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Autonomic Dysfunction and Its Association with Obstructive Sleep Apnea Severity: A Cross-Sectional Study Using Level III Sleep Monitoring

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is characterized by recurrent upper airway obstruction, intermittent hypoxia, and sleep fragmentation, which contribute to autonomic nervous system (ANS) dysfunction and increased cardiovascular risk. This study evaluated the association between autonomic dysfunction and OSA severity.

Methods: In this cross-sectional study, 84 adults with suspected OSA underwent overnight evaluation using the Deck Mount Level III Sleep Apnea Monitoring Device. OSA severity was classified based on the apnea–hypopnea index (AHI) as mild, moderate, or severe. Demographic characteristics, oxygen saturation indices, and autonomic function parameters including heart rate variability (HRV), Valsalva ratio, deep breathing expiratory-to-inspiratory (E:I) ratio, and orthostatic blood pressure response were analyzed. Correlations between autonomic parameters and AHI were assessed.

Results: Of the 84 participants, 13 (15.5%) had mild OSA, 32 (38.1%) had moderate OSA, and 39 (46.4%) had severe OSA. Increasing OSA severity was associated with higher body mass index, greater oxygen desaturation, and worsening autonomic dysfunction. HRV decreased significantly across mild, moderate, and severe OSA groups (42.6 ± 8.1 ms, 36.4 ± 7.5 ms, and 29.8 ± 6.9 ms, respectively; $p<0.001$). Significant reductions were also observed in Valsalva ratio and E:I ratio. AHI showed a significant negative correlation with HRV ($r=-0.52$, $p<0.001$) and a positive correlation with orthostatic blood pressure fall ($r=0.46$, $p=0.001$).

Conclusion: Autonomic dysfunction worsens with increasing OSA severity and is characterized by impaired parasympathetic activity and altered cardiovascular autonomic regulation. Assessment of autonomic function may enhance cardiovascular risk stratification in patients with OSA.

Key-words: Obstructive sleep apnea, Autonomic dysfunction, Heart rate variability, Apnea–hypopnea index, Cardiovascular risk

INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder. It is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. These episodes lead to intermittent hypoxia, hypercapnia, sleep fragmentation, and repeated arousals. It is estimated that nearly one billion adults worldwide may be affected by OSA, making it a major global public health concern .[1] The prevalence of OSA has increased substantially in recent years, largely due to rising obesity rates, sedentary lifestyles, and increasing awareness of sleep disorders. [2]

OSA is associated with several adverse cardiovascular, metabolic, and neurocognitive consequences. Recurrent

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hypoxic episodes and sleep fragmentation activate multiple pathophysiological pathways, including oxidative stress, systemic inflammation, endothelial dysfunction, and autonomic nervous system (ANS) dysregulation. [3] Among these mechanisms, autonomic dysfunction is considered a key contributor to cardiovascular morbidity in patients with OSA. Persistent sympathetic over activity has been implicated in the development of hypertension, arrhythmias, coronary artery disease, heart failure, and cerebrovascular events. [4]

Under physiological conditions, sleep is associated with increased parasympathetic activity and reduced sympathetic tone. However, recurrent apneic events in OSA produce repetitive surges in sympathetic nervous system activity through activation of peripheral chemoreceptors and arousal responses. [5] This chronic sympathoexcitation persists even during wakefulness and contributes to abnormal cardiovascular regulation. [6] Previous studies have demonstrated that the severity of autonomic dysfunction correlates with the severity of OSA, suggesting a dose-dependent relationship between intermittent hypoxia and autonomic imbalance. [7]

Autonomic dysfunction in OSA can be assessed using several cardiovascular autonomic function tests, including HRV, Valsalva ratio, deep breathing tests, orthostatic blood pressure responses, and pulse rate variability.[8] Reduced HRV and impaired parasympathetic activity have been reported in patients with moderate-to-severe OSA and are considered predictors of adverse cardiovascular outcomes.[9] Early identification of autonomic dysfunction may therefore help in cardiovascular risk stratification and guide timely therapeutic interventions.

