



ASSOCIATION OF OBSTRUCTIVE SLEEP APNEA SEVERITY WITH METABOLIC SYNDROME AND HYPERTENSION: A CROSS-SECTIONAL STUDY

Medicine

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is increasingly recognized as an important contributor to cardiometabolic morbidity. Its relationship with metabolic syndrome and individual metabolic components, particularly across different levels of disease severity, remains an area of active investigation. **Objectives:** To evaluate the association between the severity of obstructive sleep apnea and the prevalence of metabolic syndrome and its individual components, with particular emphasis on hypertension. **Methods:** This cross-sectional study included 128 consecutive adult patients undergoing overnight polysomnography. OSA was defined using apnea-hypopnea index (AHI) thresholds of ≥ 5 and ≥ 10 events/hour. Metabolic syndrome and its components were assessed using standard clinical and biochemical criteria. Comparisons were made between OSA and non-OSA groups, as well as between mild and moderate-to-severe OSA. Statistical analyses included independent-sample tests and chi-square analysis, with $p < 0.05$ considered significant. **Results:** The mean age of the patients was 61.86 ± 11.98 years, and 60.2% were male. Patients with OSA were significantly older and more likely to be male than non-OSA patients. Hypertension was significantly more prevalent in patients with OSA ($p = 0.01$). Using an AHI ≥ 10 threshold, metabolic syndrome ($p = 0.004$) and hypertension ($p < 0.001$) were significantly more common in patients with moderate-to-severe OSA. Hyperglycemia and dyslipidemia did not show significant associations with OSA severity. **Conclusion:** Increasing severity of obstructive sleep apnea is significantly associated with a higher prevalence of hypertension and metabolic syndrome. These findings highlight the importance of early identification and risk stratification of patients with OSA to mitigate cardiometabolic risk.

KEYWORDS

Apnea-hypopnea Index; Cardio-metabolic Risk; Hypertension; Metabolic Syndrome; Obstructive Sleep Apnea

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 hypoxemia and sleep fragmentation. Epidemiological studies have demonstrated that OSA affects a substantial proportion of the adult population and is associated with increased cardiovascular morbidity and mortality (1–4). Large community-based investigations, including the Sleep Heart Health Study, have established strong associations between sleep-disordered breathing and cardiovascular disease, particularly systemic hypertension (1, 3, 4, and 15). Prospective cohort studies further support a temporal relationship between OSA and the development of hypertension, emphasizing the clinical importance of early identification and treatment of OSA (3). Metabolic syndrome represents a cluster of interrelated cardio-metabolic risk factors, including central obesity, insulin resistance, dyslipidemia, and hypertension, and is a well-recognized predictor of cardiovascular disease and all-cause mortality (8–12). Population-based studies have consistently reported a high prevalence of metabolic syndrome and its strong association with adverse cardiovascular outcomes across diverse populations (9–12). Several pathophysiological mechanisms have been proposed to explain the relationship between OSA and metabolic dysfunction. Intermittent hypoxia and sleep fragmentation lead to increased sympathetic activity, oxidative stress, endothelial dysfunction, and systemic inflammation, all of which contribute to the development of hypertension and metabolic abnormalities (7, 13). Clinical studies have also demonstrated that OSA is independently associated with insulin resistance, even after adjustment for obesity and other confounding factors (6). Evidence suggests that the cardiometabolic consequences of OSA may be influenced by disease severity. Patients with moderate-to-severe OSA appear to have a higher burden of metabolic abnormalities and cardiovascular risk compared with those with mild disease (5). However, data from hospital-based cohorts examining the association between OSA severity and individual components of metabolic syndrome remain limited. Therefore, the present study was undertaken to evaluate the association between the severity of obstructive sleep apnea and the prevalence of metabolic syndrome and its individual components, with particular emphasis on hypertension, in a hospital-based cohort of adult patients.

MATERIALS AND METHODS

This cross-sectional study included 128 consecutive adult patients referred for evaluation of suspected sleep-disordered breathing. All participants underwent overnight polysomnography. The study

protocol was reviewed and approved by the Institutional Ethics Committee of Government Medical College, Jammu (Approval No. IEC/GMCJ/2025/1971).

The sample size was calculated to detect a significant difference in the prevalence of metabolic syndrome between patients with mild OSA and those with moderate-to-severe OSA. Assuming a prevalence of 40% in the mild group and 60% in the moderate-to-severe group, with 80% power and a two-sided α error of 0.05, the minimum sample size required was 118 patients. To compensate for possible dropouts or incomplete records, 128 patients were finally analyzed. Adults aged ≥ 18 years referred for evaluation of suspected sleep-disordered breathing who underwent overnight polysomnography and provided informed consent were included. Patients with prior treatment for obstructive sleep apnea, central sleep apnea, chronic pulmonary or cardiac disease, chronic renal or liver disease, pregnancy, psychiatric illness, or incomplete clinical data were excluded.

