



Cardiometabolic Implications of Obstructive Sleep Apnea: Linking Insulin Resistance to Right Ventricular Dysfunction

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Abstract

Obstructive sleep apnea is an increasingly recognized sleep-related breathing disorder strongly associated with cardiometabolic dysfunction, including insulin resistance and right ventricular impairment. The present study aimed to investigate the association between insulin resistance and right ventricular dysfunction in patients with obstructive sleep apnea and to evaluate its cardiometabolic implications in a hospital-based population. A cross-sectional observational study was conducted on 280 adults aged 30–65 years diagnosed with polysomnography-confirmed obstructive sleep apnea. Insulin resistance was assessed using the Homeostatic Model Assessment for Insulin Resistance, while right ventricular function was evaluated through echocardiographic parameters including tricuspid annular plane systolic excursion and right ventricular fractional area change. Results demonstrated significantly higher HOMA-IR values in patients with moderate-to-severe obstructive sleep apnea compared to mild disease ($p < 0.001$). Right ventricular systolic dysfunction was more prevalent in the insulin-resistant group, with a strong inverse correlation between HOMA-IR and tricuspid annular plane systolic excursion ($r = -0.62$, $p < 0.001$). Multivariate regression identified insulin resistance and apnea–hypopnea index as independent predictors of right ventricular dysfunction. The findings indicate a strong pathophysiological link between intermittent hypoxia-induced metabolic dysfunction and subclinical right heart impairment. Early identification of insulin resistance may serve as a prognostic marker for cardiovascular risk stratification in obstructive sleep apnea patients, supporting integrated cardiometabolic screening in sleep medicine practice.

Keywords: Obstructive sleep apnea, insulin resistance, right ventricular dysfunction

1. Introduction

Obstructive sleep apnea (OSA) is a chronic sleep-related breathing disorder characterized by recurrent upper airway collapse during sleep, leading to intermittent hypoxia, sleep fragmentation, and sympathetic nervous system activation. Over the past decade, OSA has emerged as a major contributor to cardiovascular morbidity and mortality, extending beyond a simple respiratory condition to a systemic cardiometabolic disorder. Increasing epidemiological evidence suggests that OSA is closely associated with metabolic dysfunction, particularly insulin resistance, which significantly amplifies cardiovascular risk [1–3].

Insulin resistance represents a key metabolic abnormality frequently observed in patients with OSA, independent of obesity status. The pathophysiological mechanisms include intermittent hypoxia-induced oxidative stress, systemic inflammation, and neurohormonal dysregulation. These mechanisms contribute to impaired glucose uptake, increased hepatic glucose production, and altered lipid metabolism. Recent studies have demonstrated that even mild OSA can trigger early metabolic disturbances, suggesting a dose-dependent relationship between sleep-disordered breathing severity and insulin sensitivity impairment [2–4].

Right ventricular dysfunction has increasingly been recognized as a subclinical but important cardiovascular consequence of OSA. Unlike left ventricular dysfunction, right ventricular impairment is primarily driven by pulmonary vascular changes resulting from hypoxic pulmonary vasoconstriction and increased pulmonary artery pressure. Chronic intermittent hypoxia leads to structural remodeling and functional impairment of the right ventricle, which may progress silently until advanced disease stages [3–5].

Recent cardiology research has highlighted the interplay between metabolic dysfunction and right ventricular performance. Insulin resistance contributes to endothelial dysfunction, vascular stiffness, and impaired myocardial energy metabolism, which together exacerbate right ventricular strain. This cardiometabolic interaction suggests that OSA should be conceptualized as a systemic disorder affecting both metabolic and cardiac systems simultaneously rather than an isolated respiratory condition [4–6].

Despite increasing awareness, there remains a lack of integrated clinical models that simultaneously evaluate insulin resistance and right ventricular function in OSA patients. Most existing studies focus independently on metabolic or cardiovascular outcomes, limiting understanding of their combined impact. Contemporary evidence emphasizes the need for comprehensive cardiometabolic assessment models to improve early detection of high-risk OSA phenotypes [5–8].

Given these gaps, the present study was designed to evaluate the relationship between insulin resistance and right ventricular dysfunction in patients with obstructive sleep apnea. By integrating metabolic and echocardiographic parameters, this study aims to provide a clinically relevant framework for early risk stratification and improved management of cardiometabolic complications associated with OSA [7–10].

2. Methodology

This hospital-based cross-sectional observational study was conducted at University Of Lahore. A total of 280 adult patients aged 30–65 years with newly diagnosed obstructive sleep apnea confirmed by overnight polysomnography were enrolled. Ethical approval was obtained from the institutional review board, and verbal informed consent was obtained from all participants prior to inclusion.

Sample size was calculated using Epi Info software based on an expected moderate correlation ($r = 0.3$) between insulin resistance and right ventricular dysfunction, with 95% confidence interval and 80% statistical power. The minimum required sample size was 252, which was increased to 280 to improve statistical validity and account for incomplete data.

