

## Original Research Article

# PREVALENCE OF OBSTRUCTIVE SLEEP APNEA IN PATIENTS WITH RESISTANT HYPERTENSION: A CROSS-SECTIONAL STUDY

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## ABSTRACT

**Background:** Resistant hypertension (RH) is a challenging clinical condition often complicated by obstructive sleep apnea (OSA), a treatable cause of secondary hypertension. Understanding the prevalence and impact of OSA in RH patients is essential for optimal management. The aim is to determine the prevalence of obstructive sleep apnea in patients diagnosed with resistant hypertension.

**Materials and Methods:** This cross-sectional study included 200 adult patients with resistant hypertension attending a tertiary care hospital. All patients underwent overnight polysomnography to diagnose OSA, defined by an apnea-hypopnea index (AHI)  $\geq 5$  events/hour. Demographic, clinical, and blood pressure data were collected and analyzed. Statistical significance was set at  $p < 0.05$ .

**Results:** OSA was diagnosed in 65% (130/200) of patients. Patients with OSA were significantly older ( $54.1 \pm 10.2$  vs.  $50.5 \pm 11.0$  years;  $p = 0.008$ ), predominantly male (70.8% vs. 51.4%;  $p = 0.004$ ), had higher BMI ( $30.2 \pm 4.1$  vs.  $28.2 \pm 4.0$  kg/m<sup>2</sup>;  $p < 0.001$ ), and longer duration of hypertension ( $8.5 \pm 4.6$  vs.  $6.6 \pm 4.8$  years;  $p = 0.006$ ). Severity of OSA correlated positively with systolic and diastolic blood pressure, number of antihypertensive medications, and nocturnal blood pressure non-dipping ( $p < 0.001$ ).

**Conclusion:** OSA is highly prevalent among patients with resistant hypertension and is associated with worse blood pressure control. Routine screening for OSA should be considered in this population to facilitate timely diagnosis and targeted treatment.

**Keywords:** Resistant Hypertension; Obstructive Sleep Apnea; Polysomnography.

## INTRODUCTION

Resistant hypertension (RH) is a significant clinical problem characterized by blood pressure that remains above target levels despite the concurrent use of three antihypertensive agents of different classes, typically including a diuretic, at optimal doses.<sup>[1]</sup> It accounts for approximately 10-20% of hypertensive patients and is associated with an increased risk of cardiovascular morbidity and mortality due to persistent elevation of blood pressure.<sup>[2]</sup> Understanding the underlying causes and associated comorbidities of resistant hypertension is crucial for

effective management and reduction of adverse outcomes.

One such commonly associated and underdiagnosed condition is obstructive sleep apnea (OSA). OSA is a sleep-related breathing disorder characterized by repeated episodes of partial or complete upper airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation, and sympathetic nervous system activation.<sup>[3]</sup> The prevalence of OSA in the general population varies widely, estimated between 9-38% in adults, with higher rates reported in patients with cardiovascular diseases and metabolic syndrome.<sup>[4]</sup>

The pathophysiological link between OSA and hypertension has been extensively studied. OSA contributes to elevated blood pressure through several mechanisms including intermittent hypoxia-induced oxidative stress, activation of the renin-angiotensin-aldosterone system (RAAS), endothelial dysfunction, and increased sympathetic activity.<sup>[5]</sup> These mechanisms not only induce hypertension but also complicate its treatment, contributing to resistant hypertension in affected individuals.

Despite the recognized association, OSA remains frequently undiagnosed in patients with resistant hypertension. This underdiagnosis is often attributed to the non-specificity of symptoms and the lack of routine screening in hypertensive populations.<sup>[6]</sup> Identifying OSA in resistant hypertension patients is critical because treatment with continuous positive airway pressure (CPAP) and other modalities can significantly improve blood pressure control and reduce cardiovascular risk.<sup>[7]</sup>

**Aim:** To determine the prevalence of obstructive sleep apnea in patients diagnosed with resistant hypertension.

#### **Objectives**

1. To assess the prevalence of obstructive sleep apnea among patients with resistant hypertension attending the tertiary care hospital.
2. To evaluate the severity of obstructive sleep apnea using polysomnography in resistant hypertension patients.
3. To analyze the correlation between obstructive sleep apnea severity and blood pressure control parameters in resistant hypertension.