Although polysomnography remains the gold standard for diagnosing OSA, Level III sleep monitoring devices offer a practical, cost-effective, and accessible alternative for evaluating sleep-disordered breathing in routine clinical settings.[10] However, limited studies from the Indian population have explored the relationship between autonomic dysfunction and OSA severity using Level III sleep monitoring systems. Therefore, the present study was undertaken to evaluate autonomic nervous system dysfunction and its association with the severity of obstructive sleep apnea among patients undergoing Level III sleep monitoring.

MATERIALS AND METHODS

Study Design and Setting- This hospital-based cross-sectional observational study was conducted in the Department of Physiology, Sri Aurobindo Medical College and PG Institute, Indore, Madhya Pradesh, India. The study was carried out among patients referred for evaluation of suspected OSA.

Study Population- A total of 84 adult participants with symptoms suggestive of sleep-disordered breathing, including snoring, excessive daytime sleepiness, witnessed apnea, non-restorative sleep, or unexplained fatigue, were included in the study.

Inclusion Criteria

1. Adults aged 18 years and above.
2. Patients clinically suspected to have obstructive sleep apnea.
3. Patients willing to provide written informed consent.

Exclusion Criteria

1. Patients with previously diagnosed central sleep apnea.
2. Patients with severe cardiovascular instability or acute respiratory illness.
3. Individuals with known neurological disorders affecting autonomic nervous system function.
4. Patients receiving medications significantly influencing autonomic function.
5. Incomplete or technically inadequate sleep study recordings.

Sleep Study Assessment- All participants underwent overnight sleep assessment using the Deck Mount Level III Sleep Apnea Device. The device recorded multiple physiological parameters including airflow, respiratory effort, oxygen saturation, pulse rate, snoring events, and AHI. Sleep recordings were analyzed according to standard sleep study scoring criteria.

OSA severity was classified based on AHI values as follows:

- Mild OSA: AHI 5–14.9 events/hour
- Moderate OSA: AHI 15–29.9 events/hour
- Severe OSA: AHI ≥30 events/hour

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Clinical and Anthropometric Assessment- Detailed demographic and clinical data including age, sex, body mass index (BMI), history of snoring, daytime sleepiness, and associated comorbidities were recorded. Height and weight measurements were obtained using standardized methods, and BMI was calculated as weight in kilograms divided by height in meters squared (kg/m²).

Assessment of Autonomic Nervous System Function- Autonomic nervous system function was assessed using cardiovascular autonomic parameters obtained from overnight sleep recordings and standard autonomic function tests. The evaluated parameters included average, minimum, and maximum pulse rates, HRV, Valsalva ratio, deep breathing expiratory-to-inspiratory (E:I) ratio, and orthostatic blood pressure response. These measures were used to assess both sympathetic and parasympathetic components of autonomic regulation. Heart rate variability, Valsalva ratio, and E: I ratio primarily reflected parasympathetic activity, whereas orthostatic blood pressure response served as an indicator of sympathetic cardiovascular function. Together, these parameters provided a comprehensive assessment of autonomic nervous system dysfunction and its relationship with obstructive sleep apnea severity [11,12].

Statistical Analysis- Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean±standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons among OSA severity groups were performed using one-way analysis of variance (ANOVA) for continuous variables and chi-square test for categorical variables. Pearson correlation analysis was used to assess the relationship between autonomic function parameters and AHI. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 84 patients with OSA were included in the study. Body mass index (BMI), AHI, oxygen desaturation index (ODI), and oxygen saturation parameters differed significantly across OSA severity groups. Patients with severe OSA had higher BMI, higher AHI and ODI values, and lower average and minimum oxygen saturation levels compared with those with mild and moderate OSA, indicating worsening nocturnal hypoxemia with increasing disease severity (Table 1).

Table 1: Baseline Characteristics of Study Participants According to OSA Severity

Variable	Mild OSA (n=13)	Moderate OSA (n=32)	Severe OSA (n=39)	p-value
Age (years)	53.7 ± 12.4	52.4 ± 11.1	45.4 ± 10.8	0.041
BMI (kg/m²)	29.1 ± 4.8	31.2 ± 5.1	32.0 ± 5.9	0.032
AHI (events/hr)	10.2 ± 2.8	22.1 ± 4.5	46.8 ± 12.7	<0.001
ODI	29.7 ± 8.4	36.7 ± 10.1	39.0 ± 11.6	0.018
Average SpO_2 (%)	93.8 ± 2.1	92.5 ± 2.8	90.9 ± 3.4	0.006
Lowest SpO_2 (%)	82.6 ± 5.2	77.8 ± 7.1	71.2 ± 8.6	<0.001

AHI= Apnea-Hypopnea Index; ODI= Oxygen Desaturation Index; SpO_2= oxygen saturation.

