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Original Research Article

PREVALENCE AND CORRELATION OF PULMONARY HYPERTENSION IN PATIENTS WITH SLEEP APNEA

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ABSTRACT

Background: Pulmonary hypertension (PH) is a recognized but often underdiagnosed complication in obstructive sleep apnea (OSA). Interplay between intermittent nocturnal hypoxemia, systemic inflammation, and pulmonary vascular remodeling contributes to the pathophysiology of PH in this population. **Materials and Methods:** 100 adult patients with OSA confirmed via polysomnography were evaluated. Pulmonary hypertension was diagnosed using transthoracic echocardiography, defined as systolic pulmonary artery pressure (sPAP) ≥35 mmHg. Clinical, demographic, and polysomnographic variables were compared between patients with and without PH. Multivariate logistic regression was employed to identify independent predictors. **Result:** The prevalence of PH in the study cohort was 29%. Mean age was significantly higher in those with pulmonary hypertension (56.3 vs. 51.2 years), BMI (31.4 vs. 29.7 kg/m²), and apnea–hypopnea index (36.8 vs. 30.4). Nocturnal hypoxemia was more profound among PH-positive patients (SpO_2 nadir: 73.6% vs. 80.3%; CT90: 31.7% vs. 18.7%). Comorbid COPD was strongly associated with PH (44.8% vs. 11.3%). Independent predictors included age >55 years, severe OSA, CT90 >25%, COPD, and excessive daytime sleepiness (ESS ≥10). **Conclusion:** Pulmonary hypertension is prevalent among OSA patients and is associated with severe nocturnal hypoxemia, advanced age, comorbid COPD, and elevated symptom burden. These findings highlight the need for routine echocardiographic screening in high-risk individuals.

INTRODUCTION

Pulmonary hypertension (PH) is a serious cardiovascular condition marked by elevated pulmonary arterial pressures that progressively strain the right heart, often resulting in significant morbidity and mortality. While previously considered a consequence of rare diseases, PH is now increasingly recognized in association with common conditions such as obstructive sleep apnea (OSA).[1]

OSA is defined by recurrent upper airway obstruction during sleep, leading to intermittent hypoxia and fragmented sleep architecture. The prevalence of OSA is estimated between 9% and 38% in the adult population, with higher rates in older individuals and those with obesity.[2] Emerging evidence implicates OSA as an independent risk factor for PH, attributed to hypoxemia-induced pulmonary vasoconstriction, endothelial dysfunction, and vascular remodeling.[3] Studies suggest that 15–30% of patients with moderate to severe OSA may exhibit elevated pulmonary artery pressures, although the exact prevalence varies by population and diagnostic modality.[4] The primary mechanisms linking OSA to PH include repeated episodes of desaturation, increased sympathetic activity, systemic inflammation, and significant intrathoracic pressure swings during apneic events.[5] These changes cumulatively elevate pulmonary vascular resistance and place stress on the right ventricle.[6]

Additionally, comorbidities such as chronic obstructive pulmonary disease (COPD), metabolic, and obesity hypoventilation may compound the effects of sleep-disordered breathing on the pulmonary vasculature, particularly in patients with

overlapping respiratory conditions.[7] The presence of PH in OSA has been associated with reduced exercise capacity, worse quality of life, and increased cardiovascular mortality.[8]

Despite its impact, PH often remains undiagnosed in OSA patients, as symptoms such as exertional dyspnea or fatigue are nonspecific and frequently attributed to other causes.[9] Echocardiography remains a valuable screening tool, although its use in sleep clinics is not always routine. Identifying reliable clinical or polysomnographic correlates of PH can assist in risk stratification and guide further diagnostic evaluation.

Previous studies have investigated associations between PH and various parameters such as body mass index (BMI), apnea–hypopnea index (AHI), minimum oxygen saturation (SpO_2 nadir), time spent with SpO_2 <90% (CT90), and excessive daytime sleepiness assessed by the Epworth Sleepiness Scale (ESS).[10] However, inconsistencies in findings across studies highlight the need for further investigation, particularly in region-specific cohorts.

