e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(6); 2648-2654

Original Research Article

Screening for Comorbidities in Obstructive Sleep Apnea

Ameet H.[1], Vandini Singh[2], Shyam B. Kale[3]

[1]Assistant Professor, Department of Respiratory Medicine, Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India

[2]Associate Professor, Department of Physiology, Naraina Medical College and Research Centre, Kanpur, U.P., India

[3]Assistant Professor, Department of Community Medicine, JIET Medical College and Hospital, Jodhpur, Rajasthan, India

Received: 01-04-2026 / Revised: 25-05-2026 / Accepted: 12-06-2026

Corresponding Author: Dr. Vandini Singh

Conflict of interest: Nil

Abstract

Background: Obstructive sleep apnea (OSA) is increasingly recognized as a multisystem disorder associated with significant cardiometabolic and systemic comorbidities. However, structured screening for associated conditions remains inconsistently practiced, particularly in resource-limited settings. This study aimed to evaluate the prevalence and pattern of comorbidities among patients diagnosed with OSA and examine their relationship with disease severity.

Methods: This hospital-based cross-sectional study included 173 adult patients diagnosed with OSA by overnight polysomnography (AHI ≥5 events/hour). OSA severity was categorized as mild, moderate, and severe. Patients underwent systematic screening for hypertension, diabetes mellitus, dyslipidemia, metabolic syndrome, coronary artery disease, chronic kidney disease, chronic obstructive pulmonary disease, and neuropsychiatric disorders. Continuous variables were analyzed using ANOVA or Kruskal–Wallis test, and categorical variables using Chi-square test. Spearman's correlation was used to assess associations between AHI and comorbidity parameters.

Results: Severe OSA constituted 39.3% of cases. Hypertension (50.9%), dyslipidemia (55.5%), and diabetes mellitus (42.2%) were highly prevalent and increased significantly with OSA severity ($p<0.001$). Coronary artery disease and atrial fibrillation were significantly more common in severe OSA ($p=0.028$ and $p=0.041$, respectively). HbA1c ($\rho=0.51$), systolic blood pressure ($\rho=0.42$), triglycerides ($\rho=0.38$), and BMI ($\rho=0.46$) correlated positively with AHI (all $p<0.001$), while eGFR correlated negatively ($\rho=-0.44$, $p<0.001$). Depression was significantly associated with severe OSA ($p=0.041$).

Conclusion: OSA is associated with a substantial and severity-dependent burden of systemic comorbidities. Structured screening for cardiometabolic and organ dysfunction in OSA patients is essential for early intervention and integrated management.

Keywords: Obstructive sleep apnea; Comorbidity screening; Metabolic syndrome; Chronickidney disease; Apnea–hypopnea index.

DOI: 10.25258/ijcpr.18.6.409

Introduction

Obstructive sleep apnea (OSA) is a common yet underdiagnosed sleep-related breathing disorder characterized by recurrent upper airway collapse during sleep, resulting in intermittent hypoxia, sleep fragmentation, and sympathetic overactivity [1]. Diagnosis is established by polysomnography or validated sleep studies, and severity is defined using the apnea–hypopnea index (AHI): mild (5–14 events/hour), moderate (15–29 events/hour), and severe (≥30 events/hour) [1].

OSA represents a major global health burden. It is estimated that nearly 1 billion adults aged 30–69 years worldwide have OSA, with approximately 425 million having moderate-to-severe disease [2]. In India, prevalence ranges from 9–14% in men and 4–7% in women, increasing with obesity and advancing age [3]. Despite this high prevalence, 70–80% of affected individuals remain undiagnosed, particularly in low- and middle-income countries [2,3].

Beyond being a sleep disorder, OSA is now recognized as a multisystem disease with significant cardiometabolic, neurocognitive, and respiratory implications. Intermittent hypoxia,

oxidative stress, endothelial dysfunction, systemic inflammation, and persistent sympathetic activation play central roles in the development of associated comorbidities [4].

Cardiovascular disease is the most extensively studied comorbidity cluster. Hypertension occurs in 30–50% of OSA patients, and nearly half of individuals with resistant hypertension have coexisting OSA [5]. OSA is also independently associated with coronary artery disease, heart failure, atrial fibrillation, and stroke, with cardiovascular risk increasing proportionally to AHI severity [6].

