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ORIGINAL ARTICLE

Association of Sleep Apnea with Resistant Hypertension and Cardiovascular Morbidity in Obese Adults. A Comparative Clinical Study

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ABSTRACT

Background: Obstructive sleep apnea (OSA) and resistant hypertension are interrelated conditions that frequently coexist in obese individuals, markedly increasing the risk of cardiovascular complications. Although their association is well recognized, the extent and severity of cardiovascular morbidity attributable to their coexistence remain inadequately explored in clinical practice.

Objective: To evaluate the association between OSA and resistant hypertension in obese adults and to assess the impact of OSA severity on cardiovascular outcomes.

Methodology: This comparative clinical study included 200 obese hypertensive patients aged 30–65 years, comprising both males and females. Participants were categorized into two groups: controlled hypertension (n = 104) and resistant hypertension (n = 96). All subjects underwent overnight polysomnography to determine the severity of OSA. Comprehensive biochemical profiling was performed, including metabolic and inflammatory biomarkers such as hs-CRP, IL-6, TNF-α, and serum aldosterone. Cardiovascular evaluation included echocardiography and documentation of clinical history of myocardial infarction, stroke, and heart failure.

Results: OSA was significantly more prevalent in the resistant hypertension group (92.7%) compared to the controlled hypertension group (56.7%) ($p < 0.001$). Severe OSA was disproportionately higher among patients with resistant hypertension. Inflammatory markers and serum aldosterone levels were significantly elevated in the resistant group ($p < 0.001$). Cardiovascular complications, including left ventricular hypertrophy, diastolic dysfunction, and myocardial infarction, were also markedly more common in patients with resistant hypertension and concurrent OSA.

Conclusion: This study establishes a strong association between OSA and resistant hypertension, highlighting the substantial contribution of OSA to cardiovascular morbidity in obese individuals. Early identification and management of sleep-disordered breathing in hypertensive patients may improve therapeutic outcomes and mitigate long-term cardiovascular risk.

Keywords: Resistant hypertension, Obesity, Cardiovascular morbidity, Inflammatory biomarkers, Left ventricular hypertrophy, Diastolic dysfunction, Polysomnography.

INTRODUCTION

Obstructive sleep apnea (OSA), characterized by repetitive episodes of upper airway obstruction during sleep, has emerged as a critical, yet underdiagnosed, contributor to cardiovascular morbidity, particularly in the context of obesity and resistant hypertension [1]. The pathophysiological interplay between intermittent hypoxia, sympathetic nervous system overactivation, systemic inflammation, endothelial dysfunction, and metabolic dysregulation forms a mechanistic nexus that links OSA with adverse cardiovascular outcomes [2]. Obesity, a well-established risk factor for OSA, exacerbates upper airway collapsibility through anatomical and functional alterations in pharyngeal patency and respiratory control, creating a vicious cycle that perpetuates cardiorespiratory burden [3].

Resistant hypertension defined as blood pressure that remains above target despite concurrent use of three or more antihypertensive agents, including a diuretic—has shown increasing prevalence in patients with moderate to severe OSA [4]. This phenotype of hypertension is not only therapeutically challenging but also strongly associated with increased risk of left ventricular hypertrophy, myocardial infarction, stroke, and overall cardiovascular mortality. Several epidemiological and interventional studies have demonstrated that OSA may be both a causative and perpetuating factor in the pathogenesis of resistant hypertension, driven by nocturnal surges in blood pressure, non-dipping blood pressure patterns, and elevated plasma aldosterone concentrations [5].

Despite mounting evidence of the triadic relationship among OSA, obesity, and resistant hypertension, clinical recognition and systematic evaluation remain limited, particularly in high-risk populations[6]. Moreover, the additive impact of OSA on cardiovascular morbidity in obese adults with resistant hypertension is not fully delineated in existing literature. Therefore, this study was designed to elucidate the association between OSA and resistant hypertension in obese individuals and to evaluate the burden of cardiovascular comorbidities in this clinically vulnerable population [7]. By integrating clinical, polysomnographic, and hemodynamic assessments, this investigation aims to contribute to the growing body of literature advocating for early identification and management of sleep-disordered breathing in hypertensive obese patients to mitigate long-term cardiovascular risk [8].