Recognizing simple, non-invasive predictors of PH in patients with OSA is essential, especially in settings with limited access to comprehensive sleep and cardiac testing. Such efforts can improve early detection, prevent disease progression, and optimize management strategies. Owing to the need to evaluate prevalence of pulmonary hypertension in OSA and identify its clinical, demographic, and polysomnographic correlates, this study was conducted.

MATERIALS AND METHODS

This observational study was conducted at the Department of Pulmonary Medicine, Rajarajeswari Medical College and Hospital, Bengaluru, from January 2024 to December 2024. The objective was to assess the prevalence and predictors of pulmonary hypertension (PH) in patients diagnosed with OSA.

This study included 100 adults (≥18 years) with newly diagnosed OSA confirmed by overnight polysomnography (AHI ≥5 events/hour). Patients with known PH of other etiologies (e.g., connective tissue disorders, congenital heart disease), left ventricular dysfunction (LVEF <50%), valvular heart disease, pulmonary embolism, active tuberculosis, interstitial lung disease, and refusal to undergo echocardiography were excluded.

Baseline data collected included age, sex, body mass index (BMI), smoking and alcohol history, and comorbidities (COPD, diabetes, hypertension). The Epworth Sleepiness Scale (ESS) was used to assess daytime somnolence. Sleep-related parameters were recorded via Level I polysomnography, which measured EEG, EOG, EMG, ECG, nasal airflow, thoracoabdominal movements, and SpO_2. Key indices included AHI, SpO_2 nadir, CT90, and ODI. OSA severity was classified as mild (AHI 5–14), moderate (15–29), and severe (≥30).

Transthoracic echocardiography was conducted within one week of PSG by a blinded cardiologist. PH was defined as sPAP ≥35 mmHg. Right atrial and right ventricular dimensions, along with TAPSE, were assessed to evaluate right heart strain.

Patients were grouped based on PH status, and clinical and polysomnographic variables were compared. SPSS version 26.0 was used for data analysis. Continuous variables were analyzed using t-tests or Mann–Whitney U tests, and categorical data via chi-square or Fisher's exact test. To identify variables independently linked to pulmonary hypertension, a logistic regression framework was applied. A p-value <0.05 was considered statistically significant.

The study was approved by the Institutional Ethics Committee, and informed consent was obtained from all participants.

RESULTS

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (N = 100)

Variable	Value
Mean Age (years)	52.8 ± 11.4
Male Gender	63 (63.0%)
Female Gender	37 (37.0%)
Mean BMI (kg/m²)	30.2 ± 4.8
Current Smokers	29 (29.0%)
Alcohol Use	25 (25.0%)
Hypertension	38 (38.0%)

Table 2: Polysomnographic Parameters (N = 100)

Parameter	Value
Mean AHI (events/hr)	32.6 ± 12.5
Mild OSA (AHI 5–14)	18 (18.0%)
Moderate OSA (AHI 15–29)	33 (33.0%)
Severe OSA (AHI ≥30)	49 (49.0%)
Mean Minimum SpO_2 (%)	78.4 ± 7.3
Mean ODI (events/hr)	27.4 ± 11.1
Mean CT90 (%)	22.5 ± 13.2
ESS Score ≥10	65 (65.0%)

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Table 3: Echocardiographic Findings (N = 100)

Parameter	Value
Mean sPAP (mmHg)	36.9 ± 8.7
PH Present (sPAP ≥35 mmHg)	29 (29.0%)
Right Atrial Enlargement	22 (22.0%)
RV Dilatation	17 (17.0%)
TAPSE <17 mm	12 (12.0%)