## **MATERIALS AND METHODS**

**Source of Data:** The study data were collected from patients diagnosed with resistant hypertension attending the outpatient and inpatient departments of the Department of Respiratory Medicine at tertiary care hospital, during the study period.

**Study Design:** This was a hospital-based cross-sectional observational study designed to assess the prevalence of obstructive sleep apnea among patients with resistant hypertension.

**Study Location:** The study was conducted at Department of Respiratory Medicine at tertiary care hospital.

**Study Duration:** The study was conducted over a period of 12 months.

**Sample Size:** A total of 200 patients diagnosed with resistant hypertension were enrolled in the study based on sample size calculations considering prevalence estimates from previous studies, with a confidence level of 95% and allowable error margin of 7%.

#### **Inclusion Criteria**

- Adult patients aged 18 years and above.
- Diagnosed with resistant hypertension defined as blood pressure above target despite treatment with three or more antihypertensive medications

including a diuretic, or controlled blood pressure requiring four or more medications.

- Patients willing to provide informed consent and undergo sleep study evaluations.

#### **Exclusion Criteria**

- Patients with secondary causes of hypertension other than OSA - renal artery stenosis, primary aldosteronism, pheochromocytoma.
- Patients with congestive heart failure (NYHA Class III or IV).
- Patients with severe chronic obstructive pulmonary disease or other pulmonary pathologies.
- Patients unable to undergo polysomnography due to any physical or cognitive limitations.
- Pregnant women.

**Procedure and Methodology:** Eligible patients were identified from the outpatient and inpatient settings after reviewing clinical history, medication records, and blood pressure measurements. Detailed history taking included assessment of sleep-related symptoms such as snoring, witnessed apneas, daytime sleepiness, and use of the Epworth Sleepiness Scale (ESS).

All patients underwent overnight in-lab polysomnography (PSG), which is the gold standard diagnostic modality for OSA. The PSG parameters recorded included electroencephalography, electrooculography, electromyography, airflow by nasal pressure transducer, respiratory effort by thoracoabdominal belts, oxygen saturation by pulse oximetry, and body position monitoring.

The apnea-hypopnea index (AHI) was calculated as the number of apneas and hypopneas per hour of sleep. OSA severity was classified as mild (AHI 5-14), moderate (AHI 15-29), or severe (AHI  $\geq 30$ ) based on the American Academy of Sleep Medicine criteria.

Blood pressure measurements were taken using a calibrated sphygmomanometer following standard protocols. Medication adherence was assessed through patient interview and review of prescriptions.

**Sample Processing:** Polysomnography data were analyzed by experienced sleep technologists and interpreted by qualified sleep physicians blinded to the clinical data. The collected data were coded and entered into a pre-designed data collection sheet for further statistical analysis.

**Statistical Methods:** Data analysis was performed using Statistical Package for Social Sciences (SPSS) version 25.0. Continuous variables were presented as mean  $\pm$  standard deviation (SD), and categorical variables as frequencies and percentages. The prevalence of OSA was calculated with 95% confidence intervals.

Comparisons between groups (e.g., OSA vs. no OSA) were performed using Chi-square test for categorical variables and Student's t-test or Mann-Whitney U test for continuous variables, depending on data distribution. Correlation between AHI and blood pressure levels was evaluated using Pearson's or Spearman's correlation coefficient.

A p-value < 0.05 was considered statistically significant.

**Data Collection:** Data were collected using a structured proforma including demographic details, clinical history, medication details, blood pressure readings, sleep study findings, and relevant laboratory investigations. Patients' confidentiality was maintained throughout, and informed consent was obtained before inclusion in the study.

## RESULTS

[Table 1] presents the baseline demographic and clinical characteristics of 200 patients with resistant hypertension, categorized by the presence or absence of obstructive sleep apnea (OSA). The mean age of the entire cohort was  $52.8 \pm 10.6$  years, with those having OSA significantly older ( $54.1 \pm 10.2$  years) compared to those without OSA ( $50.5 \pm 11.0$  years),

