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Original Research Article

Study to Evaluate the Role of Inflammatory Biomarker- IL-6 in Obstructive Sleep Apnea in Correlation with Apnea-Hypopnea Index

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Abstract

Background: Obstructive sleep apnea (OSA) is characterized by recurrent upper airway obstruction during sleep, leading to intermittent hypoxia and sleep fragmentation. Increasing evidence suggests that OSA is associated with chronic low-grade systemic inflammation, which may contribute to its cardiometabolic complications. Interleukin-6 (IL-6) is a key pro-inflammatory cytokine implicated in hypoxia-induced inflammatory pathways; however, its relationship with OSA severity remains incompletely understood, particularly in the Indian population.

Methods: This hospital-based observational analytical study included 89 adult patients diagnosed with obstructive sleep apnea using overnight polysomnography. OSA severity was classified based on the apnea–hypopnea index (AHI) into mild, moderate, and severe categories. Fasting venous blood samples were collected the morning after polysomnography, and serum IL-6 levels were measured using enzyme-linked immunosorbent assay. Correlations between IL-6 levels and polysomnographic parameters were assessed, and comparisons across OSA severity groups were performed using appropriate statistical tests. Analysis of covariance was used to adjust for body mass index.

Results: The mean age of the study population was 51.8 ± 10.6 years, with a male predominance (71.9%). Moderate-to-severe OSA was observed in 74.2% of patients. Serum IL-6 levels increased progressively with OSA severity, with mean values of 3.9 ± 1.4 pg/mL in mild OSA, 6.2 ± 2.1 pg/mL in moderate OSA, and 9.1 ± 3.0 pg/mL in severe OSA ($p < 0.001$). IL-6 levels showed a strong positive correlation with AHI ($r = 0.62$, $p < 0.001$) and oxygen desaturation index ($r = 0.55$, $p < 0.001$), and an inverse correlation with minimum oxygen saturation ($r = -0.48$, $p < 0.001$). The association between IL-6 levels and OSA severity remained significant after adjustment for body mass index (adjusted $p < 0.001$).

Conclusion: Serum IL-6 levels are significantly elevated in obstructive sleep apnea and increase progressively with disease severity. The strong correlation between IL-6 and apnea–hypopnea index, independent of obesity, supports the role of IL-6 as a biomarker of OSA severity and highlights the contribution of systemic inflammation to the pathophysiology of OSA.

Keywords: Obstructive sleep apnea; Interleukin-6; Apnea–hypopnea index; Inflammation; Intermittent hypoxia.
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Introduction

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxia, sleep fragmentation, and marked fluctuations in intrathoracic pressure [1]. These pathophysiological disturbances contribute to excessive daytime sleepiness, impaired neurocognitive function, and reduced quality of life. OSA is increasingly recognized as a major public health problem, with a reported global prevalence ranging from 9% to 38% in adults, and substantially higher rates among males, obese individuals, and older populations [2]. In the Indian context, community-based studies have reported OSA prevalence rates of approximately 13–19% in middle-aged adults, reflecting a rising burden paralleling increasing obesity and metabolic disorders [3]. The severity of OSA is conventionally quantified using the apnea–

hypopnea index (AHI), which represents the number of apneas and hypopneas per hour of sleep recorded during overnight polysomnography [4]. AHI remains the gold standard for grading disease severity and stratifying cardiovascular and metabolic risk, with higher AHI values strongly associated with adverse clinical outcomes [4]. However, AHI alone does not fully capture the systemic biological consequences of OSA, particularly the inflammatory and metabolic alterations that mediate long-term complications [5].

Emerging evidence suggests that OSA is a state of chronic low-grade systemic inflammation. Repetitive cycles of hypoxia and reoxygenation, termed chronic intermittent hypoxia, activate oxidative stress pathways, sympathetic nervous system overactivity, and endothelial dysfunction [6]. These processes collectively stimulate the release of pro-inflammatory cytokines, thereby promoting a sustained inflammatory milieu [6]. This inflammatory activation is believed to play a pivotal role in the development of OSA-related comorbidities, including hypertension, atherosclerosis, insulin resistance, and cardiovascular disease [6].