Autonomic function assessment revealed progressive autonomic impairment with increasing OSA severity. Heart rate variability (HRV), Valsalva ratio, and deep breathing expiratory-to-inspiratory (E:I) ratio decreased significantly from mild to severe OSA, reflecting reduced parasympathetic activity. In contrast, orthostatic blood pressure fall increased significantly among patients with severe OSA, suggesting greater autonomic dysregulation (Table 2).

Correlation analysis demonstrated significant associations between autonomic dysfunction and OSA severity. AHI showed significant negative correlations with HRV ($r = -0.52$, $p < 0.001$), Valsalva ratio ($r = -0.41$, $p = 0.003$), and E:I ratio ($r = -0.38$, $p = 0.007$).

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A significant positive correlation was observed between AHI and orthostatic blood pressure fall (r = 0.46, p = 0.001). However, average, minimum, and maximum pulse rates were not significantly correlated with OSA severity (Table 3). Overall, increasing OSA severity was associated with progressive autonomic nervous system dysfunction, characterized by impaired parasympathetic function and altered sympathetic cardiovascular regulation.

Table 2: Comparison of Autonomic Function Parameters Across OSA Severity Groups

Autonomic Parameter	Mild OSA	Moderate OSA	Severe OSA	p-value
Average Pulse Rate (bpm)	92.8 ± 8.4	94.1 ± 9.2	91.3 ± 10.5	0.412
Minimum Pulse Rate (bpm)	61.2 ± 7.1	59.8 ± 8.2	57.4 ± 8.5	0.228
Maximum Pulse Rate (bpm)	110.4 ± 12.5	110.2 ± 13.1	110.9 ± 14.3	0.964
Resting Heart Rate Variability (ms)*	42.6 ± 8.1	36.4 ± 7.5	29.8 ± 6.9	<0.001
Valsalva Ratio*	1.42 ± 0.18	1.31 ± 0.16	1.18 ± 0.14	0.002
Deep Breathing E:I Ratio*	1.28 ± 0.11	1.21 ± 0.10	1.13 ± 0.09	0.004
Sympathetic BP Fall on Standing (mmHg)*	6.2 ± 3.1	9.4 ± 4.2	13.1 ± 5.8	<0.001

Table 3: Correlation between Autonomic Parameters and OSA Severity (AHI)

Variable	Correlation Coefficient (r)	p-value
Average Pulse Rate	-0.19	0.079
Minimum Pulse Rate	-0.04	0.731
Maximum Pulse Rate	0.02	0.845
Heart Rate Variability*	-0.52	<0.001
Valsalva Ratio*	-0.41	0.003
E: I Ratio*	-0.38	0.007
Orthostatic BP Fall*	0.46	0.001

DISCUSSION

The present cross-sectional study evaluated the association between autonomic nervous system dysfunction and the severity of OSA using Level III sleep monitoring. The findings demonstrated that autonomic dysfunction progressively worsened with increasing OSA severity. Significant reductions in HRV, Valsalva ratio, and deep breathing expiratory-to-inspiratory (E:I) ratio were observed among patients with severe OSA, indicating impaired parasympathetic activity and enhanced sympathetic predominance. Furthermore, orthostatic blood pressure fall showed a positive correlation with AHI, suggesting increasing sympathetic dysregulation with worsening disease severity.