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MATERIALS AND METHODS

This comparative clinical study was conducted over an eight-month period from January 2023 to August 2023 at two tertiary care cardiology centers in Punjab, Pakistan: the Department of Cardiology, Chahudhary Pervaiz Elahi Institute of Cardiology, Wazirabad, and the Department of Cardiology, Nawaz Shareef Medical College, Aziz Bhatti Shaheed Teaching Hospital, Gujrat. The primary objective was to evaluate the association between obstructive sleep apnea (OSA) and resistant hypertension and to assess their combined impact on cardiovascular morbidity in obese adults.

A total of 200 obese hypertensive patients, comprising both males and females aged between 30 and 65 years, were enrolled through consecutive sampling from the outpatient departments of both institutions. Inclusion criteria included patients with a body mass index (BMI) ≥30 kg/m², a confirmed diagnosis of hypertension for at least one year, and the absence of secondary causes of hypertension. Resistant hypertension was defined as blood pressure ≥140/90 mmHg despite adherence to a regimen of three or more antihypertensive agents, including a diuretic.

Patients with secondary hypertension (e.g., renovascular, endocrine), known structural heart disease, psychiatric disorders, pregnancy, or non-compliance to prescribed medications were excluded.

Detailed clinical histories were recorded, including demographic variables (age, sex, weight, height, BMI, neck circumference, waist-hip ratio), lifestyle factors (smoking, alcohol use, physical activity), and comorbidities (diabetes mellitus, dyslipidemia, ischemic heart disease, stroke, heart failure). Blood pressure measurements were obtained in a controlled environment using standardized protocols with at least two readings taken on separate visits.

All participants underwent overnight in-laboratory polysomnography using a standardized diagnostic setup to assess sleep architecture and respiratory disturbances. Parameters recorded included the apnea–hypopnea index (AHI), oxygen desaturation index (ODI), total sleep time, and sleep efficiency. The diagnosis and severity of OSA were categorized based on the American Academy of Sleep Medicine (AASM) criteria: mild (AHI 5–14.9), moderate (AHI 15–29.9), and severe (AHI ≥30).

Venous blood samples were collected after overnight fasting to evaluate a panel of metabolic and inflammatory biomarkers. These included fasting blood glucose, glycated hemoglobin (HbA1c), total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides, serum creatinine, high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and serum aldosterone levels. Renal function was assessed using estimated glomerular filtration rate (eGFR) calculated by the CKD-EPI equation.

All patients also underwent transthoracic echocardiography to evaluate cardiac structure and function, specifically assessing left ventricular hypertrophy, ejection fraction, and diastolic function using standardized echocardiographic criteria.

Data were entered into a structured proforma and analyzed using SPSS version 25.0. Continuous variables were presented as mean ± standard deviation (SD) and compared using independent samples t-tests or ANOVA where appropriate. Categorical data were presented as frequencies and percentages and analyzed using chi-square or Fisher's exact tests. Multivariate logistic regression models were employed to determine independent predictors of resistant hypertension and cardiovascular complications, adjusting for age, gender, BMI, and relevant biochemical variables. A p-value of <0.05 was considered statistically significant for all analyses.

RESULTS

A total of 200 obese hypertensive patients were enrolled in this study, including 112 males (56%) and 88 females (44%). The mean age of the participants was 51.3 ± 8.7 years, with no significant difference in age distribution between males and females. Among the enrolled subjects, 96 patients (48%) were diagnosed with resistant hypertension, while the remaining 104 (52%) had controlled hypertension on conventional therapy.

In this study involving 200 obese hypertensive patients, a comparative analysis between those with controlled hypertension (n = 104) and those with resistant hypertension (n = 96) revealed several notable demographic and anthropometric differences. The mean age was slightly higher in the resistant hypertension group (52.0 ± 9.1 years) compared to the controlled group (50.7 ± 8.3 years), although this difference was not statistically significant (p = 0.18). Gender distribution was comparable across both groups, with males comprising 53.8% of the controlled hypertension group and 58.3% of the resistant hypertension group, while females constituted 46.2% and 41.7%, respectively. However, significant differences were observed in body mass index (BMI), with resistant hypertensive patients exhibiting a higher mean BMI (34.6 ± 3.8 kg/m²) than those with controlled hypertension (32.9 ± 3.5 kg/m²; p = 0.01), suggesting a stronger correlation between obesity and blood pressure resistance. Furthermore, neck circumference and waist-hip ratio were significantly elevated in the resistant group (43.2 ± 2.6 cm and 0.96 ± 0.04, respectively) compared to the controlled group (40.1 ± 2.3 cm and 0.91 ± 0.05; p < 0.001 for both), indicating greater central adiposity and upper body fat deposition among patients with resistant hypertension. Although smoking history and sedentary lifestyle were more prevalent in the resistant group (41.6% and 67.7%, respectively) compared to the controlled group (32.7% and 55.8%), these differences did not reach statistical significance (p = 0.17 and p = 0.09, respectively). These findings collectively emphasize the role of obesity-related anthropometric measures in the pathogenesis and persistence of resistant hypertension as shown in table 1.