Interleukin-6 (IL-6) is a pleiotropic pro-inflammatory cytokine produced by a wide range of cells, including macrophages, endothelial cells, adipocytes, and skeletal muscle cells. IL-6 plays a central role in immune regulation, acute-phase responses, and metabolic homeostasis [7]. Elevated circulating IL-6 levels have been implicated in the pathogenesis of cardiovascular disease, type 2 diabetes mellitus, and metabolic syndrome—conditions that frequently coexist with OSA [7]. Importantly, IL-6 synthesis is upregulated in response to hypoxia and oxidative stress, making it a biologically plausible mediator linking intermittent hypoxia to systemic inflammation in OSA [7].

Several studies have demonstrated increased serum IL-6 levels in patients with OSA compared to healthy controls, with some reporting a positive association between IL-6 concentrations and disease severity as assessed by AHI [8]. Nonetheless, the existing literature remains heterogeneous, with variability in study populations, confounding factors such as obesity, and inconsistent adjustment for comorbid conditions. Moreover, data from Indian populations are limited, despite potential ethnic and phenotypic differences influencing inflammatory responses and OSA severity [9].

Understanding the relationship between IL-6 and OSA severity is clinically relevant, as IL-6 may serve not only as a biomarker of disease burden but also as a potential predictor of cardiometabolic risk in OSA patients. Establishing a correlation between IL-6 levels and AHI could strengthen the role of inflammatory biomarkers in risk stratification, disease monitoring, and potentially in guiding therapeutic interventions beyond conventional polysomnographic parameters.

In this context, the present study aimed to evaluate the role of inflammatory biomarker IL-6 in patients with obstructive sleep apnea and to assess its correlation with apnea–hypopnea index, thereby contributing to a better understanding of the inflammatory pathophysiology of OSA in the studied population.

Materials and Methods

Study Design and Study Setting: This study was designed as a hospital-based observational analytical study conducted in the department of Medicine in a tertiary care teaching hospital in North India, during period of 2 years between July 2023 to July 2025, with a dedicated sleep laboratory facility. The study was undertaken to evaluate serum interleukin-6 (IL-6) levels in patients diagnosed with obstructive sleep apnea (OSA) and to assess the correlation between IL-6 concentration and disease severity as measured by the apnea–hypopnea index (AHI).

Study Population and Recruitment: Adult patients attending the outpatient or inpatient services of the Department of Medicine with clinical features suggestive of sleep-disordered breathing were screened for eligibility. Patients presenting with habitual snoring, excessive daytime sleepiness, witnessed apneas during sleep, nocturnal choking, or unrefreshing sleep were evaluated clinically and referred for overnight polysomnography. Consecutive patients who fulfilled the diagnostic criteria for obstructive sleep apnea on polysomnography were enrolled in the study after obtaining written informed consent. Where applicable, an age- and sex-matched control group comprising individuals without symptoms of sleep-disordered breathing and with normal polysomnography findings was also included for comparison.

Inclusion and Exclusion Criteria: Patients aged 18 years and above with newly diagnosed obstructive sleep apnea, defined by an AHI of ≥5 events per hour on overnight polysomnography, were included in the study. Patients with a history of acute or chronic inflammatory conditions, autoimmune diseases, malignancy, chronic kidney disease, chronic liver disease, or acute infections within the preceding four weeks were excluded to avoid confounding influences on inflammatory biomarker levels. Individuals receiving systemic corticosteroids, immunosuppressive agents, or long-term anti-inflammatory medications were also excluded. Patients previously diagnosed with OSA

and already on treatment such as continuous positive airway pressure therapy were not included. Pregnant women were excluded from the study.

Clinical and Anthropometric Assessment: All enrolled participants underwent a detailed clinical evaluation at baseline. Demographic data including age and sex were recorded. Anthropometric measurements such as height, weight, and body mass index (BMI) were obtained using standardized techniques. BMI was calculated as weight in kilograms divided by height in meters squared. Daytime sleepiness was assessed using the Epworth Sleepiness Scale. Blood pressure was measured using a standardized sphygmomanometer, and information regarding comorbid conditions such as hypertension, diabetes mellitus, and dyslipidemia was documented based on medical history and available records.

