and both minimum and mean SpO_2 further highlight that increasing event frequency is associated with worsening oxygenation. However, variability in desaturation profiles among patients with similar AHI values suggests that AHI alone may not fully capture disease severity. This heterogeneity has been reported in several western studies by Coso et al., and Martinez-Garcia et al., where patients with comparable AHI demonstrate markedly different hypoxic burdens and clinical outcomes.[28,29] Our findings support the growing consensus that incorporating oxygen desaturation metrics into routine OSA assessment may provide a more comprehensive evaluation of disease severity, particularly in populations with high cardiometabolic risk.[30]

When patients were dichotomized into non-severe and severe OSA groups, those with severe disease exhibited significantly worse desaturation parameters across all indices. This finding has important clinical implications, as patients with severe hypoxaemia are likely to be at higher risk for adverse cardiovascular and metabolic outcomes and may benefit from early and aggressive therapeutic interventions such as continuous positive airway pressure (CPAP) therapy. Indian studies have similarly shown that severe OSA patients with greater hypoxic burden demonstrate better symptomatic and blood pressure responses following CPAP initiation, further emphasizing the clinical relevance of hypoxia-based severity assessment.[31,32]

Limitations

This study has certain limitations that should be acknowledged while interpreting the findings. First, the cross-sectional design limits the ability to establish a causal relationship between oxygen desaturation parameters and the severity of obstructive sleep apnoea (OSA). Longitudinal studies are required to determine whether a higher hypoxic burden independently predicts disease progression or adverse clinical outcomes. Second, the study was conducted at a single tertiary care centre, which may limit the generalizability of the results to community-based populations or primary care settings. Third, although standard in-laboratory polysomnography was used, night-to-night variability in sleep architecture and respiratory events could not be assessed. Fourth, potential confounders such as alcohol intake, smoking status, and concurrent medication use were not analyzed in detail, which may have influenced nocturnal oxygenation. Finally, cardiovascular outcomes and response to therapy were not evaluated, precluding assessment of the

prognostic significance of hypoxic burden indices beyond their association with OSA severity.

CONCLUSION

The present study demonstrates a strong and consistent correlation between the degree of nocturnal oxygen desaturation and the severity of obstructive sleep apnoea. Oxygen desaturation parameters—including minimum and mean SpO_2, oxygen desaturation index, and time spent with SpO_2 below 90%—worsened progressively with increasing apnoea–hypopnoea index and showed strong correlations with disease severity. These findings highlight that nocturnal hypoxaemia is a central pathophysiological component of OSA rather than a mere epiphenomenon.

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