Metabolic dysfunction is equally significant. Approximately 40–60% of patients with type 2 diabetes mellitus (T2DM) have coexisting OSA, and nearly 30% of OSA patients demonstrate impaired glucose metabolism [7]. Mechanisms include sympathetic activation, insulin resistance, adipokine imbalance, and systemic inflammation [7].

Neurocognitive impairment, excessive daytime sleepiness, mood disorders, and increased accident risk are common, particularly in moderate-to-severe OSA [8]. OSA also frequently coexists with chronic respiratory diseases such as COPD ("overlap syndrome"), chronic kidney disease, and non-alcoholic fatty liver disease [9].

Given this broad comorbidity spectrum, systematic screening in patients with OSA is essential for early detection and integrated management. However, comprehensive screening remains inconsistent in routine practice. The present study therefore aimed to evaluate the prevalence and pattern of comorbidities among patients with OSA to facilitate improved risk stratification and holistic care.

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 Departments of General Medicine and Biochemistry at a tertiary care teaching hospital of North India. The study was carried out over a period of 24 months between June 2023 to May 2025 after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment.

Study Population: Adult patients (≥18 years) presenting with symptoms suggestive of obstructive sleep apnea (OSA), such as loud snoring, witnessed apneas, excessive daytime sleepiness, non-refreshing sleep, or morning headaches, were screened for eligibility. Patients were diagnosed with obstructive sleep apnea (OSA) by attended overnight Level I polysomnography (PSG) conducted in the sleep laboratory using a computerized digital recording system. The study montage included electroencephalography (EEG), electrooculography (EOG), chin and limb electromyography (EMG), electrocardiography (ECG), nasal airflow (pressure transducer), oronasal thermistor, thoracic and abdominal respiratory effort belts, pulse oximetry, body position sensor, and snoring microphone. Sleep stages and respiratory events were scored manually according to the American Academy of Sleep Medicine (AASM) criteria. Apnea was defined as complete cessation of airflow for ≥10 seconds, and hypopnea as ≥30% reduction in airflow lasting ≥10 seconds associated with ≥3% oxygen desaturation or arousal. The apnea–hypopnea index (AHI) was calculated as the total number of apneas and hypopneas per hour of sleep. Patients with AHI ≥5 events/hour were diagnosed with OSA. Severity was categorized as mild (AHI 5–14 events/hour), moderate (15–29 events/hour), and severe (≥30 events/hour).Patients with central sleep apnea, previously treated OSA (continuous positive airway pressure therapy or upper airway surgery), acute systemic illness, pregnancy, chronic inflammatory or malignant disorders, or those unwilling to provide consent were excluded.

Clinical Evaluation: A detailed clinical history was obtained using a structured proforma, including demographic characteristics (age, sex), lifestyle factors (smoking, alcohol intake), and sleep-related symptoms. Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS). Anthropometric measurements including height, weight, body mass index (BMI), neck circumference, and waist circumference were recorded using standardized techniques. Blood pressure was measured in the sitting position using a calibrated sphygmomanometer, and the average of two readings was considered.

Sleep Study Assessment: All participants underwent overnight polysomnography (Level I) or a validated Level II sleep study as per institutional protocol. Parameters recorded included airflow (nasal pressure transducer), respiratory effort (thoracic and abdominal belts), oxygen saturation (pulse oximetry), electrocardiogram, electroencephalogram, electrooculogram, and electromyogram. Apnea was defined as complete cessation of airflow for ≥10 seconds, and hypopnea as ≥30% reduction in airflow for ≥10 seconds associated with ≥3% oxygen desaturation or arousal. The AHI was calculated as the number of apneas and hypopneas per hour of sleep.

Screening for Comorbidities: All diagnosed OSA patients were systematically screened for cardiometabolic, respiratory, renal, and neuropsychiatric comorbidities. Hypertension was defined as systolic blood pressure ≥140 mmHg

and/or diastolic blood pressure ≥90 mmHg or current use of antihypertensive medication. Diabetes mellitus was diagnosed based on fasting plasma glucose ≥126 mg/dL, HbA1c ≥6.5%, or use of antidiabetic therapy. Dyslipidemia was defined according to standard lipid profile criteria (total cholesterol ≥200 mg/dL, LDL ≥130 mg/dL, triglycerides ≥150 mg/dL, or HDL <40 mg/dL in men and <50 mg/dL in women).

