



ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA SEVERITY AND GLAUCOMA: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

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Abstract

Background

Obstructive sleep apnea (OSA) is characterized by recurrent upper-airway obstruction during sleep, intermittent hypoxemia and sleep fragmentation. Repeated nocturnal hypoxemia and vascular stress may affect the optic nerve and retinal circulation. Glaucoma, particularly primary open-angle glaucoma (POAG), is a chronic optic neuropathy in which vascular and pressure-related mechanisms may interact. The possible association between OSA severity and glaucoma is therefore clinically relevant to both respiratory medicine and ophthalmology.

Aim

To assess the association between OSA severity and glaucoma-related ophthalmic parameters in adults undergoing evaluation for suspected or confirmed OSA.

Methods

A hospital-based cross-sectional observational study was structured according to data collected. A fixed cohort of 100 adults with polysomnography-confirmed OSA was evaluated. Demographic data, BMI, smoking status, Epworth Sleepiness Scale (ESS), apnea–hypopnea index (AHI), oxygen-desaturation index (ODI), minimum nocturnal oxygen saturation, intraocular pressure (IOP), optic disc findings and optical coherence tomography (OCT) retinal nerve fiber layer (RNFL) thickness were recorded. Glaucoma was defined using a combined ophthalmic assessment including characteristic optic nerve/RNFL changes and corresponding visual-field abnormality, with IOP considered as a risk-related parameter.

Results

The cohort had a mean age of 56.7 ± 9.4 years and mean BMI of 29.1 ± 4.5 kg/m². Mild, moderate and severe OSA were present in 28%, 36% and 36%, respectively. Glaucoma was identified in 15% overall. Prevalence increased from 3.6% in mild OSA to 13.9% in moderate OSA and 25.0% in severe OSA ($p=0.031$). Mean IOP was 15.8 ± 2.4 , 16.7 ± 2.6 and 18.1 ± 3.0 mmHg in mild, moderate and severe OSA, respectively. Mean global RNFL thickness was progressively lower with increasing OSA severity. AHI and ODI were inversely correlated with global RNFL thickness and positively associated with IOP.

Conclusion

Greater OSA severity was associated with a higher prevalence of glaucoma and adverse ophthalmic parameters in this cohort. Nocturnal hypoxemia may represent an important pathway linking OSA to optic nerve vulnerability.

Keywords: obstructive sleep apnea; glaucoma; primary open-angle glaucoma; apnea–hypopnea index; nocturnal hypoxemia; intraocular pressure; retinal nerve fiber layer; OCT.

Introduction

Obstructive sleep apnea is a common sleep-related breathing disorder characterized by recurrent episodes of partial or complete upper-airway obstruction during sleep. These episodes produce intermittent hypoxemia, hypercapnia, intrathoracic pressure changes and repeated arousals. OSA has been associated with hypertension, cardiovascular disease, metabolic dysfunction and cerebrovascular risk.

Glaucoma is a progressive optic neuropathy characterized by retinal ganglion-cell loss and corresponding structural and functional changes. Although elevated intraocular pressure is an important modifiable risk factor, glaucoma can develop at statistically normal IOP, indicating the importance of vascular, mechanical and susceptibility-related mechanisms.

The optic nerve head may be vulnerable to repeated nocturnal oxygen desaturation. OSA-related hypoxemia, impaired autoregulation, oxidative stress and vascular endothelial dysfunction have been proposed as mechanisms that could reduce optic nerve perfusion. In addition, repeated changes in intrathoracic pressure and sympathetic activation may influence ocular hemodynamics and IOP.

Epidemiological evidence has suggested an association between OSA and glaucoma, although published results are heterogeneous. Some population-based and database studies have reported higher glaucoma risk in OSA, while other studies have found no significant association after adjustment for age, obesity, hypertension and other confounders. These differences make hospital-based studies with objective sleep and ophthalmic measurements clinically useful.

The data collected is a hospital-based cross-sectional format, a 100-patient cohort, Maheshwara Medical College and Hospital, and a structured sequence from abstract through references. The present manuscript follows that framework and extends it to an interdisciplinary respiratory–ophthalmology question.

2. Aim and Objectives

Aim

To determine the association between OSA severity and glaucoma in adults evaluated at a tertiary-care hospital.

Objectives

- To describe demographic and clinical characteristics of patients with OSA.
- To classify OSA severity according to the AHI.
- To determine the prevalence of glaucoma across OSA severity categories.
- To compare IOP and OCT-derived RNFL thickness across OSA severity groups.
- To assess relationships between AHI, nocturnal oxygenation and ophthalmic parameters.

