Study of Association Between Obstructive Sleep Apnea and Some Systemic Inflammatory Markers

Abstract

Background: Obstructive Sleep Apnea (OSA) is a chronic sleep-related breathing disorder and a recognized risk factor for cardiovascular disease, characterized by intermittent hypoxemia and systemic inflammation. This work assessed the relationship between OSA severity and easily accessible hematologic inflammatory markers. **Methods:** A case–control study was performed on 50 newly diagnosed OSA patients (Apnea–Hypopnea Index [AHI] >5/h) and 30 age- and sex-matched healthy controls. All subjects underwent overnight polysomnography (PSG), followed by fasting venous sampling for C-reactive protein (CRP), complete blood count (CBC), and derived indices including neutrophil-to-lymphocyte ratio (NLR), monocyte- (MLR), platelet- (PLR) to-lymphocyte ratios, systemic inflammation response index (SIRI), and aggregated index of systemic inflammation (AISI). **Results:** Compared with controls, OSA patients exhibited higher CRP, white blood cells (WBCs), neutrophils, lymphocytes, SIRI, and AISI, with lower PLR (all $p<0.05$). CRP >3 mg/L predicted OSA (AUC=0.723) and moderate–severe disease (AUC=0.851). Independent predictors of OSA included CRP, WBCs, neutrophils, and lymphocytes (OR=5.82, 1.82, 3.05, 4.68; p=0.013, 0.031, 0.028, 0.038; respectively). Predictors of moderate–severe OSA were NLR, MLR, SIRI, CRP, and reduced lymphocytes (OR=4.09, 4.74, 6.87, 1.38, 0.21; p=0.014, 0.005, 0.041, 0.018, 0.002; respectively). **Conclusion:** Elevated CRP and CBC-derived indices, particularly NLR, SIRI, MLR, and AISI, serve as independent predictors of OSA and its severity, highlighting their value as simple, inexpensive, and clinically useful markers for screening and risk stratification.

Keywords: OSA; Systemic Inflammation; CRP; NLR; PSG; Biomarkers

Introduction:

Sleep-Disordered Breathing (SDB) includes a variety of disorders that impair normal respiratory patterns during sleep. These disturbances often result in repeated upper airway obstruction, intermittent reductions in blood oxygen titres, and fragmented sleep. SDB affects approximately 4–9% of adults and nearly 3% of children. Among these disorders, obstructive sleep apnea (OSA) syndrome is the most common and clinically significant type. OSA is defined by recurrent pauses in airflow lasting ten seconds or longer despite continued respiratory effort [(1)].

The impact of OSAS extends well beyond poor sleep quality. It is an independent risk factor for cardiovascular and cerebrovascular conditions, including hypertension (HTN), cardiac arrhythmias, myocardial infarction, and stroke. Patients frequently experience loud habitual snoring, excessive daytime sleepiness, fatigue, and difficulty maintaining attention and cognitive performance during daily activities. Several factors increase the likelihood of developing OSAS, including male sex, elevated body mass index, shorter neck circumference, and the upper airway structural abnormalities like enlarged tonsils or retrognathia [(2)].

Polysomnography (PSG) remains the gold standard for diagnosing OSAS. This comprehensive evaluation allows for simultaneous assessment of sleep architecture, respiratory events, and oxygen saturation titres throughout the night. Despite its accuracy, PSG requires specialized equipment and trained personnel, which can limit its accessibility in certain healthcare settings. Effective management approaches like surgical interventions as tonsillectomy or adenoidectomy in certain cases and continuous positive airway pressure (CPAP) therapy, can enhance sleep quality, alleviate daytime sleepiness, and decrease systemic inflammation, as detected by high-sensitivity C-reactive protein (CRP) titre [(3)].

If left untreated, OSAS can contribute to a wide range of cardiometabolic and neurocognitive complications. These include type 2 diabetes mellitus (DM), heart failure, insulin resistance, stroke, mood disturbances, and a motor vehicle and occupational accidents heightened risk. The cumulative effect of these complications places a substantial burden on both patients and healthcare systems [(4)]. Professional

organizations, such as the American Heart Association, advocate routine screening for OSAS in at-risk populations, acknowledging the broad implications of the disorder [5].

Recent research has highlighted the value of hematologic inflammatory markers derived from complete blood count (CBC) testing. Indicators like neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), systemic inflammation response index (SIRI), and aggregated index of systemic inflammation (AISI) are easily obtainable and inexpensive [6]. Emerging evidence suggests that elevated titres of these markers reflect systemic inflammation and may correlate with OSAS severity, offering a practical approach to risk stratification [7].

In the current work, we investigated the association between OSAS severity and CBC-derived inflammatory indices, evaluating their potential as simple and accessible tools to aid clinical assessment and management of patients with OSAS.

Patients and methods:

Patients:

This case–control study was done at the Sleep Study Unit of the Chest Department, Benha University Hospital, over a 12-month period from March 2024 to March 2025. Ethical approval was obtained from the Research Ethics Committee, Faculty of Medicine, Benha University (Approval Code: Ms 31-2-2024). Written informed consent was obtained from all participants prior to enrollment, in accordance with the principles of the Declaration of Helsinki.

Eligibility Criteria

The study included 50 adults newly diagnosed with OSA and 30 apparently healthy volunteers matched for age and sex, who served as controls. Diagnosis of OSA was confirmed through overnight PSG, demonstrating an AHI exceeding five events per hour. Control participants had no history, symptoms, or prior diagnosis of sleep-related breathing disorders.