Cardiovascular disease was assessed through history, clinical examination, electrocardiography, and available echocardiographic findings. Cardiovascular comorbidities were assessed through detailed history, clinical examination, and relevant investigations. Arrhythmias were identified based on resting 12-lead electrocardiography and, where indicated, 24-hour Holter monitoring, and were classified according to standard cardiology guidelines. Ischemic heart disease was documented in patients with a prior history of myocardial infarction, angina with supportive electrocardiographic changes, positive stress test, or coronary angiography findings. Heart failure was diagnosed based on clinical features (dyspnea, edema), elevated natriuretic peptides where available, and echocardiographic evidence of reduced or preserved ejection fraction in accordance with established criteria. Chronic obstructive pulmonary disease (COPD) was diagnosed using spirometry (post-bronchodilator FEV1/FVC <0.70). Renal function was evaluated using fasting serum creatinine measured by the enzymatic method, and estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation. Chronic kidney disease was defined as eGFR<60 mL/min/1.73 m² or evidence of kidney damage persisting for ≥3 months where applicable. Depression and anxiety were screened using validated standardized questionnaires such as the Patient Health Questionnaire-9 (PHQ-9) and the Generalized Anxiety Disorder-7 (GAD-7) scale. Scores were interpreted according to recommended cut-off values for mild, moderate, and severe symptomatology.

Laboratory Investigations: After an overnight fast of 8–12 hours, 5–7 mL of venous blood was collected from the antecubital vein under aseptic precautions. Samples for plasma glucose were collected in fluoride oxalate tubes and analyzed within two hours of collection. Fasting plasma glucose was measured using the glucose oxidase–peroxidase (GOD-POD) enzymatic method. Glycated hemoglobin (HbA1c) was estimated from EDTA-anticoagulated whole blood using high-performance liquid chromatography (HPLC) and reported as percentage of total hemoglobin, standardized to NGSP/DCCT reference values. Serum lipid profile, including total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol, was measured using enzymatic colorimetric assays. LDL cholesterol was calculated using the Friedewald formula where triglyceride levels were <400 mg/dL. Serum creatinine was estimated using an enzymatic method traceable to isotope dilution mass spectrometry (IDMS), and the estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 20.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Continuous variables were analyzed using one-way ANOVA or Kruskal–Wallis test as appropriate, while categorical variables were compared using Chi-square test or Fisher's exact test. Post-hoc analysis was performed where applicable. Correlation between AHI and comorbidity parameters was assessed using Spearman's correlation coefficient. A p-value <0.05 was considered statistically significant.

Results

Among 173 patients diagnosed with OSA, 48 (27.7%) had mild, 57 (32.9%) moderate, and 68 (39.3%) severe disease. Increasing OSA severity was significantly associated with older age ($p<0.001$), higher BMI ($p<0.001$), and larger neck circumference ($p<0.001$). Mean age increased from 44.6±9.2 years in mild OSA to 53.8±9.8 years in severe OSA. Similarly, BMI rose progressively from 27.8±2.9 kg/m² to 31.8±3.9 kg/m² across severity categories. ESS scores also demonstrated a significant upward trend ($p<0.001$), indicating greater daytime sleepiness with increasing severity. Male predominance increased across severity groups (62.5% in mild vs 82.4% in severe; $p=0.018$). Smoking prevalence showed no statistically significant association with OSA severity ($p=0.214$) (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics According to OSA Severity (n = 173)

Variable	Mild (n=48)	Moderate (n=57)	Severe (n=68)	p-value
	Frequency (%)/mean ± SD			
Age (years)	44.6 ± 9.2	49.3 ± 10.4	53.8 ± 9.8	<0.001
Gender				
Female	18 (37.5%)	16 (28.1%)	12 (17.6%)	0.018
Male	30 (62.5%)	41 (71.9%)	56 (82.4%)	
BMI (kg/m²)	27.8 ± 2.9	29.6 ± 3.4	31.8 ± 3.9	<0.001
Neck circumference (cm)	37.4 ± 2.1	39.2 ± 2.5	41.1 ± 2.8	<0.001
ESS score	8.9 ± 3.2	11.6 ± 4.1	14.8 ± 4.6	<0.001
Current smokers	8 (16.7%)	12 (21.1%)	20 (29.4%)	0.214

OSA: Obstructive Sleep Apnea; BMI: Body Mass Index; ESS: Epworth Sleepiness Scale.