Polysomnography and Diagnosis of Obstructive Sleep Apnea: All participants underwent overnight attended polysomnography in the sleep laboratory using a standardized digital polysomnography system. The recording montage included electroencephalography, electrooculography, submental and leg electromyography, electrocardiography, nasal airflow (pressure transducer and thermistor), thoracic and abdominal respiratory effort belts, pulse oximetry, and body position sensors. Sleep stages and respiratory events were scored manually by a trained sleep technologist in accordance with standard guidelines. Apnea was defined as a complete cessation of airflow lasting at least 10 seconds, while hypopnea was defined as a reduction in airflow of at least 30% lasting for 10 seconds or more, associated with oxygen desaturation or arousal. The apnea–hypopnea index was calculated as the total number of apneas and hypopneas per hour of sleep. Based on AHI values, OSA severity was classified as mild (5–14.9 events/hour), moderate (15–29.9 events/hour), and severe (≥30 events/hour).

Blood Sample Collection and Processing: Venous blood samples were collected from all study participants in the morning following overnight polysomnography after an overnight fast of at least 8 hours. Blood samples were drawn under aseptic conditions using standard venipuncture techniques. The collected samples were allowed to clot and were then centrifuged at appropriate speed to separate serum. The serum was aliquoted into sterile cryovials and stored at −80°C until further analysis. All samples were processed following uniform pre-analytical protocols to minimize variability.

Estimation of Serum Interleukin-6 Levels: Serum IL-6 levels were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit specific for human IL-6, following the manufacturer's instructions. The assay was based on a quantitative sandwich immunoassay technique employing monoclonal antibodies directed against IL-6.

Absorbance was measured using a microplate reader at the recommended wavelength, and serum IL-6 concentrations were determined from a standard calibration curve generated using known standards. All samples were analyzed in duplicate, and the mean of the two readings was used for final analysis. Internal quality control procedures were followed to ensure assay reliability and reproducibility.

Outcome Measures: The primary outcome measure of the study was the serum IL-6 level in patients with obstructive sleep apnea and its correlation with the apnea–hypopnea index. Secondary outcomes included comparison of IL-6 levels across different grades of OSA severity and assessment of the relationship between IL-6 levels and clinical variables such as BMI and daytime sleepiness.

Statistical Analysis: Data were entered into a spreadsheet and analyzed using SPSS version 20.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Comparisons of serum IL-6 levels across obstructive sleep apnea severity groups were performed using one-way analysis of variance (ANOVA), followed by post-hoc Bonferroni correction, and analysis of covariance (ANCOVA) was applied to adjust for the effect of body mass index where appropriate. The relationship between serum IL-6 levels and AHI was assessed using Spearman's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Ethics Committee prior to commencement of the study. Written informed consent was obtained from all participants after explaining the objectives and procedures of the study. Participant confidentiality was maintained throughout the study, and all procedures were conducted in accordance with ethical principles for biomedical research involving human subjects.

Results

A total of 89 patients diagnosed with obstructive sleep apnea were included in the analysis. The mean age of the study population was 51.8 ± 10.6 years, with a male predominance (71.9%). The mean body mass index was 29.6 ± 4.3 kg/m², and the majority of patients were obese according to Asian BMI criteria. The mean Epworth Sleepiness

Scale score was 14.2 ± 4.1, indicating significant daytime sleepiness. Common comorbidities included hypertension (42.7%), type 2 diabetes mellitus (32.6%), and dyslipidemia (23.6%), reflecting a high cardiometabolic risk profile among the study participants (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics of Patients with Obstructive Sleep Apnea (n = 89)

Variable	Frequency (%)/mean ± SD
Age (years)	51.8 ± 10.6
Gender	
Male	64 (71.9)
Female	25 (28.1)
Body mass index (kg/m²)	29.6 ± 4.3
BMI category	
Overweight (BMI 23–24.9 kg/m²)	14 (15.7)
Obese (BMI ≥25 kg/m²)	63 (70.8)
Epworth Sleepiness Scale score	14.2 ± 4.1
Comorbidity	
Hypertension	38 (42.7)
Type 2 diabetes mellitus	29 (32.6)
Dyslipidemia	21 (23.6)

Values are expressed as mean ± standard deviation or number (percentage). BMI: body mass index; ESS: Epworth Sleepiness Scale.

Based on apnea–hypopnea index values obtained from overnight polysomnography, 23 patients (25.8%) had mild OSA, 28 patients (31.5%) had moderate OSA, and 38 patients (42.7%) had severe OSA. Thus, more than two-thirds of the study population had moderate-to-severe disease, consistent with the referral pattern of a tertiary care sleep clinic (Table 2).