showing a statistically significant difference ( $t = 2.69$ ,  $p = 0.008$ ). Males predominated the study population (64.0%), and the prevalence of OSA was significantly higher among males (70.8%) than females (29.2%) with a chi-square value of 8.34 ( $p = 0.004$ ). The mean body mass index (BMI) was  $29.5 \pm 4.2$  kg/m<sup>2</sup>, with the OSA group having significantly higher BMI ( $30.2 \pm 4.1$ ) than the non-OSA group ( $28.2 \pm 4.0$ ), indicating obesity as a significant risk factor ( $t = 3.85$ ,  $p < 0.001$ ). Duration of hypertension was longer in the OSA group ( $8.5 \pm 4.6$  years) than in the non-OSA group ( $6.6 \pm 4.8$  years), also statistically significant ( $t = 2.78$ ,  $p = 0.006$ ). Smoking was more common among those with OSA (28.5%) compared to those without (15.7%) ( $\chi^2 = 4.36$ ,  $p = 0.037$ ). These findings suggest that older age, male gender, higher BMI, longer duration of hypertension, and smoking are associated with the presence of OSA in resistant hypertension patients.

**Table 1: Baseline Demographic and Clinical Characteristics of Study Population (N=200)**

Parameter	Category	Total (n=200)	OSA Present (n=130)	OSA Absent (n=70)	Test Statistic (t/ $\chi^2$ )	95% CI for Difference / Mean	P-value
Age (years)	—	$52.8 \pm 10.6$	$54.1 \pm 10.2$	$50.5 \pm 11.0$	$t = 2.69$	1.15 to 5.48	0.008*
Gender	Male	128 (64.0%)	92 (70.8%)	36 (51.4%)	$\chi^2 = 8.34$	—	0.004*
	Female	72 (36.0%)	38 (29.2%)	34 (48.6%)			
BMI (kg/m <sup>2</sup> )	—	$29.5 \pm 4.2$	$30.2 \pm 4.1$	$28.2 \pm 4.0$	$t = 3.85$	0.7 to 3.2	<0.001*
Duration of HTN (yrs)	—	$7.8 \pm 4.7$	$8.5 \pm 4.6$	$6.6 \pm 4.8$	$t = 2.78$	0.48 to 3.18	0.006*
Smoking status	Current smoker	48 (24.0%)	37 (28.5%)	11 (15.7%)	$\chi^2 = 4.36$	—	0.037*
	Non-smoker	152 (76.0%)	93 (71.5%)	59 (84.3%)			

\*Significant at  $p < 0.05$

**Table 2: Prevalence of Obstructive Sleep Apnea in Resistant Hypertension Patients (N=200)**

Parameter	Category	Total (n=200)	OSA Present (n=130)	OSA Absent (n=70)	Test Statistic ( $\chi^2$ )	95% CI for Prevalence in OSA Group	P-value
OSA Diagnosis by PSG	Positive (AHI $\geq 5$ )	130 (65.0%)	130 (100%)	0 (0%)	—	58.0% to 71.4%	—
Severity of OSA	Mild (AHI 5-14)	46 (23.0%)	46 (35.4%)	0 (0%)	—	17.3% to 28.9%	—
	Moderate (AHI 15-29)	53 (26.5%)	53 (40.8%)	0 (0%)	—	20.5% to 32.3%	—
	Severe (AHI $\geq 30$ )	31 (15.5%)	31 (23.8%)	0 (0%)	—	11.1% to 19.6%	—
Snoring	Present	145 (72.5%)	115 (88.5%)	30 (42.9%)	$\chi^2 = 49.7$	—	<0.001*
Excessive daytime sleepiness	ESS score $\geq 10$	118 (59.0%)	94 (72.3%)	24 (34.3%)	$\chi^2 = 27.2$	—	<0.001*

\*Significant at  $p < 0.05$

[Table 2] details the prevalence and clinical features of obstructive sleep apnea among the 200 resistant hypertension patients. OSA was diagnosed in 130 patients, corresponding to a prevalence rate of 65.0% (95% CI: 58.0% to 71.4%) based on an apnea-hypopnea index (AHI)  $\geq 5$ . Among those diagnosed with OSA, the severity was distributed as mild in 35.4%, moderate in 40.8%, and severe in 23.8% of patients. Clinically, snoring was present in 88.5% of

OSA patients compared to 42.9% without OSA, which was highly significant ( $\chi^2 = 49.7$ ,  $p < 0.001$ ). Excessive daytime sleepiness, measured by an Epworth Sleepiness Scale score  $\geq 10$ , was significantly more common in the OSA group (72.3%) compared to the non-OSA group (34.3%) ( $\chi^2 = 27.2$ ,  $p < 0.001$ ). These results confirm a high prevalence of OSA among resistant hypertension patients with distinct clinical symptoms.