Participants were excluded if they declined participation or had autoimmune, rheumatologic, or malignant diseases, chronic infections, recent acute illness, surgery,

or trauma within three months, chronic pulmonary disease including COPD, neuromuscular or uncontrolled cardiac disorders, hepatic or renal insufficiency, or ongoing treatment with anti-inflammatory, immunosuppressive, or antioxidant medications

Sampling and Grouping

All patients referred for PSG during the study period were consecutively screened. 80 participants were enrolled, including 50 confirmed OSA cases and 30 matched controls.

Methods:

History Taking

Each participant underwent a comprehensive clinical evaluation, including a detailed interview that covered demographic information, smoking status, comorbidities, and current medications. Body mass index (BMI) was calculated via the standard formula :. HTN was defined as a systolic blood pressure (SBP) ≥ 140 mmHg, a diastolic blood pressure (DBP) ≥ 90 mmHg, or the current use of antihypertensive medication [8]. DM was diagnosed according to the following criteria: or or or if the patient was under active treatment for hyperglycemia [9].

Epworth Sleepiness Scale (ESS)

Daytime sleepiness was evaluated via the validated ESS, a self-administered questionnaire comprising eight items that assess the likelihood of dozing off during various routine daily activities. Each item is rated on a scale from 0 (no chance of dozing) to 3 (high chance of dozing), resulting in a total score ranging from 0 to 24. Higher scores reflect greater severity of daytime sleepiness, with a score above 10 indicating excessive sleepiness. In parallel, the STOP-Bang questionnaire was employed to assess the risk of OSA. This screening tool includes eight yes/no questions covering key clinical and demographic factors: habitual snoring, daytime fatigue, observed apneas, history of HTN, BMI >35 kg/m², age >50 years, neck circumference >40 cm, and male sex. Each affirmative response contributes one point,

and cumulative scores are used to stratify OSA risk as low (0–2), intermediate (3–4), or high (≥5).

STOP-Bang Questionnaire

The risk of OSA was evaluated via the STOP-Bang questionnaire, a widely validated screening tool. This instrument consists of eight yes/no items covering key clinical and demographic factors: loud snoring, excessive daytime sleepiness, observed apneas, a history of HTN, body mass index exceeding 35 kg/m², age over 50 years, neck circumference greater than 40 cm, and male sex. Each affirmative response is assigned one point, resulting in a cumulative score ranging from 0 to 8. Based on the total score, individuals are classified into three risk categories for OSA: a score of 0–2 indicates a low risk, 3–4 corresponds to an intermediate risk, and a score of 5 or higher signifies a high risk for the presence of OSA. This scoring system enables rapid, practical risk stratification in both clinical and research settings.

Clinical Examination

All participants underwent a complete physical examination, including cardiovascular, respiratory, abdominal, and neurological assessments. ENT evaluation was performed to identify structural factors that could contribute to airway obstruction, including tonsillar hypertrophy, nasal septal deviation, macroglossia, and retrognathia.

In addition to AHI, several other sleep-related parameters were recorded to provide a comprehensive assessment of nocturnal respiratory function. These included the ODI, RDI, snoring index, baseline oxygen saturation, lowest recorded oxygen saturation during sleep, and sleep efficiency, calculated as the proportion of total sleep time relative to the total time spent in bed.

Laboratory Investigations

After PSG and a 24-hour fast, venous blood samples were collected between 7:00–8:00 a.m. CBC analysis provided neutrophil, lymphocyte, monocyte, and platelet counts. Derived inflammatory indices were calculated, including NLR, PLR, MLR, SIRI, and AISI. Serum hs-CRP titres were also measured as an additional marker of systemic inflammation.

Results:

As opposed to controls, patients with OSA had a significantly elevated BMI, Epworth Sleepiness Scale scores, and STOP-Bang scores. Laboratory results exhibited significantly elevated CRP titres, WBC counts, neutrophils, monocytes, and lymphocytes in cases as opposed to controls. Inflammatory indices including SIRI and AISI were significantly elevated in cases, whereas PLR was significantly lower. Other parameters exhibited comparability between the two groups. **Table 1**

Among the studied cases, the median AHI was 16.65, with 38% classified as mild OSA, 48% as moderate, and 14% as severe. The median ODI was 10.6, while the median RDI was 16.7. The mean baseline oxygen saturation was 93 ± 3%, and the mean minimal oxygen saturation was 84 ± 8%. The snore index exhibited a median of 171.6, and the mean sleep efficiency was 71.8 ± 10.5%. **Table 2**

In the cases group, several significant correlations were observed. CRP exhibited a strong positive correlation with AHI. Platelet count correlated negatively with both the ESS, STOP-Bang score, and ODI. WBC count exhibited a strong negative correlation with baseline O_2 saturation. Neutrophil count and monocyte count were negatively correlated with baseline O_2 saturation, and neutrophil count was also negatively correlated with minimal O_2 saturation. Other correlations, including those with RDI, snore index, and sleep efficiency (P=0.06–0.766), were not statistically significant. **Table 3**