Table 2: Distribution of Obstructive Sleep Apnea Severity Based on Apnea–Hypopnea Index

OSA Severity	AHI Range (events/hour)	Frequency (%)
Mild OSA	5–14.9	23 (25.8)
Moderate OSA	15–29.9	28 (31.5)
Severe OSA	≥30	38 (42.7)

OSA: obstructive sleep apnea; AHI: apnea–hypopnea index, expressed as events per hour of sleep.

Serum IL-6 levels showed a progressive and statistically significant increase with increasing severity of obstructive sleep apnea. Mean IL-6 levels were 3.9 ± 1.4 pg/mL in mild OSA, 6.2 ± 2.1 pg/mL in moderate OSA, and 9.1 ± 3.0 pg/mL in severe OSA. One-way ANOVA demonstrated a highly significant difference across severity groups ($p < 0.001$). Post-hoc analysis revealed significant differences between mild and moderate OSA, moderate and severe OSA, and mild and severe OSA, indicating a clear dose–response relationship between OSA severity and systemic inflammation (Table 3).

Table 3: Comparison of Serum Interleukin-6 Levels Across Obstructive Sleep Apnea Severity Groups

OSA Severity	Serum IL-6 (pg/mL), mean ± SD	P value
Mild OSA (n = 23)	3.9 ± 1.4	< 0.001
Moderate OSA (n = 28)	6.2 ± 2.1	
Severe OSA (n = 38)	9.1 ± 3.0	

IL-6: interleukin-6; values expressed as mean ± standard deviation. Statistical analysis performed using one-way ANOVA with post-hoc Bonferroni correction.

Serum IL-6 levels demonstrated a strong positive correlation with apnea–hypopnea index ($r = 0.62$, $p < 0.001$) and oxygen desaturation index ($r = 0.55$, $p < 0.001$). A moderate negative correlation was observed between IL-6 levels and minimum oxygen saturation ($r = −0.48$, $p < 0.001$). IL-6 levels also correlated positively with Epworth Sleepiness Scale scores ($r = 0.36$, $p = 0.001$) and body mass index ($r = 0.29$, $p = 0.006$). These findings suggest that IL-6 levels are closely associated with both the severity of sleep-disordered breathing and hypoxic burden (Table 4).

Table 4: Correlation Between Serum Interleukin-6 Levels and Polysomnographic and Clinical Parameters

Variable	Correlation coefficient (r)	p-value
Apnea–Hypopnea Index (AHI)	0.62	< 0.001
Minimum oxygen saturation (%)	−0.48	< 0.001
Oxygen desaturation index (events/hour)	0.55	< 0.001
Epworth Sleepiness Scale score	0.36	0.001
Body mass index (kg/m²)	0.29	0.006

AHI: apnea–hypopnea index; ODI: oxygen desaturation index; ESS: Epworth Sleepiness Scale; BMI: body mass index. Correlation assessed using Spearman's correlation coefficient.

The scatter plot demonstrates a clear positive linear relationship between serum IL-6 levels and apnea–hypopnea index. Higher IL-6 concentrations were observed with increasing AHI values, visually supporting the statistically significant correlation identified in correlation analysis.This pattern indicates a progressive increase in systemic inflammatory burden with worsening severity of obstructive sleep apnea (Figure 1).

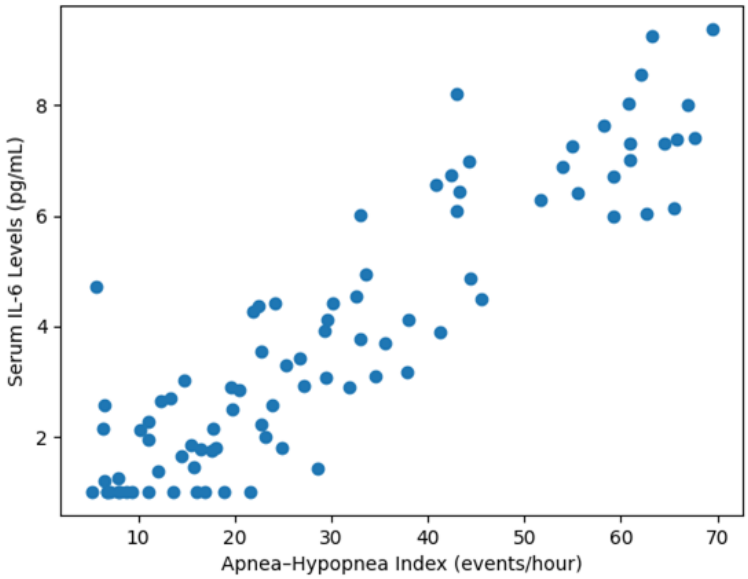

Figure 1: Scatter plot showing the relationship between serum interleukin-6 (IL-6) levels and apnea–hypopnea index (AHI) in patients with obstructive sleep apnea (n = 89)

