Section: Miscellaneous

Original Research Article

SERUM BIOMARKER PROFILE IN OBSTRUCTIVE SLEEP APNOEA SYNDROME AND ITS CORRELATION WITH DISEASE SEVERITY: A CASE–CONTROL ANALYSIS

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ABSTRACT

Background: Obstructive Sleep Apnoea Syndrome (OSAS) is increasingly recognized as a multisystem disorder characterized by recurrent upper airway obstruction during sleep, resulting in intermittent hypoxia, oxidative stress, and systemic inflammation. These pathophysiological mechanisms contribute to elevated cardiovascular and metabolic risk. Identifying reliable circulating biomarkers could aid in assessing disease severity and associated systemic effects, particularly in resource-limited settings. This study aimed to evaluate the utility of selected serum markers—representing inflammation, oxidative stress, and adipokine imbalance—in patients with OSAS compared with healthy controls.

Materials and Methods: A hospital-based case–control study was conducted at a tertiary care center in North India among 167 participants, including 84 newly diagnosed OSAS patients and 83 age- and sex-matched controls. All participants underwent overnight polysomnography to confirm diagnosis and determine the Apnoea–Hypopnoea Index (AHI). Fasting venous blood samples were analyzed for serum C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), malondialdehyde (MDA), leptin, and adiponectin using standardized immunoassay techniques. Between-group comparisons were performed using t-tests or Mann–Whitney U tests, analysis of variance (ANOVA) assessed severity-based differences, and Spearman's correlation evaluated associations with AHI.

Results: Mean serum levels of CRP (4.9 ± 1.7 vs. 2.3 ± 1.0 mg/L), IL-6 (6.8 ± 2.3 vs. 3.2 ± 1.1 pg/mL), TNF-α (10.4 ± 3.1 vs. 6.1 ± 2.0 pg/mL), MDA (4.1 ± 1.2 vs. 2.8 ± 0.9 nmol/mL), and leptin (14.2 ± 4.6 vs. 9.4 ± 3.2 ng/mL) were significantly higher in OSAS cases than in controls (all $p < 0.001$), while adiponectin was significantly lower (6.1 ± 2.3 vs. 8.9 ± 2.8 µg/mL; $p < 0.001$). Biomarker levels increased progressively with disease severity (ANOVA $p < 0.001$). Significant correlations were observed between AHI and CRP ($\rho = 0.58$), IL-6 ($\rho = 0.61$), TNF-α ($\rho = 0.54$), MDA ($\rho = 0.49$), leptin ($\rho = 0.56$), and adiponectin ($\rho = –0.42$) (all $p < 0.001$).

Conclusion: Patients with OSAS exhibit a distinct biochemical profile characterized by systemic inflammation, oxidative stress, and altered adipokine regulation, which worsen with increasing disease severity. These serum markers may serve as useful adjuncts for disease assessment and cardiovascular risk stratification, especially where polysomnography resources are limited.

Keywords: Obstructive Sleep Apnoea Syndrome; Serum biomarkers; Inflammation; Oxidative stress; Adiponectin.

INTRODUCTION

Obstructive Sleep Apnoea Syndrome (OSAS) is a common yet underdiagnosed sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation, and daytime somnolence.[1] The prevalence of OSAS has been increasing globally due to rising rates of obesity, sedentary lifestyle, and ageing populations. Epidemiological studies estimate that moderate-to-severe OSAS affects approximately 9–38% of adults, with higher prevalence in males and older individuals.[2,3] Despite its frequency and significant impact on quality of life, OSAS often remains unrecognized in clinical practice, partly due to its nonspecific symptoms and the limited availability of polysomnography—the gold standard diagnostic tool.[4]

The pathophysiological consequences of OSAS extend beyond disturbed sleep architecture. Repeated cycles of hypoxia and reoxygenation induce oxidative stress, systemic inflammation, sympathetic overactivity, endothelial dysfunction, and metabolic dysregulation.[5] These mechanisms contribute to a heightened risk of cardiovascular morbidity, insulin resistance, dyslipidemia, and neurocognitive impairment.[6] Given these complex systemic effects, a growing body of research has focused on identifying circulating serum biomarkers that could reflect disease severity, predict complications, or aid in diagnosis and monitoring.[6]

Various serum markers have been explored in relation to OSAS, including C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), adiponectin, leptin, and markers of oxidative stress such as malondialdehyde (MDA).[7,8] Elevated inflammatory cytokines and acute-phase reactants have been consistently reported in OSAS patients, suggesting the presence of low-grade systemic inflammation.[9] Similarly, alterations in metabolic markers and endothelial function indicators—such as serum lipids, homocysteine, and nitric oxide metabolites—have been associated with disease severity and treatment response.[10] However, findings across studies remain heterogeneous, with inconsistencies arising from variations in study design, population characteristics, and marker selection.[9,10]