Sleep Study and OSA Classification: Polysomnography was performed and scored according to standard criteria.⁶ The apnea-hypopnea index (AHI) was calculated as the number of apnea and hypopnea events per hour of sleep. OSA was defined as AHI ≥ 5 events/hour, and moderate-to-severe OSA as AHI ≥ 10 events/hour. Assessment of Metabolic Syndrome: Metabolic syndrome and its components (hypertension, hyperglycemia, and dyslipidemia) were assessed using standard clinical and laboratory measurements obtained at baseline. Statistical Analysis: Statistical analysis was performed using IBM SPSS Statistics version 21.0. Continuous variables are presented as mean $\pm$ standard deviation, and categorical variables are expressed as frequency and percentage. Group comparisons were performed using the independent samples t-test for continuous variables and the chi-square test for categorical variables. Associations between obstructive sleep apnea severity and metabolic abnormalities were evaluated using chi-square analysis. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1. Distribution of Patients According to their Demographical Profile

Demographical profile	No. of patients (n=128)	Percentage (%)
Age (in years)		
25-35	12	9.4
36-45	27	21.1

46-55	29	22.7
56-65	36	28.1
>66	24	18.8
Mean age $\pm$ S.D	61.86 $\pm$ 11.98	
Gender		
Female	51	39.8
Male	77	60.2
Clinical characteristics		
BMI (Mean $\pm$ S.D)	30.14 $\pm$ 6.76	
AHI (Mean $\pm$ S.D)	23.65 $\pm$ 12.86	

A total of 128 patients were included in the study. The mean age of the study population was 61.86 $\pm$ 11.98 years, with the highest proportion of patients in the 56–65-year age group (28.1%), followed by those aged 46–55 years (22.7%). Patients aged 25–35 years constituted the smallest proportion (9.4%). Males predominated in the cohort, accounting for 60.2% of participants, while females comprised 39.8%. The mean body mass index (BMI) of the study population was 30.14 $\pm$ 6.76 kg/m², indicating an overall overweight to obese profile. The mean apnea–hypopnea index (AHI) was 23.65 $\pm$ 12.86 events/hour, consistent with a predominance of moderate obstructive sleep apnea severity in the cohort.

Table 2. Comparison of OSA Versus Non OSA Patients with Respect to Age, Gender, BMI and AHI

OSA versus Non OSA patients	Clinical characteristics			
	Age (Mean $\pm$ S.D)	Female (N)	BMI (Mean $\pm$ S.D)	AHI (Mean $\pm$ S.D)
AHI < 5 (n=34)	49.36 $\pm$ 7.11	21	29.40 $\pm$ 7.23	3.18 $\pm$ 0.88
AHI $\geq$ 5 (n=94)	60.40 $\pm$ 11.55	30	31.98 $\pm$ 6.5	28.91 $\pm$ 17.12
p-value	<0.001*	0.002*	0.056	<0.001*
AHI < 10 (n=45)	55.34 $\pm$ 10.13	28	31.55 $\pm$ 6.95	5.18 $\pm$ 2.65
AHI $\geq$ 10 (n=83)	65.61 $\pm$ 12.56	23	32.60 $\pm$ 7.80	33.80 $\pm$ 21.34
p-value	<0.001*	<0.001	0.451	<0.001*

*indicates statistically significant, student t-test used to test significance.

Comparison of patients with and without obstructive sleep apnea (OSA) demonstrated significant differences in demographic and clinical characteristics (Table 2). Patients with OSA (AHI $\geq$ 5) were significantly older than non-OSA patients (60.40 $\pm$ 11.55 versus 49.36 $\pm$ 7.11 years; p < 0.001) and had a significantly lower proportion of females (31.9% versus 61.8%; p = 0.002). Mean body mass index (BMI) did not differ significantly between OSA and non-OSA groups (31.98 $\pm$ 6.50 versus 29.40 $\pm$ 7.23 kg/m²; p = 0.056). As expected, mean AHI was markedly higher in the OSA group (28.91 $\pm$ 17.12 versus 3.18 $\pm$ 0.88 events/hour; p < 0.001). When OSA severity was assessed using an AHI $\geq$ 10 threshold, patients with moderate-to-severe OSA were again significantly older (65.61 $\pm$ 12.56 versus 55.34 $\pm$ 10.13 years; p < 0.001) and predominantly male (female sex: 27.7% versus 62.2%; p < 0.001) compared to those with AHI < 10. BMI remained comparable between groups (32.60 $\pm$ 7.80 versus 31.55 $\pm$ 6.95 kg/m²; p = 0.451). Mean AHI differed significantly between severity groups (33.80 $\pm$ 21.34 versus 5.18 $\pm$ 2.65 events/hour; p < 0.001). These findings indicate that age and male sex are strongly associated with both the presence and severity of OSA, while BMI did not differ significantly between OSA and non-OSA groups.

Table 3. Prevalence of Metabolic Syndrome and Its Components (N = 128)

Comparison by OSA Definition (AHI $\geq$ 5 versus AHI < 5)

Diagnosis	AHI $\geq$ 5 (n=94)	AHI < 5 (n=34)	p-value
Metabolic syndrome	53 (56%)	15 (44%)	0.08
Hyperglycemia	28 (30%)	10 (29%)	0.64
Dyslipidemia	53 (56%)	18 (53%)	0.69
Hypertension	68 (72%)	18 (53%)	0.01*

* indicates statistically significant, chi-square test is used to test significance.