**Table 3: Severity of Obstructive Sleep Apnea Using Polysomnography in Resistant Hypertension Patients (N=130 with OSA)**

Parameter	Category	Total (n=130)	Controlled HTN (n=54)	Uncontrolled HTN (n=76)	Test Statistic (t/ $\chi^2$ )	95% CI for Difference	P-value
AHI (events/hour)	—	27.3 ± 11.8	22.7 ± 8.3	30.5 ± 12.4	t = 4.31	4.2 to 10.5	<0.001*
Severe OSA (AHI ≥ 30)	Yes	31 (23.8%)	7 (13.0%)	24 (31.6%)	$\chi^2 = 6.55$	—	0.011*
Moderate OSA (AHI 15-29)	Yes	53 (40.8%)	20 (37.0%)	33 (43.4%)	$\chi^2 = 0.50$	—	0.480
Mild OSA (AHI 5-14)	Yes	46 (35.4%)	27 (50.0%)	19 (25.0%)	$\chi^2 = 7.39$	—	0.007*
Mean Oxygen Desaturation (%)	—	82.4 ± 6.3	85.7 ± 5.1	79.9 ± 6.8	t = 5.14	3.5 to 8.1	<0.001*

\*Significant at p<0.05

[Table 3] compares the severity of OSA as determined by polysomnography between patients with controlled and uncontrolled resistant hypertension. The mean apnea-hypopnea index (AHI) was significantly higher in patients with uncontrolled hypertension ( $30.5 \pm 12.4$  events/hour) compared to controlled hypertensives ( $22.7 \pm 8.3$  events/hour) (t = 4.31, p < 0.001). Severe OSA (AHI ≥ 30) was more frequent in the uncontrolled group (31.6%) than in the controlled group (13.0%) ( $\chi^2 = 6.55$ , p = 0.011). Conversely, mild OSA was more

common among controlled hypertensives (50.0%) compared to uncontrolled patients (25.0%) ( $\chi^2 = 7.39$ , p = 0.007). No significant difference was found in the prevalence of moderate OSA between the groups (p = 0.480). Mean oxygen desaturation during sleep was significantly lower in uncontrolled hypertensives ( $79.9 \pm 6.8\%$ ) than controlled patients ( $85.7 \pm 5.1\%$ ) (t = 5.14, p < 0.001). This indicates that greater severity of OSA and hypoxemia are associated with poor blood pressure control in resistant hypertension.

**Table 4: Correlation of OSA Severity (AHI) with Blood Pressure Control Parameters in Resistant Hypertension Patients (N=130 with OSA)**

Parameter	Mean ± SD	Correlation with AHI	Test Statistic (r)	95% CI for r	P-value
Systolic BP (mmHg)	152.8 ± 18.5	Positive correlation	0.43	0.28 to 0.56	<0.001*
Diastolic BP (mmHg)	94.2 ± 12.3	Positive correlation	0.38	0.22 to 0.52	<0.001*
Number of antihypertensive drugs	3.8 ± 0.6	Positive correlation	0.31	0.14 to 0.46	0.001*
Nocturnal BP non-dipping status	78 (60.0%)	Strong association	$\chi^2 = 18.2$	—	<0.001*

\*Significant at p<0.05

[Table 4] shows the correlation between OSA severity, measured by the apnea-hypopnea index (AHI), and various blood pressure control parameters among the 130 patients with OSA. There was a moderate positive correlation between AHI and systolic blood pressure (r = 0.43, 95% CI: 0.28 to 0.56, p < 0.001) and diastolic blood pressure (r = 0.38, 95% CI: 0.22 to 0.52, p < 0.001), indicating that higher OSA severity is associated with elevated blood pressure levels. The number of antihypertensive medications also showed a positive correlation with AHI (r = 0.31, p = 0.001), suggesting that patients with more severe OSA required more intensive pharmacological therapy. Additionally, nocturnal blood pressure non-dipping status was present in 60% of patients and had a strong association with OSA severity ( $\chi^2 = 18.2$ , p < 0.001). These findings emphasize that OSA severity is significantly linked to poor blood pressure control and may contribute to the complexity of managing resistant hypertension.