Cardiometabolic comorbidities demonstrated a significant positive association with OSA severity. Hypertension was present in 50.9% of the cohort and increased from 31.3% in mild OSA to 66.2% in severe OSA (p<0.001). Type 2 diabetes mellitus was observed in 42.2% overall, with a marked rise across severity groups (25.0% in mild vs 57.4% in severe; p<0.001). Dyslipidemia (55.5%) and metabolic syndrome (39.9%) similarly showed significant increasing trends with severity (p=0.002 and p<0.001 respectively). Coronary artery disease prevalence was significantly higher in severe OSA (22.1%) compared to mild disease (6.3%) (p=0.028). Atrial fibrillation was predominantly observed in severe OSA (11.8%), reaching statistical significance (p=0.041) (Table 2).

Table 2: Distribution of Cardiometabolic Comorbidities Across OSA Severity Groups (n = 173)

Comorbidity	Mild (n=48)	Moderate (n=57)	Severe (n=68)	Total	p-value
	Frequency (%)				
Hypertension	15 (31.3%)	28 (49.1%)	45 (66.2%)	88 (50.9%)	<0.001
Type 2 Diabetes Mellitus	12 (25.0%)	22 (38.6%)	39 (57.4%)	73 (42.2%)	<0.001
Dyslipidemia	18 (37.5%)	31 (54.4%)	47 (69.1%)	96 (55.5%)	0.002
Metabolic Syndrome	10 (20.8%)	21 (36.8%)	38 (55.9%)	69 (39.9%)	<0.001
Coronary Artery Disease	3 (6.3%)	6 (10.5%)	15 (22.1%)	24 (13.9%)	0.028
Atrial Fibrillation	0 (0%)	3 (5.3%)	8 (11.8%)	11 (6.4%)	0.041

CAD: Coronary Artery Disease.

A significant worsening of glycemic and lipid parameters was observed with increasing OSA severity. Mean fasting plasma glucose increased from 104.6±18.5 mg/dL in mild OSA to 136.9±38.4 mg/dL in severe OSA (p<0.001). HbA1c demonstrated a progressive rise (5.9±0.7% vs 7.3±1.4%; p<0.001). Lipid abnormalities followed a similar pattern, with significant elevations in total cholesterol (p=0.003), triglycerides (p=0.001), and LDL cholesterol (p=0.002) across severity groups. Conversely, renal function declined significantly, with mean eGFR decreasing from 96.8±12.4 mL/min/1.73m² in mild OSA to 78.4±18.3 mL/min/1.73m² in severe OSA (p<0.001) (Table 3).

Table 3: Comparison of Laboratory Parameters According to OSA Severity

Parameter	Mild (n=48)	Moderate (n=57)	Severe (n=68)	p-value
	mean ± SD			
Fasting Plasma Glucose (mg/dL)	104.6 ± 18.5	118.3 ± 26.2	136.9 ± 38.4	<0.001
HbA1c (%)	5.9 ± 0.7	6.5 ± 1.1	7.3 ± 1.4	<0.001
Total Cholesterol (mg/dL)	182.4 ± 32.6	201.8 ± 41.3	219.6 ± 46.8	0.003
Triglycerides (mg/dL)	148.6 ± 48.1	176.4 ± 62.3	208.7 ± 84.9	0.001
LDL (mg/dL)	108.3 ± 28.7	123.6 ± 33.9	138.2 ± 36.5	0.002
eGFR (mL/min/1.73m²)	96.8 ± 12.4	89.2 ± 15.7	78.4 ± 18.3	<0.001

LDL: Low-Density Lipoprotein; eGFR: Estimated Glomerular Filtration Rate.

Respiratory and neuropsychiatric comorbidities were more prevalent in patients with severe OSA. COPD (overlap syndrome) was observed in 12.7% of patients and increased significantly with severity (4.2% in mild vs 20.6% in severe; p=0.032). Chronic kidney disease was identified in 10.4% overall and showed a strong association with severe OSA (19.1%; p=0.006). Depression (PHQ-9 ≥10) was present in 25.4% of the cohort and was significantly more common in severe OSA (35.3%; p=0.041). Although anxiety prevalence increased numerically with severity (12.5% in mild vs 29.4% in severe), this did not reach statistical significance (p=0.072) (Table 4).