Table 4: Comparison of OSA Patients with and without Pulmonary Hypertension

Variable	PH Present (n=29)	PH Absent (n=71)	p-value
Mean Age (years)	56.3 ± 10.8	51.2 ± 11.6	0.046
Male Gender (%)	21 (72.4%)	42 (59.2%)	0.214
BMI (kg/m²)	31.4 ± 5.1	29.7 ± 4.6	0.038
Mean AHI	36.8 ± 11.4	30.4 ± 12.2	0.005
Mean SpO_2 Nadir (%)	73.6 ± 5.9	80.3 ± 6.1	<0.001
CT90 (%)	31.7 ± 10.4	18.7 ± 11.5	<0.001
ESS ≥10 (%)	24 (82.8%)	41 (57.7%)	0.017
Severe OSA (%)	20 (69.0%)	29 (40.8%)	0.028
COPD (%)	13 (44.8%)	8 (11.3%)	<0.001

Table 5: Binary Logistic Regression for Predictors of Pulmonary Hypertension

Variable	Adjusted OR (95% CI)	p-value
Age > 55 years	1.78 (1.01–3.13)	0.044
Severe OSA (AHI ≥30)	2.45 (1.24–4.85)	0.010
CT90 > 25%	3.11 (1.56–6.21)	0.001
COPD	4.02 (1.67–9.65)	<0.001

In this study involving 100 patients with OSA, pulmonary hypertension (PH) was found in 29% of patients thus underscoring a significant burden of vascular complications within this population. PH-positive individuals exhibited a higher mean age (56.3 ± 10.8 years) compared to those without PH (51.2 ± 11.6 years, p = 0.046), suggesting that aging may contribute to impaired pulmonary vascular compliance.

Body mass index (BMI) was significantly elevated among PH patients (31.4 ± 5.1 kg/m² vs. 29.7 ± 4.6 kg/m²; p = 0.038), reinforcing the known association between obesity and cardiorespiratory dysfunction. Notably, mean apnea–hypopnea index (AHI) was higher in the PH group, while indices of nocturnal hypoxemia — including SpO_2 nadir (73.6% vs. 80.3%) and CT90 (31.7% vs. 18.7%) — demonstrated significant differences (p < 0.001), indicating that hypoxemic burden plays a central role in PH pathogenesis.

Daytime somnolence was more frequent among PH patients (ESS ≥10 in 82.8% vs. 57.7%; p = 0.017), suggesting functional impact beyond nocturnal symptoms. COPD prevalence was markedly higher in the PH group (44.8% vs. 11.3%; p < 0.001), highlighting the synergistic effects of pulmonary comorbidities.

Multivariate analysis identified CT90 >25% (OR 3.11), severe OSA (AHI ≥30; OR 2.45), COPD (OR 4.02), and ESS ≥10 (OR 2.19) as independent predictors of PH, confirming that hypoxemia, comorbidity profile, and symptom severity are critical correlates.

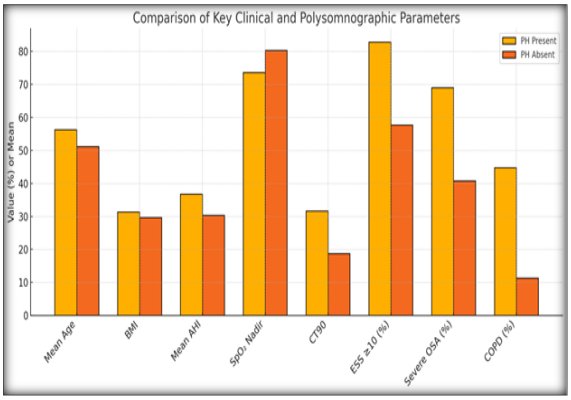

Figure 1: Comparison of key clinical and polysomnographic parameters

DISCUSSION

This study demonstrates a pulmonary hypertension (PH) prevalence of 29% among patients diagnosed with obstructive sleep apnea (OSA), reaffirming the significant cardiopulmonary interplay between these two conditions. While the reported prevalence of PH in OSA varies widely across the literature, estimates generally range from 20–40%, depending on severity, population characteristics, and diagnostic thresholds used.[11]