Identifying reliable serum biomarkers in OSAS could have significant clinical utility, particularly in settings where access to sleep laboratories is limited. Such markers might serve as non-invasive adjuncts for screening, risk stratification, or monitoring therapeutic efficacy following interventions like continuous positive airway pressure (CPAP) therapy or weight reduction.[11,12] In this context, the present case-control study was undertaken with an aim to evaluate the utility of selected serum markers in patients with Obstructive Sleep Apnoea Syndrome as compared to healthy controls. By correlating these biomarkers with disease presence and severity, the study aims to contribute to the growing evidence on the role of systemic inflammation and oxidative stress in OSAS pathophysiology and to explore their potential as cost-effective, easily measurable diagnostic tools.

MATERIALS AND METHODS

Study Design and Setting

This study was designed as a hospital-based, observational case–control study conducted in the Department of Internal Medicine, in collaboration with the Department of Biochemistry, at a tertiary care teaching hospital in North India. The study was carried out over a period of 2 years between January 2022 to December 2023, following approval by the Institutional Ethics Review Board. Written informed consent was obtained from all participants prior to enrolment. The study adhered to the principles outlined in the Declaration of Helsinki (2013 revision).

Study Population and Sample Size

Participants were recruited from patients attending the sleep clinic with complaints suggestive of sleep-disordered breathing, such as loud snoring, witnessed apnoeas, excessive daytime sleepiness, or non-restorative sleep. A total of 167 were enrolled, comprising 84 cases diagnosed with Obstructive Sleep Apnoea Syndrome (OSAS) and 83 age- and sex-matched healthy controls.

Inclusion and Exclusion Criteria

Cases included adults aged 30–65 years diagnosed with OSAS based on overnight polysomnography (PSG) using standard criteria of the American Academy of Sleep Medicine (AASM 2012 update), with an apnoea–hypopnoea index (AHI) ≥ 5 events/hour.

Controls were healthy volunteers without symptoms suggestive of sleep apnoea, matched for age, sex, and body mass index (BMI), and confirmed to have an AHI < 5 on PSG.

Participants with acute or chronic inflammatory diseases, recent infection (within past 4 weeks), autoimmune disorders, malignancy, hepatic or renal dysfunction, diabetes mellitus, uncontrolled hypertension, or those on anti-inflammatory or lipid-lowering therapy were excluded to avoid confounding influences on serum biomarker levels.

Clinical Evaluation

All participants underwent a detailed clinical assessment including demographic data, anthropometric measurements (height, weight, BMI, neck circumference, waist-to-hip ratio), medical and sleep history, and lifestyle factors such as smoking and alcohol use.

Daytime sleepiness was quantified using the Epworth Sleepiness Scale (ESS). Blood pressure and oxygen saturation were recorded using standardized equipment.

Polysomnography and Diagnosis of OSAS

Overnight level I polysomnography was performed for all participants using a standardized digital sleep system. Parameters recorded included electroencephalogram (EEG), electro-oculogram (EOG), submental and tibial electromyogram (EMG), nasal airflow, thoracoabdominal movements, pulse oximetry, and body position. Sleep stages and respiratory events were scored manually by a trained sleep technologist according to AASM 2012 guidelines. The Apnoea–Hypopnoea Index (AHI) was calculated as the total number of apnoeas and hypopnoeas per hour of sleep. Based on AHI, cases were categorized into mild (5–14.9), moderate (15–29.9), and severe (≥30) OSAS. Only newly diagnosed, untreated patients were included to avoid confounding by therapy effects.

Blood Sample Collection and Biochemical Analysis

Fasting venous blood samples (5 mL) were collected from all participants in the morning between 7:00 and 8:00 AM, following an overnight fast and immediately after polysomnography to minimize diurnal and short-term variability. Samples were allowed to clot and centrifuged at 3000 rpm for 10 minutes to obtain serum, which was aliquoted and stored at –80°C until analysis.

Serum concentrations of C-reactive protein (CRP) were measured using a high-sensitivity immunoturbidimetric assay. Interleukin-6 (IL-6) and Tumor Necrosis Factor-alpha (TNF-α) levels were determined using commercially available enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's protocol. Malondialdehyde (MDA), a marker of lipid peroxidation, was quantified spectrophotometrically using the thiobarbituric acid-reactive substances (TBARS) method.