Table 4: Respiratory and Neuropsychiatric Comorbidities Across OSA Severity Groups (n = 173)

Condition	Mild (n=48)	Moderate (n=57)	Severe (n=68)	Total (%)	p-value
	Frequency (%)				
COPD (Overlap Syndrome)	2 (4.2%)	6 (10.5%)	14 (20.6%)	22 (12.7%)	0.032
Chronic Kidney Disease	1 (2.1%)	4 (7.0%)	13 (19.1%)	18 (10.4%)	0.006
Depression (PHQ-9 ≥10)	7 (14.6%)	13 (22.8%)	24 (35.3%)	44 (25.4%)	0.041
Anxiety (GAD-7 ≥10)	6 (12.5%)	11 (19.3%)	20 (29.4%)	37 (21.4%)	0.072

COPD: Chronic Obstructive Pulmonary Disease; CKD: Chronic Kidney Disease.

Spearman's correlation analysis demonstrated significant positive associations between AHI and several cardiometabolic parameters. AHI showed a moderate positive correlation with BMI ($\rho = 0.46$, $p<0.001$) and systolic blood pressure ($\rho = 0.42$, $p<0.001$), indicating increasing obesity and blood pressure with worsening OSA severity. Glycemic status exhibited a stronger correlation, with HbA1c showing a moderate-to-strong positive relationship with AHI ($\rho = 0.51$, $p<0.001$). Triglyceride levels were also positively correlated with AHI ($\rho = 0.38$, $p<0.001$).Conversely, renal function demonstrated a significant negative correlation, as eGFR declined with increasing AHI ($\rho = -0.44$, $p<0.001$). Daytime sleepiness assessed by ESS showed the strongest association among studied variables ($\rho = 0.57$, $p<0.001$), suggesting a robust relationship between disease severity and symptom burden (Table 5).

Table 5: Correlation of Apnea–Hypopnea Index (AHI) with Selected Comorbidity Parameters (n = 173)

Variable	Spearman's ρ	p-value
BMI	0.46	<0.001
Systolic BP	0.42	<0.001
HbA1c	0.51	<0.001
Triglycerides	0.38	<0.001
eGFR	-0.44	<0.001
ESS Score	0.57	<0.001

AHI: Apnea–Hypopnea Index; BMI: Body Mass Index; HbA1c: Glycated Hemoglobin; eGFR: Estimated Glomerular Filtration Rate; ESS: Epworth Sleepiness Scale.

Discussion

The present study highlights the substantial and progressively increasing burden of systemic comorbidities among patients with obstructive sleep apnea (OSA), reinforcing the concept that OSA is a multisystem disorder rather than an isolated sleep-related breathing abnormality. Nearly 39.3% of our cohort had severe OSA, reflecting the tertiary-care referral pattern commonly reported in Indian studies by Goyal et al., Suri et al., and Ninan et al., [10,11,12]. Increasing age, male predominance, higher body mass index (BMI), and larger neck circumference were significantly associated with greater disease severity ($p<0.001$), consistent with established epidemiological evidence in the studies by Kapse et al., Dixit et al., and Afriyie-Mensah et al., identifying male sex and central obesity as major determinants of upper airway collapsibility and pharyngeal fat deposition [13,14,15].

Hypertension emerged as the most prevalent comorbidity (50.9%) and demonstrated a strong stepwise association with OSA severity (31.3% in mild vs 66.2% in severe OSA, $p<0.001$). Furthermore, systolic blood pressure correlated positively with AHI ($\rho=0.42$, $p<0.001$). These findings are concordant with prior studies by Krishna et al., and Shiinaet al., reporting hypertension in 45–70% of moderate-to-severe OSA patients [16,17]. The mechanistic basis lies in recurrent intermittent hypoxia leading to chemoreceptor-mediated sympathetic activation, renin–angiotensin–aldosterone system stimulation, oxidative stress, and endothelial dysfunction, all of which promote sustained vascular remodeling and resistant hypertension [18].

Metabolic abnormalities were equally prominent. Type 2 diabetes mellitus (42.2%) and dyslipidemia (55.5%) showed significant increases with OSA severity ($p<0.001$ and $p=0.002$, respectively). HbA1c demonstrated a moderate positive correlation with AHI ($\rho=0.51$, $p<0.001$), suggesting worsening glycemic control with increasing hypoxic burden. These results align with studies by Singh et al., and Saxena et al., indicating a 1.5–2-fold increased risk of diabetes in patients with OSA independent of obesity [19,20].