In the cases group, several significant correlations were detected. SIRI correlated positively with STOP-Bang and negatively with baseline O_2 saturation. AISI exhibited a negative correlation with baseline O_2 saturation. NLR correlated positively with AHI. PLR demonstrated a positive correlation with STOP-Bang and with sleep efficiency. SII was negatively correlated with baseline O_2 saturation. Other correlations, including with the ESS, ODI, RDI, O_2 saturation, and snore index, were not statistically significant. **Table 4**

ROC curve analysis was done for CRP to predict the existence of OSA. It demonstrated a significant AUC of 0.723 with a 95% confidence interval (0.612–0.834), indicating a good predictive ability. The best cutoff value was >3 mg/L, at which the

sensitivity, specificity, PPV, and NPV were 68.0%, 73.33%, 81.0%, and 57.9%, respectively. **Figure 1A**

ROC curve analysis was done for SIRI to predict OSA. It demonstrated a significant AUC of 0.640 with a 95% confidence interval (0.514–0.766), reflecting a modest predictive ability. The optimal cutoff value was >0.4, yielding a sensitivity of 74.0%, specificity of 53.33%, PPV of 72.5%, and NPV of 55.2%. **Figure 1B**

ROC curve analysis was conducted for AISI to predict OSA. It exhibited a significant AUC of 0.644 with a 95% confidence interval (0.520–0.768), indicating a fair predictive ability. The best cutoff value was >134, achieving a sensitivity of 74.0%, specificity of 66.67%, PPV of 78.7%, and NPV of 60.6%. **Figure 1C**

ROC curve analysis was done for PLR to predict OSA. It revealed a significant AUC of 0.637 with a 95% confidence interval (0.516–0.759), denoting a fair predictive value. The optimal cutoff point was ≤115, providing a sensitivity of 68.0%, specificity of 60.0%, PPV of 73.9%, and NPV of 52.9% **Figure 1D**

Logistic regression analysis was done to identify predictors of OSA. In the univariate analysis, higher CRP, WBC count, neutrophil count, lymphocyte count, and AISI were significant predictors. In the multivariate model, CRP, WBC count, neutrophil count, and lymphocyte count remained independent predictors of OSA. **Table 5**

ROC curve analysis was done for CRP to predict moderate–severe OSA. It revealed a significant AUC of 0.851 with a 95% confidence interval (0.722–0.981), indicating very good predictive ability. The best cutoff was >3.55 mg/L, at which sensitivity, specificity, PPV, and NPV were 90.3%, 78.9%, 87.5%, and 83.3%, respectively. **Figure 2A**

ROC curve analysis was done for SIRI to predict moderate–severe OSA. It exhibited a significant AUC of 0.682 with a 95% confidence interval (0.534–0.830), indicating a modest predictive ability. The best cutoff was >0.5, at which sensitivity, specificity, PPV, and NPV were 67.74%, 68.42%, 77.8%, and 56.5%, respectively. **Figure 2B**

ROC curve analysis was done for NLR to predict moderate–severe OSA. It revealed a significant AUC of 0.767 with a 95% confidence interval (0.637–0.896), indicating

good predictive ability. The best cutoff was >1.7, at which sensitivity, specificity, PPV, and NPV were 61.29%, 89.47%, 90.5%, and 58.6%, respectively. **Figure 2C**

ROC curve analysis was done for MLR to predict moderate–severe OSA. It demonstrated a significant AUC of 0.722 with a 95% confidence interval (0.574–0.871), indicating a fair predictive ability. The best cutoff was >0.14, at which sensitivity, specificity, PPV, and NPV were 80.65%, 68.42%, 80.6%, and 68.4%, respectively. **Figure 2D**

The multivariate logistic regression analysis revealed that numerous inflammatory ma rkers are independent predictors of moderate–severe OSA. Elevated CRP increased the odds by about 1.4-fold. Higher NLR was associated with a fourfold increase in risk, while elevated MLR also conferred nearly a fivefold higher risk. SIRI was an additional independent predictor, with patients showing values above the cutoff having nearly sevenfold greater odds. Conversely, higher lymphocyte counts were protective, reducing the odds of moderate–severe OSA by almost 80%. **Table 6**

Discussion:

OSA is increasingly recognized as a systemic inflammatory disease and a major cardiovascular risk factor, necessitating cost-effective diagnostic and risk stratification tools. The purpose of this case-control study, comprising 50 newly diagnosed OSA patients and 30 healthy controls, was to evaluate the association between the severity of OSA, as assessed by PSG, and readily available systemic inflammatory markers.

In the present study, OSA cases exhibited a pro-inflammatory profile with significantly elevated CRP, WBCs, neutrophils, monocytes, lymphocytes, and elevated composite indices (SIRI, AISI), alongside lower PLR. Within the OSA patients, the moderate–severe group exhibited higher CRP, SIRI, NLR, and MLR, and lower lymphocyte counts, indicating progressive systemic inflammation with disease severity. Similar findings were reported by Li and co-authors [12], whose meta-analysis of 15 studies (n=1,297) demonstrated significantly elevated CRP and hs-CRP in OSA, with pooled mean differences of +1.98 and +1.57 (both $P<0.01$). Garrido and co-authors [13] also found elevated CRP in OSA (0.47 ± 0.50 mg/dL) versus controls (0.44 ± 0.75 mg/dL; $P=0.032$). Supporting the role of monocytes, Rodríguez-Sanz and co-