In addition, routine biochemical parameters including fasting glucose, lipid profile, liver and renal function tests were assessed using automated analyzers to ensure comparable metabolic status between groups.

Quality Control and Assay Precision

All assays were performed in duplicate, and intra-assay and inter-assay coefficients of variation were maintained below 10%. Calibrators and quality-control sera were included with each assay run. Personnel performing biochemical measurements were blinded to the case–control status of samples to minimize bias.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD). Categorical variables were expressed as frequencies and percentages. Comparisons between OSAS and control groups were performed using independent Student's t-test for continuous variables and Chi-square test for categorical variables. Analysis of variance (ANOVA) with post hoc Tukey's test was used to compare serum marker levels across OSAS severity categories. Correlations between serum markers and disease severity (AHI) were assessed using Spearman's correlation coefficients. A two-tailed p-value < 0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Review Board, and written informed consent was obtained from all participants. Confidentiality of data was maintained throughout, and no financial incentives were provided.

RESULTS

The mean age of OSAS cases (46.8 ± 8.7 years) and controls (44.5 ± 9.1 years) were comparable (p = 0.12). The male predominance was observed in both groups, though the difference was not statistically significant (80.9% vs. 74.7%, p = 0.34). Mean BMI, neck circumference, and waist–hip ratio were significantly higher among OSAS patients compared to controls (p < 0.001 for all). The mean Epworth Sleepiness Scale (ESS) score was also markedly elevated in the OSAS group (13.9 ± 3.2) compared with controls (6.2 ± 2.1; p < 0.001), reflecting greater subjective daytime sleepiness in affected individuals. [Table 1]

Table 1: Baseline Demographic and Clinical Characteristics

Variables	OSAS Cases (n=84)	Controls (n=83)	p-value
	Frequency (%)/Mean ± SD		
Age (years)	46.8 ± 8.7	44.5 ± 9.1	0.120
Gender			
Female	16 (10.1%)	21 (25.3%)	0.340
Male	68 (80.9%)	62 (74.7%)	
BMI (kg/m²)	29.7 ± 3.4	25.6 ± 2.9	<0.001
Neck circumference (cm)	40.8 ± 2.8	37.2 ± 2.5	<0.001
Waist-hip ratio	0.98 ± 0.05	0.91 ± 0.04	<0.001
Epworth Sleepiness Scale (ESS) score	13.9 ± 3.2	6.2 ± 2.1	<0.001

BMI – Body Mass Index; ESS – Epworth Sleepiness Scale; OSAS – Obstructive Sleep Apnoea Syndrome.

Mean concentrations of inflammatory markers (CRP, IL-6, and TNF-α) and oxidative stress marker (MDA) were significantly higher in OSAS patients than in controls (all p < 0.001). Likewise, mean serum leptin levels were elevated in cases (14.2 ± 4.6 ng/mL)

compared with controls (9.4 ± 3.2 ng/mL; p < 0.001), whereas adiponectin levels were significantly reduced (6.1 ± 2.3 µg/mL vs. 8.9 ± 2.8 µg/mL; p < 0.001). [Table 2]

Table 2: Serum Biomarker Levels in OSAS Cases and Controls

Serum Marker	OSAS Cases (n=84)	Controls (n=83)	p-value
	Mean ± SD		
CRP (mg/L)	4.9 ± 1.7	2.3 ± 1.0	<0.001
IL-6 (pg/mL)	6.8 ± 2.3	3.2 ± 1.1	<0.001
TNF-α (pg/mL)	10.4 ± 3.1	6.1 ± 2.0	<0.001
MDA (nmol/mL)	4.1 ± 1.2	2.8 ± 0.9	<0.001
Leptin (ng/mL)	14.2 ± 4.6	9.4 ± 3.2	<0.001
Adiponectin (µg/mL)	6.1 ± 2.3	8.9 ± 2.8	<0.001

CRP – C-reactive protein; IL-6 – Interleukin-6; TNF-α – Tumour Necrosis Factor-alpha; MDA – Malondialdehyde.

As shown in Table 3, a progressive rise in serum levels of CRP, IL-6, TNF-α, MDA, and leptin was observed across mild, moderate, and severe OSAS categories, while adiponectin levels showed a corresponding decline. These trends were statistically significant for all markers (ANOVA p < 0.001).