Table 1: Demographic Characteristics of the Study Population (N = 200)

Variable	Controlled Hypertension (n = 104)	Resistant Hypertension (n = 96)	p-value
Age (years)	50.7 ± 8.3	52.0 ± 9.1	0.18
Male Gender (%)	56 (53.8%)	56 (58.3%)	0.52
Female Gender (%)	48 (46.2%)	40 (41.7%)	
BMI (kg/m²)	32.9 ± 3.5	34.6 ± 3.8	0.01
Neck Circumference (cm)	40.1 ± 2.3	43.2 ± 2.6	<0.001
Waist-Hip Ratio	0.91 ± 0.05	0.96 ± 0.04	<0.001
Smoking History (%)	34 (32.7%)	40 (41.6%)	0.17
Sedentary Lifestyle (%)	58 (55.8%)	65 (67.7%)	0.09

Table- 2 illustrated the distribution of obstructive sleep apnea (OSA) severity among the study population, stratified by gender and hypertension status. Among the 200 obese hypertensive patients, 148 (74%) were diagnosed with OSA, with a significantly higher prevalence observed in those with resistant hypertension (92.7%) compared to those with controlled hypertension (56.7%) (p < 0.001). Gender-wise, OSA was more common in males, affecting 92 out of 112 men (82.1%), whereas 56 out of 88 women (63.6%) were diagnosed with the condition.

The severity of OSA also differed markedly between the two hypertension groups. In the resistant hypertension group, severe OSA was present in 34 patients (35.4%) compared to only 15 patients (14.4%) in the controlled hypertension group. Moderate OSA was seen in 34 patients (35.4%) with resistant hypertension versus 20 patients (19.2%) in the controlled group. Mild OSA was nearly equally distributed (21.9% vs. 23.1%). Notably, the proportion of patients without OSA was significantly higher in the controlled hypertension group (43.3%) compared to just 7.3% in the resistant group, reinforcing the strong association between OSA and treatment-resistant blood pressure. These results emphasize that not only is OSA more prevalent in males and patients with resistant hypertension, but its severity is also disproportionately greater in these subgroups as shown in table 2.

Table-3 showed a significant elevation in inflammatory and metabolic biomarkers among patients with resistant hypertension compared to those with controlled hypertension, across both genders. Levels of hs-CRP, IL-6, TNF-α, and serum aldosterone were markedly higher in resistant hypertensive males and females (p < 0.001), indicating heightened systemic inflammation and neurohormonal activation. Similarly, fasting glucose, HbA1c, LDL-C, and triglycerides were significantly elevated, while HDL-C levels were lower in the resistant group, reflecting poor metabolic control. These biomarker disparities were consistent in both males and females, underscoring a strong association between resistant hypertension, inflammation, and metabolic dysregulation.

Table- 4 highlighted the cardiovascular morbidity and complications among OSA patients, analyzed by gender and hypertension status. Left ventricular hypertrophy (LVH) was significantly more prevalent in males (70.6%) than females (39.3%) and was observed in 71.9% of resistant hypertensive patients with OSA compared to 39.0% of those with controlled hypertension (p < 0.001). Diastolic dysfunction followed a similar pattern, affecting 58.7% of males and 61.4% of resistant hypertensive individuals with OSA (p < 0.001). Myocardial infarction history was more common in the resistant group (24.7% vs. 13.5%; p = 0.03).

Congestive heart failure also showed higher prevalence in females and resistant cases (p = 0.04), while stroke incidence did not reach statistical significance (p = 0.08). These findings emphasize the elevated cardiovascular risk in OSA patients, particularly in males and those with resistant hypertension.