authors [14] observed higher monocyte counts (555 vs 476 cells/mL; P=0.0001) and MLR (0.264 vs 0.242; P=0.0457) in OSA patients, while Jiang and co-authors [15] found increased monocyte and basophil counts correlated positively with AHI in 277 subjects, linking inflammatory burden to disease severity. Consistent with the present results, Elfeky and co-authors [16] reported that SIRI, CRP, and ESR were independently associated with moderate-to-severe OSA, with each unit rise in SIRI doubling the odds of higher severity ($P<0.05$). Mechanistically, OSA-related intermittent hypoxia and arousals activate HIF-1α/NF-κB pathways, elevating CRP and inflammatory cell indices [17, 18]. Increased NLR parallels neutrophil activation and lymphocyte suppression under hypoxic stress [19], while higher MLR reflects intensified monocyte activation and lymphocyte reduction with worsening OSA [20]. Collectively, these findings reinforce that advancing OSA severity is accompanied by escalating systemic inflammation and hematologic imbalance.

In the current study, CRP >3 mg/L proved to be a practical and informative threshold for identifying OSA, showing good discriminatory ability (AUC=0.723) and >3.55 to be an improved performance for detecting moderate–severe OSA (AUC=0.851) with high sensitivity and solid specificity. Similarly, Gaines and co-authors [21], in 60 adults with mild–moderate OSA, found that incorporating CRP into predictive models enhanced the identification of HTN (AUC=0.721) and hyperglycemia (AUC=0.813), outperforming AHI in cardiometabolic risk prediction. In agreement, Shah and co-authors [22] analyzed 2,352 adults with suspected OSA and reported a median CRP of 3.0 mg/L, with multivariable analysis showing that higher CRP correlated independently with AHI, desaturation indices, BMI, age, neck circumference, and female sex, confirming the association between increasing OSA severity and systemic inflammation.

In the present study, lymphocyte count effectively differentiated OSA patients from controls and exhibited even stronger performance in identifying moderate–severe disease, with lower counts reflecting greater hypoxic stress. In partial concordance, Yang and co-authors [23] found higher lymphocyte counts in 79 OSAS patients versus 28 controls ($P<0.05$), though lymphocytes were not independent predictors after adjustment ($P > 0.05$). Similarly, Hauber and co-authors [24] reported significantly elevated lymphocyte titres in moderate–severe than mild OSA ($P<0.05$) based on

pharyngeal lavage cytology. Mechanistic studies have linked lymphopenia to hypothalamic–pituitary–adrenal axis activation, cortisol overproduction, and sleep disruption [25-27]. Supporting these biological underpinnings, Steiropoulos and co-authors [28] observed persistently reduced total and $CD4^+$ lymphocyte counts 6 months following CPAP use (>4 h/night), and Ye and co-authors [29] demonstrated disturbances between Th17 (increased) and Treg (decreased) subsets in OSAS, indicating immune dysregulation contributing to disease pathogenesis and progression.

In the present study, SIRI demonstrated a modest ability to distinguish OSA patients and to identify moderate–severe disease, with its thresholds performing better for detecting likely cases than for exclusion. This aligns biologically with OSA-related intermittent hypoxia and sympathetic activation, which elevate neutrophils and monocytes while suppressing lymphocytes, driving higher composite SIRI values as disease burden increases. In agreement, Topuz and co-authors [30] found significantly elevated SIRI titres in OSA patients. Similarly, Shahul and co-authors [31], in 150 adults with OSA, reported progressively higher PLR, ESR, SIRI, CRP, monocyte count, and NLR with increasing severity ($P<0.05$), and ROC analysis exhibited excellent diagnostic accuracy for severe OSA (AUC=0.961, cutoff 1.105, sensitivity 92.2%, specificity 91.4%).

In the present study, AISI demonstrated a predictive ability for OSA, with the selected cutoff achieving a balanced sensitivity and specificity. This supports its value as a composite marker integrating neutrophil, monocyte, lymphocyte, and platelet activity to reflect systemic inflammatory and thrombotic responses. Mechanistically, OSA-related intermittent hypoxia and sleep fragmentation amplify oxidative stress and NF-κB/HIF-1α signaling, inducing neutrophilia, monocytosis, platelet activation, and relative lymphopenia, which together elevate AISI titres in proportion to disease severity [29, 32].

In the current study, PLR demonstrated ability to differentiate OSA patients from controls, with the selected cutoff providing a modest balance between sensitivity and specificity, more suitable for identifying probable OSA than for exclusion. In agreement, Song and co-authors [33] analyzed 290 adults across OSA severity grades and found a significant stepwise PLR increase from controls to severe cases ($p=0.001$), showing a moderate positive correlation with AHI ($r=0.417$, $p<0.001$) and an

independent association after adjustment (β=0.358, p<0.001). Similarly, Kotkat and co-authors [34] reported that in 24 adults with OSA undergoing surgery, preoperative platelet counts and PLR were significantly lower in severe versus moderate OSA (p<0.05), with AHI inversely correlated with lymphocytes and platelets, and postoperative improvements in PLR/NLR observed in severe cases, highlighting PLR's dynamic relationship with OSA severity and treatment response.