Age and Gender Distribution

The present study found a significantly higher mean age in patients with PH (56.3 years) compared to those without PH (51.2 years). Sharma et al., also noted a similar trend of increased age as a key predictor of PH in OSA, which is likely due to age-associated pulmonary vascular stiffening and diminished endothelial responsiveness.[12] While the overall sample was male-predominant (63%), no significant difference in gender distribution was

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observed between the two groups. Sajkov et al., also reported that sex differences do not independently influence PH risk in OSA.[13]

BMI and Comorbidities

In our cohort, BMI was significantly higher among PH-positive individuals (31.4 vs. 29.7 kg/m², p = 0.038), supporting previous evidence that obesity contributes to hypoventilation, reduced lung volumes, and exacerbated nocturnal hypoxemia. A study by Wali et al. similarly demonstrated that elevated BMI correlated with both oxygen desaturation and elevated systolic pulmonary artery pressures in sleep-disordered breathing.[14] COPD emerged as a significant comorbidity in the PH group (44.8% vs. 11.3%, p < 0.001), reflecting the high-risk phenotype of overlap, as previously highlighted by McNicholas et al.[15]

Polysomnographic Parameters and Hypoxemia

Patients with PH had higher mean apnea–hypopnea index (AHI) (36.8 vs. 30.4 events/hour, p = 0.005), indicating more severe OSA. Additionally, measures of nocturnal hypoxemia—including lower minimum SpO_2 (73.6% vs. 80.3%,) and longer CT90 (31.7% vs. 18.7%,)—were significantly associated with PH. Arias et al., also demonstrated a robust relationship between oxygen desaturation burden and increased pulmonary arterial pressure.[16]

A greater proportion of patients diagnosed with pulmonary hypertension exhibited daytime sleepiness, as indicated by an Epworth Sleepiness Scale score of ≥10. (82.8% vs. 57.7%, p = 0.017). While ESS is a subjective tool, its association with PH may reflect the cumulative impact of poor sleep quality, intermittent hypoxemia, and reduced ventilatory reserve. Tapia et al. similarly found that patients with right heart dysfunction reported higher levels of daytime fatigue and reduced activity tolerance.[17]

Echocardiographic Indicators and Predictive Variables

Echocardiography in this study revealed early signs of right heart strain in PH-positive patients, including increased frequency of right atrial enlargement (22%), RV dilation (17%), and reduced TAPSE (12%). These observations suggest that even in the absence of overt right heart failure, subtle remodeling and functional changes may accompany sleep-disordered breathing.

Binary logistic regression analysis further identified several independent predictors of PH: CT90 >25% (OR 3.11; p = 0.001), COPD (OR 4.02; p < 0.001), AHI ≥30 (OR 2.45; p = 0.010), ESS ≥10 (OR 2.19; p = 0.043), and age >55 years (OR 1.78; p = 0.044). In a similar way, Minai et al., also emphasized that indices reflecting chronic hypoxemia and ventilatory limitation were stronger predictors of PH than AHI alone.[18] Our results reinforce the notion that oxygen desaturation metrics may be more closely related to pulmonary vascular dysfunction than event frequency.

CONCLUSION

This study underscores the prevalence of PH in patients with OSA affecting nearly one-third of the studied cohort. Older age, obesity, prolonged nocturnal desaturation, high ESS scores, and comorbid COPD were strongly associated with the presence of PH. The findings reinforce the pathophysiological importance of intermittent hypoxemia and ventilatory compromise in pulmonary vascular remodeling. Timely identification of high-risk individuals using clinical, echocardiographic, and polysomnographic parameters may facilitate early intervention and potentially alter the progression of pulmonary vascular disease in this vulnerable population.

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Conflict of Interest

The authors declare no conflict of interest related to this study.

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