Intermittent hypoxia induces insulin resistance through adipose tissue inflammation, upregulation of inflammatory cytokines (e.g., IL-6, TNF-α), oxidative stress, and altered adipokine secretion [21]. The high prevalence of metabolic syndrome (39.9%) in our cohort further underscores the clustering of cardiometabolic risk factors in OSA. Cardiovascular complications were notably more common in severe OSA. Coronary artery disease

(22.1%) and atrial fibrillation (11.8%) were significantly higher in severe disease (p=0.028 and p=0.041, respectively). These findings parallel studies by Mandal et al., and Lin et al., demonstrating increased incidence of myocardial infarction, stroke, and arrhythmias in untreated moderate-to-severe OSA [22,23]. Pathophysiologically, repetitive hypoxemia and intrathoracic pressure swings increase myocardial oxygen demand, promote atrial remodeling, and precipitate electrical instability, predisposing to arrhythmogenesis [24].

Renal impairment was also evident. Chronic kidney disease was significantly more prevalent in severe OSA (19.1%, p=0.006), and eGFR showed a moderate negative correlation with AHI ($\rho=-0.44$, $p<0.001$). Studies by Hwu et al., and Somkearti et al., supports an independent association between OSA and renal dysfunction, potentially mediated through nocturnal hypertension, endothelial dysfunction, oxidative stress, and activation of inflammatory pathways [25,26]. Our findings reinforce the need to routinely assess renal function in patients with moderate-to-severe OSA.

Respiratory overlap syndrome (COPD with OSA) was observed in 12.7% of patients and increased significantly with OSA severity (p=0.032). Overlap syndrome has been associated with worse nocturnal desaturation and higher cardiovascular morbidity compared to either condition alone in studies by Fanaridis et al., and Alhajery et al., [27,28]. Neuropsychiatric morbidity was also significant; depression was present in 35.3% of severe OSA cases (p=0.041). Sleep fragmentation, intermittent hypoxia, and inflammatory cytokine activation are believed to alter neurotransmitter regulation, thereby contributing to mood disturbances [29,30].

Limitations

This study was conducted in a single tertiary care center, which may limit generalizability to community populations. The cross-sectional design precludes causal inference between OSA severity and comorbidities. Referral bias likely contributed to the higher proportion of severe OSA cases. Some cardiovascular diagnoses were based on prior documented records rather than uniform prospective evaluation. Additionally, inflammatory biomarkers were not assessed, which could have further strengthened mechanistic interpretation of cardiometabolic associations.

Conclusion

The present study demonstrates a high burden of cardiometabolic, renal, respiratory, and neuropsychiatric comorbidities among patients with obstructive sleep apnea, with a clear stepwise increase across disease severity. Hypertension, diabetes mellitus, dyslipidemia, metabolic syndrome, and chronic kidney disease were significantly associated with higher AHI values. Correlation analysis further confirmed that worsening OSA severity parallels adverse metabolic and cardiovascular profiles. These findings reinforce the concept of OSA as a multisystem disorder and highlight the critical importance of systematic comorbidity screening in all diagnosed patients. Early identification and integrated multidisciplinary management may help reduce long-term cardiovascular and metabolic complications, particularly in high-risk populations.

References

1. Abbasi A, Gupta SS, Sabharwal N, et al. A comprehensive review of obstructive sleep apnea. Sleep Sci. 2021;14(2):142-154.
2. Iannella G, Pace A, Bellizzi MG, et al. The Global Burden of Obstructive Sleep Apnea. Diagnostics (Basel). 2025;15(9):1088.
3. Verma M, Esht V, Alshehri MM, et al. Factors Contributing to the Change in Overweight/Obesity Prevalence Among Indian Adults: A multivariate decomposition analysis of data from the National Family Health Surveys. AdvTher. 2023;40(12):5222-5242.
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. Palmiero P, Amati F, Bombini L, Ciccone MM, Maiello M. Beyond Sleep: The Cardiovascular Impact of Obstructive Sleep Apnea Syndrome. J Clin Med. 2026;15(3):1239.
6. Gao J, Shi L, Zhu X, Liu J. Association of obstructive sleep apnea with cardiometabolic diseases and cardiovascular mortality. Clin Respir J. 2023;17(8):764-770.
7. Wondie A, Taderegew MM, Girma B, et al. Obstructive sleep apnea risk and its associated factors among type 2 diabetes mellitus patients at wolkite university specialized hospital, Wolkite, Southern Ethiopia, 2021. A comparative cross-sectional study. Diabetol Metab Syndr. 2022;14(1):157.
8. Lal C, Weaver TE, Bae CJ, Strohl KP. Excessive Daytime Sleepiness in Obstructive Sleep Apnea. Mechanisms and Clinical Management. Ann Am Thorac Soc. 2021;18(5):757-768.
9. Qiang S, Wan R, Wu J, Wang C, Cui X, Zhang Y. Screening and Verification COPD-OSA Overlap Syndrome Core Genes Using Bioinformatics. Int J Chron Obstruct Pulmon Dis. 2025;20:2601-2614.
10. Goyal A, Pakhare A, Pavirala ST, et al. Prevalence and association analysis of obstructive sleep apnea in India: Results from BLESS cohort. Sleep Med. 2025;125:128-135.
11. Suri TM, Ghosh T, Mittal S, Hadda V, Madan K, Mohan A. Systematic review and meta-