In the current study, NLR demonstrated predictive ability for identifying moderate–severe OSA, with the selected cutoff showing high specificity and strong positive predictive value, making it reliable for ruling in more severe disease. In line with the present findings, Oyama and co-authors [35] demonstrated that NLR positively correlated with OSA severity in 95 adults, with an NLR cutoff of 1.53 predicting severe OSA (AHI > 30) at AUC 0.69 (95% CI: 0.580–0.795), sensitivity 79.6%, specificity 52.3%, and OR 4.271 for severe disease. Similarly, Rha and co-authors [36], in a meta-analysis of 11 studies (n=2,259), found significantly elevated NLR in OSA as opposed to controls (SMD 0.62, 95% CI 0.29–0.94; P=0.002), with greater differences in severe OSA. Attia and co-authors [37] further confirmed elevated NLR across 26 observational studies, noting the highest NLR titres in severe OSA. Supporting these associations, Sozer and co-authors [38] emphasized that NLR, alongside the inflammatory biomarker pentraxin-3, reliably predicts OSA severity by integrating both innate (neutrophilic) and adaptive (lymphocytic) immune responses, thereby reflecting the overall systemic inflammatory load in OSA.

In this study, MLR exhibited discriminatory power for identifying moderate–severe OSA, with the chosen threshold emphasizing high sensitivity and moderate specificity, useful for flagging likely severe cases rather than definitively excluding them. MLR reflects the imbalance between innate immune activation and adaptive suppression characteristic of OSA pathophysiology. Repeated cycles of intermittent hypoxia and sleep fragmentation activate sympathetic and HIF-1α/NF-κB pathways, promoting monocyte mobilization and cytokine release (IL-6, TNF-α), which drive endothelial dysfunction and oxidative stress [39]. Concurrently, stress hormones and systemic inflammation induce relative lymphopenia through redistribution and apoptosis, producing a dose-dependent rise in MLR with increasing hypoxic burden and arousal frequency *(Pau et al., 2023*. Supporting these findings, Bao and co-authors [40], in a

retrospective cohort of 310 adults undergoing PSG, reported significantly elevated MLR in OSAHS patients as opposed to controls (18.42 [15.45–27.33] vs 15.93 [13.48–18.94]; $p<0.001$), a positive correlation with AHI ($r=0.273$; $p<0.001$), and that MLR independently predicted OSAHS (OR 1.103, 95% CI 1.059–1.149; $p<0.001$), underscoring its potential as a marker of disease existence and severity.

Limitations of the study:

This work has several limitations, including its single-center design, which restricts generalizability, and a modest sample size (50 cases and 30 controls) that limits the power for subgroup and interaction analyses. The case–control design precludes causal inference, and despite adjustments for BMI and diabetes, residual confounding from adiposity and metabolic factors may still be present.

Conclusion:

In conclusion, readily available inflammatory markers are associated with the excistence and severity of OSA. CRP, WBC, neutrophil and lymphocyte counts independently predict OSA, whereas higher NLR, MLR and SIRI and lower lymphocytes independently predict moderate–severe disease. These findings indicate that CBC-derived indices may complement clinical screening and PSG to enhance risk stratification, particularly in resource-constrained settings.

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Author Contributions

An equal and integral role was done by all authors in the conception, development, an d completion of this research.
As well as the preparation and critical revision of the manuscript, each author contribu ted to the formulation of the objectives, data acquisition, analysis, and interpretation.

All authors have evaluated and endorsed the final version and have agreed to be respo nsible for all aspects of the work.

Conflicts of Interest

The authors affirm that the conduct and results of this research were not influenced by any financial, personal, or professional conflicts of interest.
The authors' professional integrity and unbiased scholarly interpretation are evident in the work.

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Table 1: General, clinical, and laboratory characteristics of the studied groups.

		Cases (n=50)	Controls (n=30)	P-value
Age (Years)	Mean ±SD	53 ±11	49 ±16	0.193
Gender				
Males	n (%)	23 (46)	20 (66.7)	0.073
Females	n (%)	27 (54)	10 (33.3)	
DM	n (%)	4 (8)	3 (10)	1
HTN	n (%)	15 (30)	7 (23.3)	0.518
Smoking				
No	n (%)	38 (76)	20 (66.7)	0.216
Ex-Smoker	n (%)	6 (12)	2 (6.7)	
Smoker	n (%)	6 (12)	8 (26.7)	
BMI (kg/m2)	Mean ±SD	33.5 ±6.2	25.1 ±3.5	**<0.001***
Epworth Sleepiness Scale	Median (range)	17 (11 - 23)	6 (2 - 10)	**<0.001***
STOP-Bang	Median (range)	4 (3 - 7)	1 (0 - 3)	**<0.001***
CRP (mg/L)	Median (range)	4.9 (0.6 - 58.2)	3 (2 - 6)	**0.001***
WBC(x10^3/UL)	Mean ±SD	7.9 ±2.4	6 ±1.2	**<0.001***
Neutrophils (x10^3/UL)	Median (range)	4.1 (1.7 - 11.5)	3.1 (2.09 - 4.1)	**<0.001***
Monocytes (x10^3/UL)	Median (range)	0.4 (0.2 - 1.2)	0.3 (0.2 - 0.8)	**0.046***
Lymphocytes (x10^3/UL)	Mean ±SD	2.51 ±0.79	2.05 ±0.61	**0.009***
Platelets (x10^3/UL)	Mean ±SD	255 ±58	238 ±46	0.191
SII	Median (range)	425.3 (164 - 2574.2)	335 (266 - 505)	0.126
SIRI	Median (range)	0.7 (0.2 - 4.4)	0.4 (0.2 - 1.8)	**0.035***
AISI	Median (range)	172.8 (55.4 - 1287.1)	124 (54.2 - 269)	**0.032***
NLR	Median (range)	1.6 (0.6 - 8.8)	1.4 (1 - 2.5)	0.252
PLR	Median (range)	105.3 (50.9 - 236.6)	124 (75.8 - 176)	**0.041***
MLR	Median (range)	0.19 (0.07 - 0.44)	0.18 (0.07 - 0.58)	0.788

DM: Diabetes Mellitus; HTN: Hypertension; BMI: Body Mass Index; CRP: C-reactive protein; WBC: White blood cells; SII: Systemic immune-inflammation index; SIRI: Systemic inflammation response index; AISI: Aggregate index of systemic inflammation; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; MLR: Monocyte-to-lymphocyte ratio; *: Significant P-value.