Table 3: Serum Biomarkers Across OSAS Severity Categories

Parameter	Mild (n=28)	Moderate (n=30)	Severe (n=26)	p-value (ANOVA)
	Mean ± SD			
CRP (mg/L)	3.6 ± 1.2	5.1 ± 1.6	6.4 ± 1.8	<0.001
IL-6 (pg/mL)	4.8 ± 1.5	6.9 ± 1.8	8.2 ± 2.0	<0.001
TNF-α (pg/mL)	8.2 ± 2.4	10.1 ± 2.6	12.9 ± 3.0	<0.001
MDA (nmol/mL)	3.2 ± 0.9	3.9 ± 1.0	4.8 ± 1.2	<0.001
Leptin (ng/mL)	11.2 ± 3.8	14.0 ± 4.1	17.1 ± 4.5	<0.001
Adiponectin (µg/mL)	7.6 ± 2.4	6.3 ± 2.1	4.8 ± 1.6	<0.001

OSAS – Obstructive Sleep Apnoea Syndrome; ANOVA – Analysis of Variance; CRP – C-reactive protein; IL-6 – Interleukin-6; TNF-α – Tumour Necrosis Factor-alpha; MDA – Malondialdehyde.

Significant positive correlations were noted for CRP (ρ = 0.58, p < 0.001), IL-6 (ρ = 0.61, p < 0.001), TNF-α (ρ = 0.54, p < 0.001), MDA (ρ = 0.49, p < 0.001), and leptin (ρ = 0.56, p < 0.001), whereas adiponectin demonstrated a significant inverse correlation (ρ = –0.42, p < 0.001). [Table 4 and Figure 1]

Table 4: Correlation Between Serum Markers and Disease Severity (AHI) Using Spearman's Rank Correlation Coefficient (ρ)

Serum Marker	r-value	p-value
CRP	0.58	<0.001
IL-6	0.61	<0.001
TNF-α	0.54	<0.001
MDA	0.49	<0.001
Leptin	0.56	<0.001
Adiponectin	-0.42	<0.001

AHI – Apnoea–Hypopnoea Index; CRP – C-reactive protein; IL-6 – Interleukin-6; TNF-α – Tumour Necrosis Factor-alpha; MDA – Malondialdehyde.

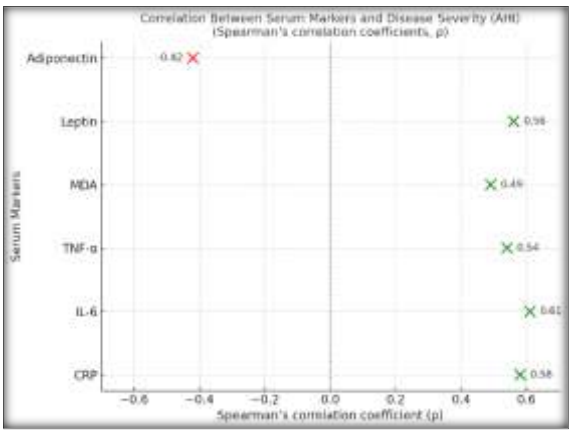

Figure 1: Correlation Between Serum Markers and Disease Severity (AHI) (Spearman's correlation coefficients, ρ)

AHI – Apnoea–Hypopnoea Index; CRP – C-reactive protein; IL-6 – Interleukin-6; TNF-α – Tumour Necrosis Factor-alpha; MDA – Malondialdehyde.

DISCUSSION

In this case–control study of 167 participants (84 OSAS cases, 83 matched controls) we found a consistent pattern of systemic inflammation, oxidative stress, and adipokine dysregulation in OSAS. In our study, the mean levels of CRP (4.9 ± 1.7 mg/L vs 2.3 ± 1.0 mg/L), IL-6 (6.8 ± 2.3 pg/mL vs 3.2 ± 1.1 pg/mL), and TNF-α (10.4 ± 3.1 pg/mL vs 6.1 ± 2.0 pg/mL) were significantly higher in cases than controls (all p < 0.001). Our findings for inflammatory cytokines (CRP, IL-6, TNF-α) align with studies by Korkmaz et al., and Liu et al., that

report elevated systemic inflammation in OSA populations and reductions after effective therapy.[13,14] Recent reviews by Lavalle et al., and Zhang et al., have documented higher CRP/IL-6/TNF-α in OSA versus controls and shown that adequate CPAP therapy can reduce these markers, particularly with good adherence and longer duration of treatment.[15,16] Mechanistically, intermittent hypoxia triggers nuclear factor-κB (NF-κB) activation and promotes production of pro-inflammatory cytokines; sympathetic activation and endothelial perturbation further amplify systemic inflammation and vascular risk.[17]