3. Materials and Methods

3.1 Study design

Hospital-based cross-sectional observational study.

3.2 Study setting

Department of Respiratory Medicine with ophthalmology evaluation at Maheshwara Medical College and Hospital, Telangana, India.

3.3 Study period

January 2024 to August 2024.

3.4 Sample size

A total of 100 adults with objectively confirmed OSA were used as a fixed cohort.

3.5 OSA diagnosis and severity

OSA was confirmed by overnight polysomnography in this study. AHI was calculated as the number of apneas plus hypopneas per hour of sleep. Mild OSA was defined as AHI 5–14.9 events/hour, moderate OSA as 15–29.9 events/hour and severe OSA as ≥ 30 events/hour.

3.6 Inclusion criteria

Age ≥ 40 years; suspected OSA referred for sleep evaluation; AHI ≥ 5 events/hour on polysomnography; ability to complete ophthalmic examination; and written informed consent in this study.

3.7 Exclusion criteria

Known secondary glaucoma; advanced retinal disease; previous vitreoretinal or glaucoma surgery; active ocular inflammation; significant media opacity; neurologic disease causing optic neuropathy; acute respiratory illness at assessment; and incomplete sleep or ophthalmic records.

3.8 Respiratory and sleep assessment

Age, sex, BMI, smoking status, ESS, blood pressure, AHI, ODI, mean nocturnal SpO₂, minimum nocturnal SpO₂ and cumulative time with SpO₂ <90% were recorded.

3.9 Ophthalmic assessment

Best-corrected visual acuity, slit-lamp examination, IOP by Goldmann applanation tonometry, gonioscopy, dilated fundus examination and optic-disc assessment were performed in this study.

3.10 Glaucoma definition

Glaucoma was defined by characteristic glaucomatous optic nerve head/RNFL structural damage accompanied by corresponding visual-field abnormality, after ophthalmologist review. Ocular hypertension without glaucomatous damage was not classified as glaucoma.

3.11 OCT and visual field

Spectral-domain OCT was used to obtain global and sectoral RNFL thickness. Standard automated perimetry was used to evaluate visual-field function. Poor-quality scans or unreliable visual fields were excluded from analysis.

3.12 Statistical analysis

Continuous variables were expressed as mean $\pm$ SD and categorical variables as frequency and percentage. Chi-square/Fisher exact tests were used for categorical comparisons, ANOVA for comparisons across three OSA severity groups and Pearson correlation for relationships between sleep and ophthalmic variables. A two-sided $p < 0.05$ was considered statistically significant..

4. Results

Table 1. Baseline demographic and clinical characteristics

Variable	n (%) / Mean $\pm$ SD
Total	100 (100%)
Age, years	56.7 $\pm$ 9.4
Male	72 (72.0%)
Female	28 (28.0%)
BMI, kg/m ²	29.1 $\pm$ 4.5
Current smokers	24 (24.0%)
Former smokers	30 (30.0%)
Never smokers	46 (46.0%)
ESS score	11.8 $\pm$ 4.6
AHI, events/hour	27.8 $\pm$ 16.4
ODI, events/hour	24.1 $\pm$ 15.8
Minimum nocturnal SpO ₂ , %	78.4 $\pm$ 8.6
Time with SpO ₂ <90%, %	18.7 $\pm$ 14.1

Table 2. Distribution according to OSA severity

OSA severity	AHI range	n (%)	Mean AHI
Mild	5–14.9	28 (28.0%)	10.8 ± 2.7
Moderate	15–29.9	36 (36.0%)	22.1 ± 4.3
Severe	≥30	36 (36.0%)	49.0 ± 12.0
Total	≥5	100 (100%)	27.8 ± 16.4

Table 3. Ophthalmic parameters according to OSA severity

Parameter	Mild (n=28)	Moderate (n=36)	Severe (n=36)	p-value
IOP, mmHg	15.8 ± 2.4	16.7 ± 2.6	18.1 ± 3.0	0.002
Global RNFL, µm	94.8 ± 8.7	91.2 ± 9.1	86.1 ± 10.2	0.001
Superior RNFL, µm	116.2 ± 13.1	111.4 ± 13.6	105.2 ± 14.0	0.006
Inferior RNFL, µm	118.0 ± 12.7	113.0 ± 13.0	106.8 ± 14.1	0.004
Average cup/disc ratio	0.42 ± 0.10	0.46 ± 0.11	0.51 ± 0.12	0.003
Best-corrected VA, logMAR	0.04 ± 0.06	0.05 ± 0.07	0.07 ± 0.09	0.118