IL-6: interleukin-6; AHI: apnea–hypopnea index (events/hour). After adjusting for body mass index using analysis of covariance, serum IL-6 levels remained significantly different across obstructive sleep apnea severity categories. The adjusted mean IL-6 levels were 4.1 ± 1.6 pg/mL in mild OSA, 6.0 ± 2.0 pg/mL in moderate OSA, and 8.7 ± 2.8 pg/mL in severe OSA. The difference across severity groups remained highly significant even after adjustment for BMI (adjusted $p < 0.001$). These findings indicate that the observed elevation in IL-6 levels is independently associated with OSA severity and is not solely attributable to obesity (Table 5).

Table 5: Comparison of Serum Interleukin-6 Levels Across Obstructive Sleep Apnea Severity After Adjustment for Body Mass Index

OSA Severity	IL-6 (pg/mL), mean ± SD	P value
Mild OSA	4.1 ± 1.6	< 0.001
Moderate OSA	6.0 ± 2.0	
Severe OSA	8.7 ± 2.8	

IL-6: interleukin-6; OSA: obstructive sleep apnea; BMI: body mass index. Values are expressed as mean ± standard deviation. Statistical analysis was performed using analysis of covariance (ANCOVA) with BMI as a covariate.

Discussion

The present study evaluated the role of the inflammatory biomarker interleukin-6 (IL-6) in patients with obstructive sleep apnea (OSA) and examined its relationship with disease severity as assessed by the apnea–hypopnea index (AHI).

In this cohort, the majority of patients had moderate to severe OSA, which is consistent with hospital-based studies by 10. Afriyie-Mensah et al., Sharma et al., and Dixit et al., from tertiary care sleep clinics, particularly in low- and middle-income countries where patients often present late in the disease course [10,11,12]. The observed male predominance and high prevalence of obesity and cardiometabolic comorbidities align with previously reported epidemiological patterns of OSA in Indian populations in studies by Kapse et al., Priyadarshini et al., and Goyal et al., [13,14,15].

A key finding of the study was the significant stepwise rise in serum IL-6 levels across mild, moderate, and severe OSA categories, with mean IL-6 concentrations nearly doubling from mild to moderate disease and showing a further marked increase in severe OSA. This graded response strongly supports a dose–response relationship between OSA severity and systemic inflammatory activation [16]. Similar trends have been reported in earlier studies by Fernandes et al., Zhong et al., and Motamedi et al., which demonstrated higher circulating IL-6 levels in patients with moderate to severe OSA compared to those with mild disease or controls [16,17,18]. However, the strength of association observed in the present study is notable and reinforces the role of IL-6 as a severity-linked biomarker rather than merely a marker of disease presence [18].

The significant positive correlation between IL-6 levels and AHI observed in this study further substantiates the link between sleep-disordered breathing severity and inflammation. AHI reflects the frequency of apneic and hypopneic events and indirectly represents the burden of repetitive intermittent hypoxia and sleep fragmentation [19].

Chronic intermittent hypoxia is known to activate nuclear factor-κB and hypoxia-inducible factor-1α signaling pathways, leading to increased transcription and release of pro-inflammatory cytokines, including IL-6 [19]. The moderate-to-strong correlation coefficient identified in this study underscores the biological plausibility of IL-6 being directly driven by hypoxic stress rather than serving as a nonspecific inflammatory marker [19]. In addition to AHI, IL-6 levels correlated positively with the oxygen desaturation index and inversely with minimum oxygen saturation, highlighting the close relationship between hypoxic burden and systemic inflammation.

These findings are consistent with mechanistic reviews by Lavalle et al., and Meliante et al., demonstrating that recurrent hypoxia–reoxygenation cycles promote oxidative stress, endothelial dysfunction, and cytokine release [20,21]. The association between IL-6 and daytime sleepiness, as reflected by Epworth Sleepiness Scale scores, suggests that inflammatory pathways may also contribute to neurocognitive and symptomatic manifestations of OSA [22].