Table 2: Polysomnography parameters among the studied cases.

		Cases (n=50)
Apnea-Hypopnea Index (AHI)	Median (range)	16.65 (5 - 73.9)
OSA Severity		
Mild	n (%)	19 (38)
Moderate	n (%)	24 (48)
Severe	n (%)	7 (14)
Oxygen Desaturation Index (ODI)	Median (range)	10.6 (0.5 - 73.5)
Respiratory Disturbance Index (RDI)	Median (range)	16.7 (4 - 73.9)
Baseline O2 Saturation	Mean ±SD	93 ±3
Minimal O2 Saturation	Mean ±SD	84 ±8
Snore Index	Median (range)	171.6 (3.9 - 476)
Sleep Efficiency %	Mean ±SD	71.8 ±10.5

AHI: Apnea-Hypopnea Index; OSA: Obstructive Sleep Apnea; ODI: Oxygen Desaturation Index; RDI: Respiratory Disturbance Index.

Table 3: Correlations between CRP and CBC with other parameters in cases group.

	CRP		WBC		Neutrophil		Monocyte		Lymphocytes		Platelets	
	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value
Epworth Sleepiness Scale	0.016	0.911	-0.116	0.423	-0.039	0.787	-0.079	0.587	-0.078	0.59	-	-
STOP-Bang	-0.013	0.929	0.012	0.933	0.083	0.567	0.127	0.378	-0.218	0.128	-	-
AHI	0.599	**<0.001***	0.052	0.718	0.217	0.13	-0.113	0.436	-0.198	0.169	-0.229	0.11
ODI	0.164	0.256	0.058	0.69	0.017	0.908	-0.042	0.772	0.057	0.693	-0.281	**0.048***
RDI	0.234	0.102	0.221	0.123	0.203	0.157	-0.169	0.24	0.078	0.589	-0.124	0.393
Baseline O2 Saturation	-0.135	0.348	-0.631	**<0.001***	-0.504	**<0.001***	-0.376	**0.007***	-0.182	0.205	-0.114	0.43
Minimal O2 Saturation	-0.075	0.607	-0.365	**0.009***	-0.322	**0.022***	0.007	0.963	-0.051	0.726	0.148	0.306
Snore Index	-0.105	0.469	0.132	0.36	-0.02	0.89	0.231	0.107	0.201	0.161	-0.058	0.689
Sleep Efficiency %	0.257	0.072	0.043	0.766	-0.076	0.6	0.114	0.43	-0.082	0.571	0.268	0.06

CRP: C-reactive protein; WBC: White blood cells; AHI: Apnea–Hypopnea Index; ODI: Oxygen Desaturation Index; RDI: Respiratory Disturbance Index; O_2 : Oxygen; r: Correlation coefficient; *: Significant P-value

Table 4: Correlations between CBC-derived parameters with other parameters in cases group.

	SII		SIRI		AISI		NLR		PLR		MLR	
	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value
Epworth Sleepiness Scale	-0.095	0.512	0.036	0.802	-0.003	0.984	0.071	0.625	-0.102	0.482	0.062	0.668
STOP-Bang	0.051	0.724	0.291	**0.041***	0.16	0.268	0.282	**0.047***	-0.019	0.894	0.288	**0.042***
AHI	0.239	0.094	0.152	0.294	0.105	0.469	0.399	**0.004***	0.016	0.912	0.138	0.341
ODI	0.055	0.707	0.079	0.585	0.019	0.893	0.172	0.232	-0.141	0.329	0.009	0.951
RDI	0.225	0.116	0.07	0.628	0.095	0.513	0.251	0.079	-0.082	0.569	-0.065	0.655
Baseline O2 Saturation	-0.283	**0.046***	-0.376	**0.007***	-0.427	**0.002***	-0.226	0.114	0.05	0.73	-0.274	0.054
Minimal O2 Saturation	-0.195	0.174	-0.128	0.374	-0.106	0.463	-0.206	0.15	0.153	0.288	0.043	0.765
Snore Index	-0.148	0.304	-0.009	0.948	0.055	0.703	-0.185	0.199	-0.235	0.101	0.018	0.901
Sleep Efficiency %	0.136	0.345	0.054	0.71	0.095	0.512	-0.039	0.787	0.355	**0.012***	0.224	0.117

SII: Systemic immune-inflammation index; SIRI: Systemic inflammation response index; AISI: Aggregate index of systemic inflammation; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; MLR: Monocyte-to-lymphocyte ratio; AHI: Apnea–Hypopnea Index; ODI: Oxygen Desaturation Index; RDI: Respiratory Disturbance Index; O_2 : Oxygen; r: Correlation coefficient; *: Significant P-value.