analysis of the prevalence of obstructive sleep apnea in Indian adults. Sleep Med Rev. 2023;71:101829.
12. Ninan M, Veni KS, Balachandran J, Sukumaran P, Thomas B. Clinical profile of patients with moderate to severe obstructive sleep apnea syndrome in a centre from South Kerala. IP Indian J ImmunolRespir Med. 2020;5(3):146-151.
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;7:2808-2812.
14. 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):37-39.
15. 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.
16. Krishna SVS, Rana S, Singh P, Choudhary R, Sharma A. A Study on Prevalence of Asymptomatic Pulmonary Hypertension in Patients of Metabolic Syndrome with Obstructive Sleep Apnea. Ann Afr Med. 2025; 24(2): 345-349.
17. Shiina K. Obstructive sleep apnea -related hypertension: a review of the literature and clinical management strategy. Hypertens Res. 2024; 47(11):3085-3098.
18. Min HJ, Cho YJ, Kim CH, et al. Clinical Features of Obstructive Sleep Apnea That Determine Its High Prevalence in Resistant Hypertension. Yonsei Med J. 2015;56(5):1258-1265.
19. Singh A, Chaudhary SC, Gupta KK, et al. Prevalence of obstructive sleep apnea in diabetic patients. Ann Afr Med. 2021; 20(3): 206-211.
20. Saxena P, Singh D, Singh Y. Prevalence and impact of obstructive sleep apnea in type 2 diabetes mellitus: A descriptive cross-sectional study. Med J Armed Forces India. 2024; 80(Suppl 1):S50-S56.
21. 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):326-337.
22. Mandal S, Kent BD. Obstructive sleep apnoea and coronary artery disease. J Thorac Dis. 2018;10(Suppl 34):S4212-S4220.
23. Lin YS, Liu PH, Chu PH. Obstructive Sleep Apnea Independently Increases the Incidence of Heart Failure and Major Adverse Cardiac Events: A Retrospective Population-Based Follow-Up Study. ActaCardiol Sin. 2017;33(6):656-663.
24. Arnaud C, Bochaton T, Pépin JL, Belaidi E. Obstructive sleep apnoea and cardiovascular consequences: Pathophysiological mechanisms. Arch Cardiovasc Dis. 2020;113(5):350-358.
25. Hwu DW, Lin KD, Lin KC, Lee YJ, Chang YH. The association of obstructive sleep apnea and renal outcomes-a systematic review and meta-analysis. BMC Nephrol. 2017;18(1):313.
26. Somkearti P, Chattakul P, Khamsai S, et al. Predictors of chronic kidney disease in obstructive sleep apnea patients. Multidiscip Respir Med. 2020;15(1):470.
27. Fanaridis M, Bouloukaki I, Stathakis G, et al. Prevalence and Characteristics of Patients with Obstructive Sleep Apnea and Chronic Obstructive Pulmonary Disease: Overlap Syndrome. Life (Basel). 2024;14(5):547.
28. Alhajery MA. The Overlap Syndrome: A Combination of Chronic Obstructive Pulmonary Disease and Obstructive Sleep Apnea. Cureus. 2024;16(1):e52349.
29. Alswat AM, Qanash S, Aldahlawi A, et al. The Risk of Obstructive Sleep Apnea Among Patients With Major Depressive Disorder and Generalized Anxiety Disorder in Saudi Arabia: A Cross-Sectional Study. Cureus. 2026;18(1):e101134.
30. Maruani J, Boiret C, Leseur J, et al. Major depressive episode with insomnia and excessive daytime sleepiness: A more homogeneous and severe subtype of depression. Psychiatry Res. 2023;330:115603.