OSA is characterized by recurrent episodes of intermittent hypoxia, hypercapnia, and sleep fragmentation, all of which contribute to chronic activation of the sympathetic nervous system. Repeated arousals during sleep stimulate peripheral chemoreceptors and increase sympathetic outflow, resulting in persistent autonomic imbalance even during wakefulness. [4] Sympathetic overactivity has been recognized as one of the major mechanisms linking OSA with hypertension, arrhythmias, coronary artery disease, and cerebrovascular complications. [5]

In the present study, severe OSA patients exhibited significantly lower HRV compared to mild OSA patients. Reduced HRV reflects impaired autonomic adaptability and diminished parasympathetic modulation of cardiac

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activity. Similar findings were reported significantly reduced nocturnal HRV among patients with moderate-to-severe OSA. [9] Alomri *et al*. also reported a strong association between sympathetic nervous system activation and increasing OSA severity, supporting the findings of the present study. [7]

The observed reduction in Valsalva ratio and deep breathing E:I ratio among severe OSA patients further suggests impaired parasympathetic function. These autonomic function tests are widely used to assess cardiovagal integrity and parasympathetic responsiveness. [8] Chronic intermittent hypoxia in OSA has been shown to impair baroreceptor sensitivity and vagal modulation, thereby contributing to cardiovascular autonomic dysfunction. [6]

Orthostatic blood pressure response was significantly altered among patients with severe OSA in the present study. Increased orthostatic blood pressure fall may indicate impaired sympathetic vasoconstrictor response and altered vascular autonomic regulation. Previous studies have similarly demonstrated abnormal autonomic cardiovascular responses in OSA patients, especially among individuals with severe nocturnal oxygen desaturation. [7,13]

The study also demonstrated worsening oxygen desaturation indices with increasing disease severity. Intermittent hypoxia is known to induce oxidative stress, endothelial dysfunction, systemic inflammation, and activation of neurohumoral pathways, all of which contribute to autonomic dysregulation. [8,14] Persistent sympathetic activation secondary to hypoxia may explain the increased cardiovascular risk observed in untreated OSA patients.

The findings of the present study have important clinical implications. Early identification of autonomic dysfunction in OSA patients may facilitate cardiovascular risk stratification and prompt therapeutic intervention. Continuous positive airway pressure (CPAP) therapy has been shown to improve autonomic function, reduce sympathetic activity, and restore parasympathetic balance in patients with moderate-to-severe OSA. [9] Therefore, assessment of autonomic dysfunction may serve as an additional marker for disease burden and treatment response.

STRENGTHS

The present study provides important insight into the relationship between autonomic nervous system dysfunction and obstructive sleep apnea severity in an Indian clinical population. One of the major strengths of the study is the use of objective overnight sleep assessment using the Deck Mount Level III Sleep Apnea Device, which enabled standardized evaluation of apnea–hypopnea index, oxygen desaturation parameters, and pulse-derived autonomic indices. The study also incorporated multiple autonomic function parameters, including heart rate variability, Valsalva ratio, deep breathing E:I ratio, and orthostatic blood pressure response, allowing comprehensive assessment of both sympathetic and parasympathetic autonomic function. In addition, the inclusion of patients across different OSA severity categories facilitated comparative evaluation of autonomic dysfunction progression with worsening disease severity.

LIMITATIONS

The study has several limitations. First, it was a single-center cross-sectional study with a relatively small sample size, which may limit the generalizability of the findings. Second, causal relationships between autonomic dysfunction and OSA severity could not be established due to the observational study design. Third, Level III sleep monitoring, although practical and cost-effective, does not provide detailed sleep architecture and electroencephalographic data comparable to full polysomnography. Furthermore, advanced autonomic assessment techniques such as muscle sympathetic nerve activity, baroreflex sensitivity analysis, and plasma catecholamine measurements were not performed. Potential confounding factors including obesity, hypertension, diabetes mellitus, and medication use may also have influenced autonomic function parameters. Future multicentric studies with larger sample sizes and comprehensive autonomic evaluation are recommended to further validate these findings.

CONCLUSIONS

The present study demonstrated a significant association between autonomic nervous system dysfunction and the severity of obstructive sleep apnea. Patients with severe OSA exhibited greater impairment in autonomic function parameters, including reduced heart rate variability,

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decreased parasympathetic responsiveness, and altered sympathetic cardiovascular regulation. Increasing apnea–hypopnea index was associated with progressive autonomic imbalance, highlighting the important role of autonomic dysfunction in the pathophysiology of OSA and its cardiovascular complications. Assessment of autonomic function alongside routine sleep evaluation may provide additional clinical value for cardiovascular risk stratification and comprehensive management of patients with obstructive sleep apnea.

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