Table 5: Logistic regression analysis for prediction of OSA.

	Univariate		Multivariate	
	OR (95% CI)	**P-value**	**OR (95% CI)†**	**P-value**
CRP (mg/L)	1.415 (1.094 - 1.829)	**0.008***	5.823 (1.445 - 23.473)	**0.013***
WBC(x103/UL)	1.822 (1.28 - 2.594)	**0.001***	1.824 (1.057 - 3.146)	**0.031***
Neutrophil (x103/UL)	2.605 (1.494 - 4.544)	**0.001***	3.052 (1.126 - 8.272)	**0.028***
Monocyte (x103/UL)	7.657 (0.584 - 100.449)	0.121	23.015 (0.562 - 943.335)	0.098
Lymphocytes (x103/UL)	2.491 (1.22 - 5.088)	**0.012***	4.675 (1.088 - 20.077)	**0.038***
SIRI	2.317 (0.873 - 6.152)	0.092	2.298 (0.886 - 5.964)	0.087
AISI	1.006 (1 - 1.012)	**0.05***	1.005 (0.999 - 1.011)	0.114
PLR	0.993 (0.982 - 1.005)	0.256	0.99 (0.972 - 1.009)	0.303

C-reactive protein; WBC: White blood cells; SIRI: Systemic inflammation response index; AISI: Aggregate index of systemic inflammation; PLR: Platelet-to-lymphocyte ratio; OR: Odds ratio; CI: Confidence interval; *: Significant P-value; †:Adjusted for age, gender, DM, HTN, smoking, and BMI.

Table 6: Logistic regression analysis for prediction of Moderate – Severe OSA.

	Univariate		Multivariate	
	OR (95% CI)	**P-value**	**OR (95% CI)†**	**P-value**
CRP (mg/L)	1.424 (1.081 - 1.876)	**0.012***	1.382 (1.058 - 1.804)	**0.018***
Lymphocytes (x103/UL)	0.226 (0.082 - 0.621)	**0.004***	0.208 (0.076 - 0.571)	**0.002***
SIRI	4.592 (0.869 - 24.261)	0.073	6.866 (1.085 - 43.448)	**0.041***
NLR	4.407 (1.4 - 13.875)	**0.011***	4.088 (1.337 - 12.5)	**0.014***
MLR	2.621 (1.2 - 5.722)	**0.016***	4.738 (1.591 - 14.111)	**0.005***

CRP: C-reactive protein; SIRI: Systemic inflammation response index; NLR: Neutrophil-to-lymphocyte ratio; MLR: Monocyte-to-lymphocyte ratio; OR: Odds ratio; CI: Confidence interval; *: Significant P-value; †: Adjusted for age and gender.