Comparison by OSA Severity (AHI $\geq$ 10 versus AHI < 10)

Diagnosis	AHI $\geq$ 10 (n=83)	AHI < 10 (n=45)	p-value
Metabolic syndrome	50 (60%)	18 (40%)	0.004*
Hyperglycemia	27 (33%)	12 (27%)	0.30
Dyslipidemia	46 (55%)	23 (51%)	0.62
Hypertension	64 (77%)	23 (51%)	<0.001*

* indicates statistically significant, chi-square test is used to test significance.

The prevalence of metabolic syndrome and its components varied according to the presence and severity of obstructive sleep apnea (OSA). When OSA was defined as an apnea–hypopnea index (AHI) $\geq$ 5 events/hour, hypertension was significantly more common among patients with OSA compared to those without OSA (72% versus 53%; p = 0.01), while metabolic syndrome showed a higher prevalence in the OSA group without reaching statistical significance (56% versus 44%; p = 0.08). No significant differences were observed in the prevalence of hyperglycemia or dyslipidemia between the two groups. When disease severity was assessed using an AHI $\geq$ 10 threshold, both metabolic syndrome (60% versus 40%; p = 0.004) and hypertension (77% versus 51%; p < 0.001) were significantly more prevalent among patients with moderate-to-severe OSA. In contrast, hyperglycemia and dyslipidemia remained comparable between severity groups. These findings indicate a clear severity-dependent association between OSA and cardiometabolic risk, driven primarily by hypertension and the overall burden of metabolic syndrome.

DISCUSSION

In this study, we found that the severity of obstructive sleep apnea (OSA) is closely linked to cardiometabolic risk, particularly hypertension and metabolic syndrome. Patients with moderate-to-severe OSA had a much higher burden of these conditions than those with milder disease, highlighting why severity assessment should be a routine part of OSA evaluation. The association between OSA and hypertension observed in our cohort is consistent with what has been reported in large community-based and longitudinal studies, which have shown that sleep-disordered breathing independently increases the risk of developing high blood pressure (1,3, 4). Repeated episodes of intermittent hypoxia and sleep fragmentation lead to sustained sympathetic activation, endothelial dysfunction, and vascular changes, all of which contribute to the development of hypertension (7, 13). Our findings reinforce this biological link and support routine blood pressure screening in patients diagnosed with OSA. We also observed a clear increase in the prevalence of metabolic syndrome with increasing OSA severity. Similar associations have been described in earlier studies, suggesting that OSA and metabolic syndrome often coexist and may share common pathophysiological pathways (5,8,10,11). Intermittent hypoxia, systemic inflammation, oxidative stress, and hormonal dysregulation are believed to promote insulin resistance and central obesity, providing a plausible explanation for this relationship (6,7). Interestingly, mild OSA did not show a strong association with metabolic syndrome in our cohort, suggesting that metabolic consequences may become clinically evident only beyond a certain severity threshold.

In contrast, hyperglycemia and dyslipidemia were not significantly associated with OSA severity in this study. This finding is in line with some hospital-based reports and may reflect the influence of factors such as ongoing treatment, dietary habits, and duration of metabolic disease, which were not assessed in detail here (9,12). As expected, older age and male sex were strongly associated with both the presence and severity of OSA, consistent with known epidemiological patterns (2). However, body mass index did not differ significantly between OSA and non-OSA groups, indicating that factors other than obesity—such as upper airway anatomy and neuromuscular control—may play an important role in determining OSA severity in this population. Taken together, these findings have clear clinical relevance. Identifying patients with moderate-to-severe OSA should prompt clinicians to actively screen for hypertension and metabolic syndrome. Integrating cardiometabolic assessment into routine sleep clinics and general medical practice could help reduce long-term cardiovascular risk through earlier intervention and comprehensive care.

CONCLUSION

This cross-sectional study demonstrates a significant association between the severity of obstructive sleep apnea and cardiometabolic risk, particularly hypertension and metabolic syndrome. Patients with moderate-to-severe OSA showed a substantially higher prevalence of both conditions compared to those with milder disease. In contrast, hyperglycemia and dyslipidemia were not significantly associated with OSA severity in this cohort. These findings underscore the importance of early diagnosis, severity-based risk stratification, and comprehensive cardiometabolic assessment in patients with obstructive sleep apnea. Routine screening for hypertension and metabolic syndrome should be integrated into the clinical evaluation of patients with moderate-to-severe OSA to reduce long-term cardiovascular morbidity.

Limitations: Several limitations should be acknowledged. First, the cross-sectional design precludes causal inference between OSA severity and metabolic abnormalities. Second, this was a single-center, hospital-based study, which may limit the generalizability of the findings to the broader population. Third, the relatively modest sample size may have reduced the ability to detect associations with certain metabolic components such as hyperglycemia and dyslipidemia. Finally, potential confounders such as physical activity, dietary habits, medication use, and duration of metabolic disorders were not assessed. Future large, multicenter, prospective studies are needed to confirm these findings and explore causal mechanisms.

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