Obesity is a well-recognized confounder in studies evaluating inflammatory markers in OSA, as adipose tissue itself is a significant source of IL-6 [23]. Importantly, even after adjusting for body mass index using analysis of covariance, the association between IL-6 levels and OSA severity remained statistically significant. This finding strengthens the argument that IL-6 elevation in OSA is not solely attributable to obesity and supports the hypothesis by Baran et al., and Shahul et al., that OSA independently contributes to systemic inflammation [23,24]. Similar observations have been reported in studies by Baran et al., and Shahul et al., that adjusted for adiposity measures and still found elevated IL-6 levels in OSA patients, emphasizing the distinct inflammatory impact of sleep-disordered breathing [23,24].

The clinical implications of these findings are substantial. Elevated IL-6 levels have been implicated in the pathogenesis of hypertension, atherosclerosis, insulin resistance, and cardiovascular disease—conditions that are highly prevalent among patients with OSA [25]. The strong association between IL-6 and OSA severity observed in this study provides a potential mechanistic link explaining the increased cardiovascular and metabolic risk associated with untreated OSA in studies by Won et al., and Adeva-Andany [25,26].

From a clinical perspective, IL-6 may serve as a useful adjunctive biomarker for risk stratification and for identifying patients with a higher inflammatory burden who may benefit from early and aggressive therapeutic interventions [27].

Limitations

Despite its strengths, the study has certain limitations. The observational design precludes causal inference, and the absence of longitudinal follow-up limits assessment of changes in IL-6 levels with treatment. Additionally, although major inflammatory confounders were excluded, residual confounding due to unmeasured variables cannot be completely ruled out. Nevertheless, the robust correlations, consistent dose–response relationship, and adjustment for obesity enhance the validity of the findings.

Conclusion

This study demonstrates that serum interleukin-6 levels are significantly elevated in patients with obstructive sleep apnea and increase progressively with disease severity. The strong positive correlation between interleukin-6 levels and apnea–hypopnea index, along with indices of nocturnal

hypoxia, highlights the role of systemic inflammation in the pathophysiology of obstructive sleep apnea.

Importantly, the association between interleukin-6 and disease severity persisted after adjustment for body mass index, indicating that inflammatory activation in obstructive sleep apnea is not solely attributable to obesity. Interleukin-6 may serve as a useful biomarker for assessing disease severity and inflammatory burden in obstructive sleep apnea.