## DISCUSSION

The present study evaluated the prevalence and severity of obstructive sleep apnea (OSA) in patients

with resistant hypertension (RH) and analyzed its clinical and hemodynamic correlates.

Table 1 showed that among the 200 RH patients, 65% were diagnosed with OSA by polysomnography. The OSA group was significantly older, predominantly male, with higher BMI and longer hypertension duration compared to non-OSA patients. Smoking prevalence was also higher among those with OSA. These findings align well with previous literature. Labarca G et al,<sup>[8]</sup> (2019) reported a higher prevalence of OSA in older, obese males with hypertension. Similarly, Chang CP et al,<sup>[9]</sup> (2016) highlighted that obesity and male gender are significant risk factors for OSA in RH. Our findings of increased smoking in OSA patients concur with Oscullo G et al. (2019),<sup>[10]</sup> who noted smoking as a contributor to airway inflammation, worsening OSA severity. The age distribution and BMI levels were comparable to data from Parati G et al. (2014),<sup>[5]</sup> confirming the strong association between OSA and metabolic risk factors in hypertensive cohorts.

[Table 2] demonstrated a high OSA prevalence (65%) in the RH cohort, consistent with meta-analyses by Iftikhar IH et al. (2014),<sup>[11]</sup> who reported prevalence rates of 60-70% in similar populations. The distribution of OSA severity showed a



predominance of moderate cases (40.8%), followed by mild and severe forms, which mirrors findings by Varounis C et al. (2014),<sup>[12]</sup> that moderate to severe OSA constitutes the majority of cases in RH. Clinical features such as snoring and excessive daytime sleepiness (ESS score  $\geq 10$ ) were significantly more common in the OSA group, reinforcing the diagnostic utility of these symptoms in high-risk populations, as demonstrated by Marcus JA et al. (2014).<sup>[7]</sup>

[Table 3] compared OSA severity in patients with controlled versus uncontrolled resistant hypertension. Notably, uncontrolled hypertensive patients had significantly higher AHI and greater oxygen desaturation, with a larger proportion experiencing severe OSA. Mild OSA was more prevalent among controlled hypertensives. These results corroborate with findings from Oscullo G et al. (2019),<sup>[13]</sup> who established that severe OSA independently contributes to poor blood pressure control due to intermittent hypoxia and sympathetic activation. The significant difference in oxygen desaturation aligns with insights from Liu L et al. (2016),<sup>[14]</sup> emphasizing hypoxia as a key pathophysiological factor in resistant hypertension. Table 4 revealed positive correlations between OSA severity (AHI) and systolic and diastolic blood pressure levels, number of antihypertensive drugs, and nocturnal blood pressure non-dipping status. This supports prior studies by Hou H et al. (2018),<sup>[15]</sup> demonstrating that OSA severity predicts higher blood pressure and medication resistance. The strong association with non-dipping nocturnal blood pressure is also well documented, underscoring OSA's impact on circadian blood pressure regulation, as shown in studies by Yang L et al. (2016).<sup>[16]</sup>

## CONCLUSION

This cross-sectional study demonstrated a high prevalence (65%) of obstructive sleep apnea (OSA) among patients with resistant hypertension. Older age, male gender, higher body mass index, longer duration of hypertension, and smoking were significantly associated with OSA presence. Furthermore, OSA severity correlated positively with poorer blood pressure control, increased number of antihypertensive medications, and nocturnal non-dipping status. These findings underscore the critical role of routine screening for OSA in patients with resistant hypertension to improve diagnosis and guide targeted management strategies. Early identification and treatment of OSA may contribute to better blood pressure control and reduce cardiovascular risk in this high-risk population.

### Limitations of Study

Several limitations must be considered when interpreting the results of this study. First, the cross-sectional design precludes establishing causal relationships between OSA and resistant hypertension. Second, the study was conducted at a

single tertiary care center, which may limit the generalizability of findings to broader populations. Third, polysomnography, though the gold standard, was performed in-lab only and may not fully capture habitual sleep patterns due to the “first night effect.” Fourth, factors such as medication adherence, lifestyle variables, and undiagnosed secondary hypertension causes were not comprehensively evaluated, potentially confounding the results. Lastly, the study did not assess the impact of OSA treatment on blood pressure control, which would be valuable for clinical application.

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