Inclusion criteria included adults with apnea–hypopnea index ≥ 5 events/hour diagnosed by polysomnography and no prior treatment with continuous positive airway pressure therapy. Exclusion criteria included history of chronic pulmonary disease, congenital heart disease, left ventricular failure, diabetes mellitus requiring insulin therapy, and chronic kidney disease.

Clinical evaluation included anthropometric measurements, blood pressure recording, and sleep study data analysis. Insulin resistance was assessed using fasting glucose and fasting insulin levels to calculate HOMA-IR. Patients were categorized into insulin-resistant and non-insulin-resistant groups based on standardized cut-off values.

Echocardiographic assessment was performed using standard transthoracic echocardiography. Right ventricular systolic function was evaluated using tricuspid annular plane systolic excursion, right ventricular fractional area change, and tissue Doppler imaging where applicable. All measurements were performed by an experienced cardiologist blinded to clinical data.

Statistical analysis was conducted using SPSS software. Continuous variables were expressed as mean $\pm$ standard deviation and categorical variables as percentages. Independent t-test and chi-square test were used for group comparisons. Pearson correlation analysis was used to assess relationships between variables. Multivariate regression analysis was performed to identify independent predictors of right ventricular dysfunction. A p-value <0.05 was considered statistically significant.

3. Results

Table 1: Baseline clinical and metabolic characteristics

Variable	OSA Mild (n=90)	Moderate (n=100)	Severe (n=90)	p-value
Age (years)	44.2 $\pm$ 9.1	46.8 $\pm$ 8.7	48.5 $\pm$ 9.3	0.03
BMI (kg/m ²)	28.1 $\pm$ 3.9	30.2 $\pm$ 4.1	32.5 $\pm$ 4.6	<0.001
HOMA-IR	2.1 $\pm$ 0.9	3.4 $\pm$ 1.2	5.2 $\pm$ 1.8	<0.001
Fasting insulin (μ IU/mL)	10.3 $\pm$ 3.4	14.7 $\pm$ 4.1	19.6 $\pm$ 5.3	<0.001

Interpretation: Insulin resistance increased significantly with severity of obstructive sleep apnea.

Table 2: Right ventricular functional parameters

Parameter	Non-IR group	IR group	p-value
TAPSE (mm)	22.4 $\pm$ 3.1	17.6 $\pm$ 2.8	<0.001
RVFAC (%)	45.8 $\pm$ 6.2	37.1 $\pm$ 5.4	<0.001
RV systolic dysfunction (%)	18%	52%	<0.001

Interpretation: Insulin-resistant patients showed significantly reduced right ventricular function.

Table 3: Multivariate regression analysis

Predictor	Odds Ratio	p-value
HOMA-IR elevation	3.9	<0.001
Severe OSA (AHI >30)	3.2	0.001
BMI >30 kg/m ²	2.4	0.01
Age >50 years	1.8	0.04

Interpretation: Insulin resistance emerged as the strongest independent predictor of right ventricular dysfunction.

Discussion

The present study demonstrates a strong association between insulin resistance and right ventricular dysfunction in patients with obstructive sleep apnea. These findings reinforce the concept that OSA is not merely a respiratory disorder but a systemic cardiometabolic disease involving complex metabolic and cardiovascular interactions [11–12].

The progressive increase in HOMA-IR with OSA severity supports the role of intermittent hypoxia in promoting metabolic dysregulation. Hypoxia-induced sympathetic activation and oxidative stress contribute to impaired insulin signaling pathways, resulting in systemic insulin resistance even in non-diabetic individuals [12–13].

The observed impairment in right ventricular function among insulin-resistant patients suggests a mechanistic link between metabolic dysfunction and cardiac remodeling. Insulin resistance may exacerbate pulmonary vascular remodeling, increasing right ventricular afterload and leading to systolic dysfunction over time [13–14].

The strong inverse correlation between HOMA-IR and TAPSE highlights the clinical relevance of metabolic markers in predicting subclinical cardiac dysfunction. These findings are consistent with prior cardiology studies demonstrating that metabolic syndrome components significantly affect right ventricular performance [14–16].

Multivariate analysis confirmed insulin resistance as an independent predictor of right ventricular dysfunction, even after adjusting for obesity and OSA severity. This indicates that metabolic dysfunction plays a direct role in cardiac impairment beyond mechanical effects of airway obstruction [15–17].

From a clinical perspective, early identification of insulin resistance in OSA patients may allow timely intervention to prevent progression of right heart dysfunction. This supports the integration of metabolic screening in sleep medicine practice as part of comprehensive cardiovascular risk assessment [16–18].

Overall, the study emphasizes the need for an integrated cardiometabolic approach in obstructive sleep apnea management. Addressing both sleep-disordered breathing and metabolic dysfunction may significantly reduce long-term cardiovascular complications and improve patient outcomes [18–20].

Conclusion

Insulin resistance is strongly associated with right ventricular dysfunction in obstructive sleep apnea.

Metabolic impairment significantly contributes to subclinical cardiovascular remodeling in OSA patients.

Integrated cardiometabolic screening is essential for early risk stratification and prevention of cardiac complications.

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