Table 2: Distribution of Obstructive Sleep Apnea Severity

OSA Severity	Male (n = 112)	Female (n = 88)	Controlled Hypertension (n = 104)	Resistant Hypertension (n = 96)	Total (N = 200)	p-value
No OSA	20	32	45 (43.3%)	7 (7.3%)	52 (26%)	<0.001
Mild OSA	26	19	24 (23.1%)	21 (21.9%)	45 (22.5%)	
Moderate OSA	34	20	20 (19.2%)	34 (35.4%)	54 (27%)	
Severe OSA	32	17	15 (14.4%)	34 (35.4%)	49 (24.5%)	
Total with OSA	92 (82.1%)	56 (63.6%)	59 (56.7%)	89 (92.7%)	148 (74%)	<0.001

Table 3: Gender-wise Comparison of Biomarkers Between Groups

Biomarker	Controlled HTN (Male, n = 56 / Female, n = 48)	Resistant HTN (Male, n = 56 / Female, n = 40)	p-value
hs-CRP (mg/L)	3.8 ± 1.2 (M), 3.9 ± 1.3 (F)	6.8 ± 1.7 (M), 7.1 ± 1.9 (F)	<0.001
IL-6 (pg/mL)	5.0 ± 1.2 (M), 5.2 ± 1.4 (F)	8.2 ± 2.0 (M), 8.6 ± 2.3 (F)	<0.001
TNF-α (pg/mL)	5.8 ± 2.1 (M), 6.2 ± 1.9 (F)	9.1 ± 2.4 (M), 9.4 ± 2.7 (F)	<0.001
Serum Aldosterone (ng/dL)	9.9 ± 3.5 (M), 10.4 ± 3.8 (F)	17.9 ± 4.2 (M), 18.8 ± 5.0 (F)	<0.001
Fasting Glucose (mg/dL)	117 ± 13 (M), 119 ± 15 (F)	131 ± 15 (M), 135 ± 17 (F)	<0.001
HbA1c (%)	6.7 ± 0.5 (M), 6.9 ± 0.6 (F)	7.4 ± 0.6 (M), 7.6 ± 0.7 (F)	<0.001
LDL-C (mg/dL)	109 ± 22 (M), 107 ± 21 (F)	128 ± 23 (M), 126 ± 25 (F)	<0.001
HDL-C (mg/dL)	43 ± 5 (M), 41 ± 5 (F)	37 ± 6 (M), 35 ± 6 (F)	<0.001
Triglycerides (mg/dL)	160 ± 30 (M), 156 ± 32 (F)	190 ± 36 (M), 183 ± 33 (F)	<0.001

Table 4: Gender-wise Cardiovascular Morbidity and Complications in Patients with OSA

Complication	Male (n = 92)	Female (n = 56)	Controlled HTN + OSA (n = 59)	Resistant HTN + OSA (n = 89)	p-value
Left Ventricular Hypertrophy (%)	65 (70.6%)	22 (39.3%)	23 (39.0%)	64 (71.9%)	<0.001
Diastolic Dysfunction (%)	54 (58.7%)	18 (32.1%)	17 (28.8%)	55 (61.4%)	<0.001
Myocardial Infarction History (%)	20 (21.7%)	10 (17.8%)	8 (13.5%)	22 (24.7%)	0.03
Stroke (%)	8 (8.7%)	9 (16.1%)	4 (6.8%)	13 (14.6%)	0.08
Congestive Heart Failure (%)	12 (13.0%)	12 (21.4%)	6 (10.1%)	18 (20.2%)	0.04

Multivariate logistic regression revealed that severe OSA (AHI ≥30) was an independent predictor of resistant hypertension (adjusted OR = 3.74; 95% CI: 2.01–6.89; p < 0.001), even after adjusting for age, gender, BMI, and metabolic risk factors. Elevated serum aldosterone and hs-CRP levels were also independently associated with resistant hypertension and adverse cardiovascular outcomes. These results suggest a strong association between obstructive sleep apnea and resistant hypertension in obese adults, further compounded by a heightened burden of systemic inflammation, metabolic dysregulation, and cardiovascular morbidity.

DISCUSSION

This comparative clinical study illustrated the compelling evidence for a strong and independent association between obstructive sleep apnea (OSA), resistant hypertension, and cardiovascular morbidity in obese adults. The findings reinforce the evolving paradigm that OSA is not merely a comorbid sleep-related breathing disorder but a significant pathophysiological contributor to treatment-resistant hypertension and its downstream cardiovascular complications [9]. The markedly higher prevalence and severity of OSA in patients with resistant hypertension (92.7%) compared to those with controlled hypertension (56.7%) underscores the critical role of nocturnal hypoxemia, sympathetic overactivation, and systemic inflammation in driving refractory blood pressure elevation [10].