References

1. Pham LV, Jun J, Polotsky VY. Obstructive sleep apnea. Handb Clin Neurol. 2022;189:105-36.
2. Faria A, Allen AH, Fox N, Ayas N, Laher I. The public health burden of obstructive sleep apnea. Sleep Sci. 2021;14(3):257-65.
3. Sharma SK, Ahluwalia G. Epidemiology of adult obstructive sleep apnoea syndrome in India. Indian J Med Res. 2010;131:171-5.
4. Malhotra A, Ayappa I, Ayas N, et al. Metrics of sleep apnea severity: beyond the apnea-hypopnea index. Sleep. 2021;44(7):zsab030.
5. Martinez-Garcia MA, Sánchez-de-la-Torre M, White DP, Azarbarzin A. Hypoxic Burden in Obstructive Sleep Apnea: Present and Future. Arch Bronconeumol. 2023;59(1):36-43.
6. Sforza E, Roche F. Chronic intermittent hypoxia and obstructive sleep apnea: an experimental and clinical approach. Hypoxia (Auckl). 2016;4:99-108.
7. Schaefer E, Wu W, Mark C, et al. Intermittent hypoxia is a proinflammatory stimulus resulting in IL-6 expression and M1 macrophage polarization. Hepatol Commun. 2017;1(4):326-37.
8. Imani MM, Sadeghi M, Khazaie H, et al. Evaluation of Serum and Plasma Interleukin-6 Levels in Obstructive Sleep Apnea Syndrome: A Meta-Analysis and Meta-Regression. Front Immunol. 2020;11:1343.
9. Viswanath A, Ramamurthy J, Dinesh SP, Srinivas A. Obstructive sleep apnea: awakening the hidden truth. Niger J Clin Pract. 2015;18(1):1-7.
10. Afriyie-Mensah JS, Aryee R, Aryee G, Amaning-Kwarteng E, Kankam O. Profile of Patients With Obstructive Sleep Apnea: An Initial Experience in a Tertiary Health Facility. Cureus. 2024;16(7):e64169.
11. Sharma S, Mather PJ, Efird JT, Kahn D, Shiue KY, Cheema M, Malloy R, Quan SF. Obstructive Sleep Apnea in Obese Hospitalized Patients: A Single Center Experience. J Clin Sleep Med. 2015;11(7):717-23.
12. Dixit M, Pawar S, Saket S. Obstructive Sleep Apnea Hypopnea Syndrome among Obese Patients Visiting the Outpatient Department of a Tertiary Care Centre. JNMA J Nepal Med Assoc. 2024;62(269):37-9.
13. Kapse V, Patel V, Mhaisekar D, Kulkarni M. To study the gender differences in clinical profile and risk factors for obstructive sleep apnea in a public health care setting. Int J Res Med Sci. 2019;7:2808-12.
14. Priyadarshini M, Sodhi MK, Bhatia C, Jaswal S, Aggarwal D, Saini V. Prevalence of Comorbidities in Obstructive Sleep Apnea and Association with Disease Severity: A Descriptive Study. Indian J Respir Care. 2024;13(3):152–7.
15. Goyal A, Wachinou AP, Imler T, et al. Obesity,obstructive sleep Apnea, and cardiometabolic risk: A comparative analysis of population-Based cohorts in Switzerland, India, and Benin. Sleep Breath. 2025;29(6):346.
16. Fernandes M, Spanetta M, Vetrugno G, et al. The potential role of interleukin-6 in the association between inflammation and cognitive performance in obstructive sleep apnea. Brain BehavImmun Health. 2024;42:100875.
17. Zhong A, Xiong X, Shi M, Xu H. Roles of interleukin (IL)-6 gene polymorphisms, serum IL-6 levels, and treatment in obstructive sleep apnea: a meta-analysis. Sleep Breath. 2016;20(2):719-31.
18. Motamedi V, Kanefsky R, Matsangas P, et al. Elevated tau and interleukin-6 concentrations in adults with obstructive sleep apnea. Sleep Med. 2018;43:71-6.
19. Parekh A. Hypoxic burden - definitions, pathophysiological concepts, methods of evaluation, and clinical relevance. Curr OpinPulm Med. 2024;30(6):600-6.
20. Lavalle S, Masiello E, Iannella G, et al. Unraveling the Complexities of Oxidative Stress and Inflammation Biomarkers in Obstructive Sleep Apnea Syndrome: A Comprehensive Review. Life (Basel). 2024;14(4):425.
21. Meliante PG, Zoccali F, Cascone F, et al. Molecular Pathology, Oxidative Stress, and Biomarkers in Obstructive Sleep Apnea. Int J Mol Sci. 2023;24(6):5478.
22. Fernandes M, Spanetta M, Vetrugno G, et al. The potential role of interleukin-6 in the association between inflammation and cognitive performance in obstructive sleep apnea. Brain BehavImmun Health. 2024;42:100875.
23. Baran B, Şimşek FM, Durmuş H, et al. Integrated Assessment of Obesity Indices and Novel Inflammatory Biomarkers in Predicting the Severity of Obstructive Sleep Apnea. J Clin Med. 2025;15(1):273.

24. ShahulS, Chaya SK, Khader Mathamveed S, et al. A Retrospective Study on the Association of the Systemic Inflammatory Response Index in Predicting the Severity of Obstructive Sleep Apnea. Diagnostics (Basel). 2025;15(16):2091.
25. Won CHJ, Qin L, Selim B, Yaggi HK. Varying Hypopnea Definitions Affect Obstructive Sleep Apnea Severity Classification and Association With Cardiovascular Disease. J Clin Sleep Med. 2018;14(12):1987-94.
26. Adeva-AndanyMM, Domínguez-Montero A, Castro-Quintela E, Funcasta-Calderón R, Fernández-Fernández C. Hypoxia-Induced Insulin Resistance Mediates the Elevated Cardiovascular Risk in Patients with Obstructive Sleep Apnea: A Comprehensive Review. Rev Cardiovasc Med. 2024;25(6):231.
27. Aliyeva A, Hashimli R, Yılmaz B. Hematological Biomarkers of the Obstructive Sleep Apnea Syndrome: A Machine Learning-Based Diagnostic and Prognostic Model. J Clin Med. 2025;14(23):8437.