The increase in MDA—an index of lipid peroxidation—observed in our cases (4.1 ± 1.2 vs 2.8 ± 0.9 nmol/mL) corroborates prior meta-analytic data by Fadaei et al., and Pau et al., demonstrating higher circulating MDA in OSA patients compared with controls, consistent with a state of heightened oxidative stress driven by repetitive hypoxia–reoxygenation cycles.[18,19] Oxidative products such as MDA not only reflect cellular lipid injury but also can propagate endothelial dysfunction and atherogenesis, providing a plausible link between sleep-disordered breathing and cardiovascular disease.[20]

In our study, serum leptin was also elevated in OSAS (14.2 ± 4.6 ng/mL vs 9.4 ± 3.2 ng/mL; $p < 0.001$), while adiponectin was lower (6.1 ± 2.3 µg/mL vs 8.9 ± 2.8 µg/mL; $p < 0.001$). Adipokine alterations in our cohort — elevated leptin and reduced adiponectin — are also concordant with recent pooled analyses by Li et al., and Xu et al., that show leptin levels tend to be higher in OSA and correlate positively with AHI, while adiponectin often shows an inverse relationship.[21,22] These adipokine shifts likely reflect the interplay of excess adiposity and OSA-specific effects: leptin resistance, sympathetic overdrive, and hypoxia-induced changes in adipose tissue biology may raise circulating leptin, whereas adiponectin suppression may remove an anti-inflammatory, anti-atherogenic influence.[23] Importantly, although adiposity is a major determinant of leptin and adiponectin, our results show persistent biomarker associations with AHI even when considering the notable between-group BMI difference (cases 29.7 ± 3.4 kg/m² vs controls 25.6 ± 2.9 kg/m²; $p < 0.001$), suggesting that OSAS per se contributes to the observed profile — a notion supported by studies Al Mutairi et al., and Zeng et al., reporting biomarker improvement after OSA treatment independent of weight loss.[24,25]

Biomarker concentrations increased stepwise with disease severity (ANOVA $p < 0.001$ for all markers), and Spearman correlations with AHI were moderate-to-strong and directionally expected (e.g., IL-6 $\rho = 0.61$, CRP $\rho = 0.58$, leptin $\rho = 0.56$, adiponectin $\rho = -0.42$; all $p < 0.001$). From a statistical and clinical perspective, the moderate-to-strong correlations between biomarkers and AHI ($\rho \approx 0.49$–0.61 for most inflammatory/oxidative markers) indicate that these markers reflect disease burden but are not perfect proxies; the imperfect correlations remind us that AHI captures an aspect of disease severity (event frequency) while biomarkers integrate multiple pathophysiological processes (hypoxia, inflammation, obesity, comorbidity) [26,27]. Thus, rather than replacing polysomnography, a panel of serum markers may have pragmatic utility as adjuncts — for triage in resource-limited settings, for stratifying cardiovascular risk in newly diagnosed patients, or for monitoring biological response to therapy in longitudinal follow-up, particularly when combined with clinical measures such as BMI, neck circumference, and ESS.[28,29]

Limitations

Strengths of our study include well-characterized, newly diagnosed and untreated OSAS cases, contemporaneous PSG for all participants, measurement of a biologically coherent panel (inflammation, oxidative stress, adipokines), and reporting of effect sizes and dispersion (mean ± SD) suited for comparisons and meta-analysis. However, there are several limitations. First, the cross-sectional case–control design precludes causal inference and cannot determine whether biomarker abnormalities precede or follow the development of OSAS. Second, despite matching and exclusion criteria, residual confounding by obesity, metabolic comorbidity, or unmeasured lifestyle factors (diet, physical activity) may partly explain group differences; we therefore recommend multivariable adjustment in future analyses and larger cohorts to disentangle these effects. Third, single-timepoint biomarker measurements (even when fasting and standardized for timing) may be influenced by short-term variability; repeated measures or integrative indices (e.g., area under curve for nocturnal changes) could provide additional insight. Finally, while many inflammatory and oxidative markers showed robust case-control differences, there is variability in assay methods across studies which complicates absolute cut-off determination — multicenter harmonization and assay standardization will be necessary before clinical translation.

CONCLUSION

In summary, our results add to accumulating evidence that OSAS is associated with a pro-inflammatory, pro-oxidative, and adverse adipokine profile, with biomarker levels scaling with disease severity. These data support the concept that circulating markers — particularly combinations of inflammatory (CRP, IL-6, TNF-α), oxidative (MDA) and metabolic (leptin/adiponectin) indices — could serve as complementary tools for risk stratification and monitoring in OSAS. Prospective studies that measure these markers before and after definitive treatment (e.g., CPAP, weight loss, or surgical interventions), adjust rigorously for adiposity and metabolic confounders, and evaluate whether

biomarker-driven management improves clinical outcomes are logical next steps.

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