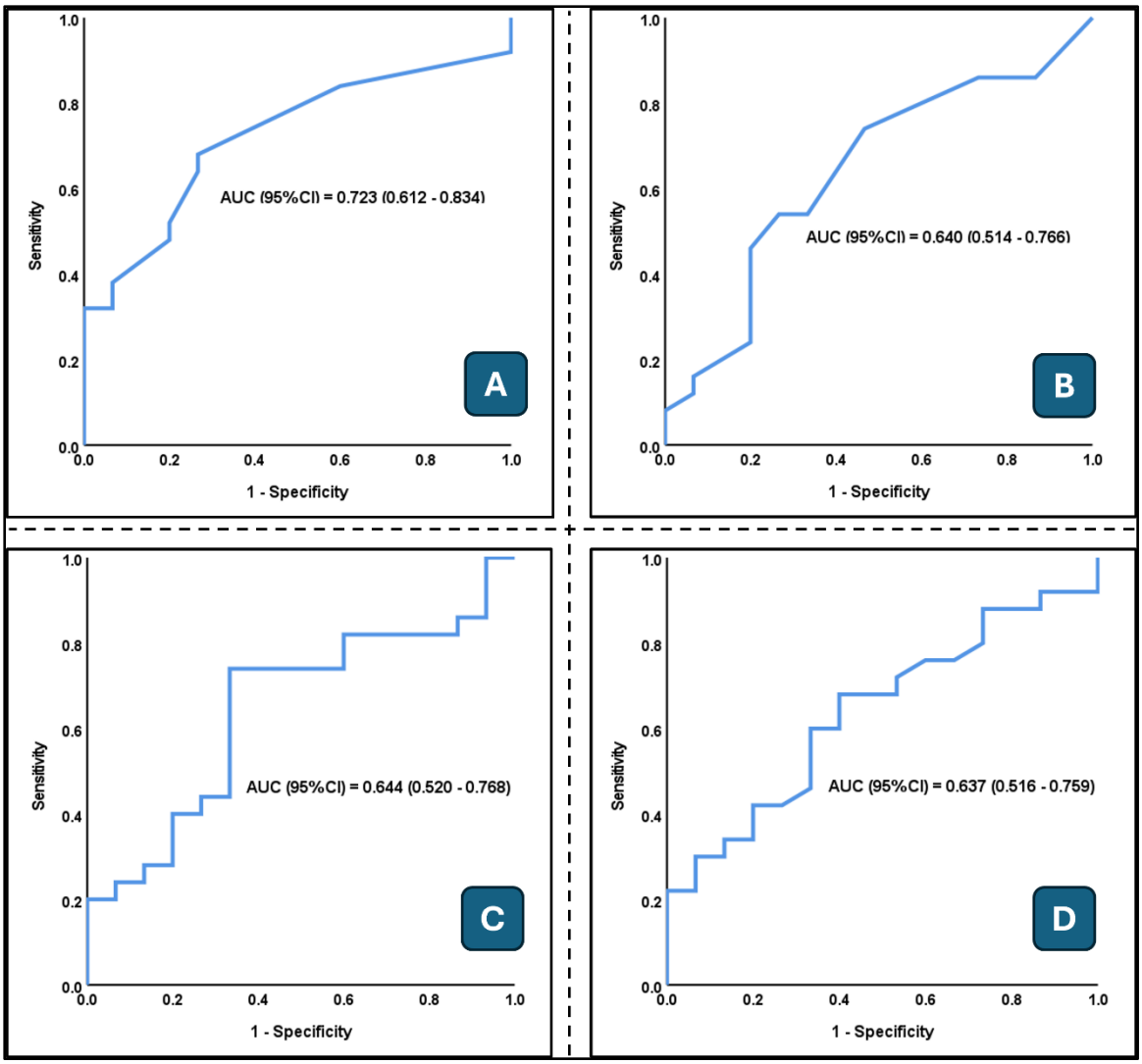

Figure 1: ROC analysis for (A) CRP, (B) SIRI, (C) AISI, and (D) PLR to predict OSA.

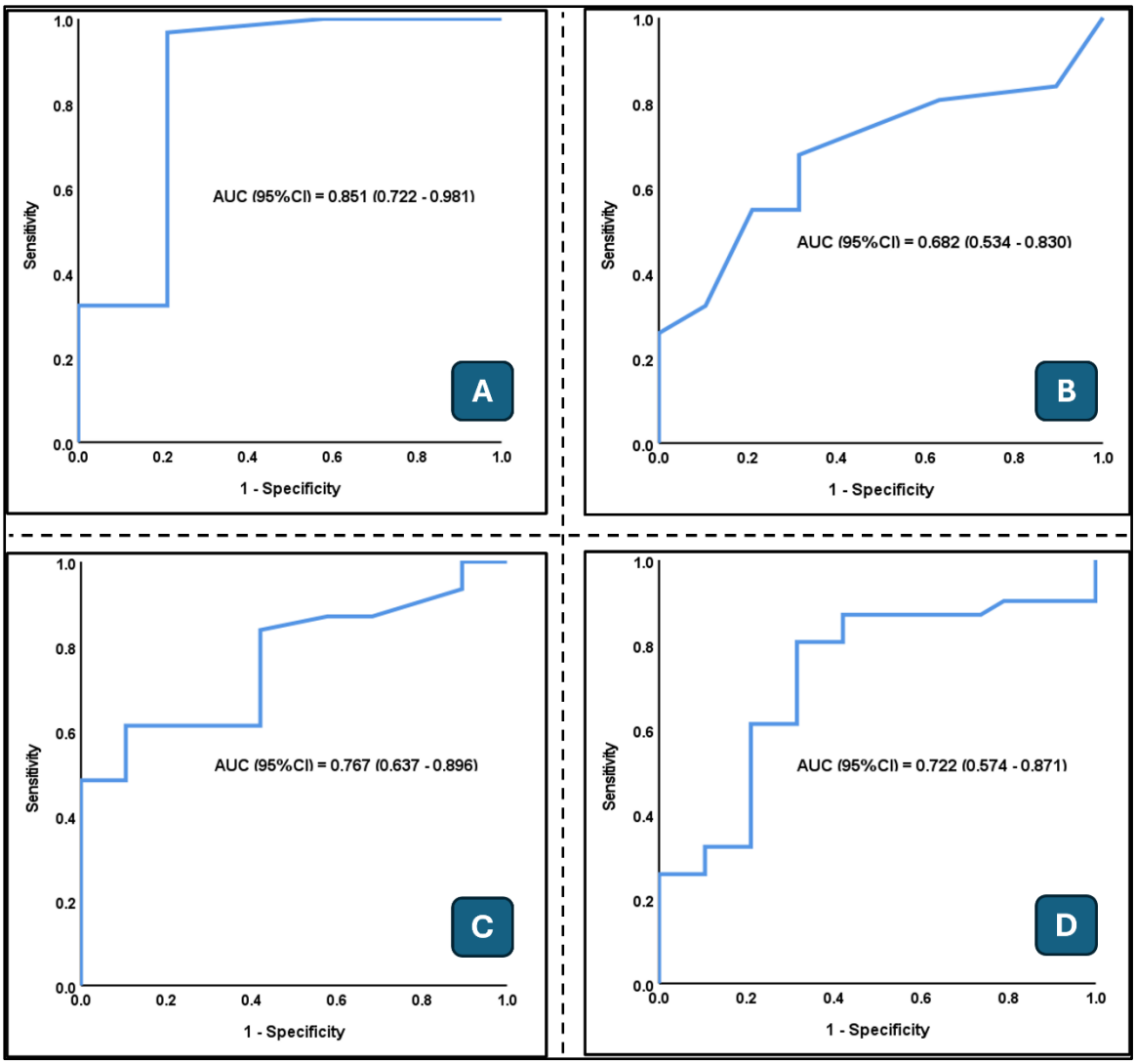

Figure 2: ROC analysis for (A) CRP, (B) lymphocytes, (C) SIRI, (D) NLR, (E) MLR to predict Moderate – Severe OSA.