Our results align with previous large-scale epidemiologic and mechanistic studies which have shown that OSA induces repetitive cycles of hypoxia and reoxygenation, leading to sustained elevations in catecholamines, endothelial dysfunction, and activation of the renin-angiotensin-aldosterone system (RAAS) [11]. The elevated levels of hs-CRP, IL-6, TNF-α, and serum aldosterone observed in the resistant hypertension group support this mechanistic hypothesis and highlight the chronic inflammatory and neurohormonal state that underlies the synergistic interaction between OSA and blood pressure dysregulation [12]. These findings are consistent with those of previous studies of scientists who reported that OSA is highly prevalent among patients with resistant hypertension and that treatment with continuous positive airway pressure (CPAP) significantly improves blood pressure control [13].

Gender-specific analysis revealed that males had a higher prevalence and severity of OSA compared to females, a pattern consistent with the literature, possibly reflecting anatomical and hormonal differences influencing upper airway collapsibility [14]. However, females in our study demonstrated a paradoxically higher incidence of certain cardiovascular outcomes such as stroke and congestive heart failure in the context of OSA, suggesting potential sex-based differences in disease expression or underdiagnosis of OSA in females due to atypical symptomatology [15]. This observation necessitates further investigation into gender-specific diagnostic and management strategies for OSA.

Importantly, the presence of OSA was significantly associated with adverse cardiac structural and functional changes, including left ventricular hypertrophy and diastolic dysfunction. These subclinical myocardial alterations likely reflect chronic hemodynamic stress from nocturnal blood pressure surges and hypoxia-induced myocardial remodeling [16]. The higher prevalence of myocardial infarction and heart failure in patients with resistant hypertension and OSA further supports the hypothesis that unrecognized and untreated sleep-disordered breathing may accelerate cardiovascular morbidity, independent of traditional metabolic risk factors [17].

The clinical implications of these findings are profound. Given the high burden of undiagnosed OSA among hypertensive individuals, particularly those with poor blood pressure control despite optimal pharmacotherapy, routine screening for OSA using validated questionnaires and overnight polysomnography should be integrated into hypertension management algorithms [18]. Additionally, the inflammatory and hormonal profiles observed in resistant hypertension with OSA suggest a potential benefit from adjunctive therapies targeting aldosterone excess and systemic inflammation. Moreover, the observed benefit of CPAP therapy in prior trials, including reductions in 24-hour blood pressure and left ventricular mass index, provides a therapeutic rationale for early diagnosis and intervention [13,19].

The strengths of this study were included its robust sample size, inclusion of both genders, use of standardized diagnostic criteria for OSA and resistant hypertension, and comprehensive biomarker profiling[19]. However, certain limitations must be acknowledged. The cross-sectional design precludes causal inferences, and longitudinal follow-up would be necessary to confirm temporal relationships. Additionally, CPAP adherence was not assessed, and its impact on outcomes could not be evaluated. Lastly, while this study included a diverse patient population, generalizability may be limited due to single-country data collection and potential referral bias from tertiary care settings [20]

CONCLUSION

This comparative clinical study reinforces the significant and reciprocal association between obstructive sleep apnea (OSA) and resistant hypertension in obese adults, highlighting a shared pathophysiological foundation involving sympathetic overactivation, systemic inflammation, and metabolic dysregulation. The high prevalence and severity of OSA in patients with resistant hypertension were strongly correlated with elevated inflammatory markers and increased incidence of cardiovascular complications such as left ventricular hypertrophy, diastolic dysfunction, and myocardial infarction. These findings emphasize the critical need for early screening, diagnosis, and integrated management of OSA in hypertensive individuals, particularly those with obesity and poor therapeutic response. Incorporating sleep medicine strategies into routine cardiovascular care could lead to improved blood pressure control, reduced cardiovascular morbidity, and overall enhancement of clinical outcomes.

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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Authors' Contributions: AS and SAZ contributed to study design, data collection, and manuscript drafting. MZAR and TM supervised polysomnographic and cardiovascular assessments. AAC and FA performed statistical analysis and contributed to data interpretation. All authors reviewed, revised, and approved the final manuscript.

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