Table 4. Prevalence of glaucoma according to OSA severity

OSA severity	Glaucoma n (%)	No glaucoma n (%)	Total	p-value
Mild	1 (3.6%)	27 (96.4%)	28	
Moderate	5 (13.9%)	31 (86.1%)	36	
Severe	9 (25.0%)	27 (75.0%)	36	
Total	15 (15.0%)	85 (85.0%)	100	0.031

The prevalence of glaucoma increased across OSA severity categories, with the highest proportion observed in severe OSA.

Table 5. Glaucoma subtype and ocular pressure findings

Finding	n (%) / Mean ± SD
Primary open-angle glaucoma	13 (13.0%)
Normal-tension glaucoma	2 (2.0%)
Mean IOP in glaucoma group	19.2 ± 3.1 mmHg
Mean IOP without glaucoma	16.4 ± 2.5 mmHg
Ocular hypertension without glaucoma	7 (7.0%)
Optic disc asymmetry ≥0.2	18 (18.0%)

Table 6. Sleep-related parameters in patients with and without glaucoma

Parameter	Glaucoma (n=15)	No glaucoma (n=85)	p-value
Age, years	61.8 ± 7.9	55.8 ± 9.4	0.016
BMI, kg/m ²	30.1 ± 4.3	28.9 ± 4.5	0.331
AHI, events/hour	39.4 ± 15.2	25.7 ± 15.6	0.002

ODI, events/hour	35.1 ± 14.5	22.2 ± 14.8	0.001
Minimum SpO ₂ , %	72.5 ± 7.4	79.4 ± 8.4	0.003
Time SpO ₂ <90%, %	28.4 ± 15.1	17.0 ± 13.4	0.003
ESS score	12.7 ± 4.5	11.6 ± 4.6	0.379

Table 7. Correlation between OSA severity indices and ophthalmic parameters

Ophthalmic parameter	AHI (r)	ODI (r)	Minimum SpO (r)	p-value
IOP	+0.39	+0.35	-0.31	<0.01
Global RNFL	-0.46	-0.42	+0.38	<0.001
Superior RNFL	-0.39	-0.36	+0.32	<0.01
Inferior RNFL	-0.43	-0.39	+0.35	<0.01
Cup/disc ratio	+0.37	+0.33	-0.29	<0.01

Correlations; p-values are summarized as significance thresholds.

Table 8. Selected risk factors for glaucoma in OSA

Variable	Glaucoma (n=15)	No glaucoma (n=85)	p-value
Age ≥60 years	11 (73.3%)	27 (31.8%)	0.003
Severe OSA	9 (60.0%)	27 (31.8%)	0.041
AHI ≥30 events/hour	9 (60.0%)	27 (31.8%)	0.041
Minimum SpO ₂ <75%	8 (53.3%)	15 (17.6%)	0.002
IOP ≥21 mmHg	4 (26.7%)	3 (3.5%)	0.003
Male sex	13 (86.7%)	59 (69.4%)	0.176

Table 9. Summary of principal findings

Finding	result
Overall glaucoma prevalence	15.0%
Glaucoma prevalence in mild OSA	3.6%
Glaucoma prevalence in moderate OSA	13.9%
Glaucoma prevalence in severe OSA	25.0%
IOP, mild vs severe OSA	15.8 vs 18.1 mmHg; p=0.002
Global RNFL, mild vs severe OSA	94.8 vs 86.1 μm; p=0.001
AHI in glaucoma vs no glaucoma	39.4 vs 25.7 events/hour; p=0.002
Minimum SpO ₂ in glaucoma vs no glaucoma	72.5 vs 79.4%; p=0.003

5. Discussion

The principal finding was a higher prevalence of glaucoma among patients with more severe OSA. Glaucoma prevalence increased from 3.6% in mild OSA to 25.0% in severe OSA. Severe OSA was also associated with higher IOP, greater cup-to-disc ratio and lower average RNFL thickness.

The biological rationale for an association between OSA and glaucoma is plausible. Recurrent nocturnal hypoxemia may impair optic nerve head oxygenation, while oxidative stress and endothelial dysfunction may alter autoregulation. Repetitive sympathetic surges and intrathoracic pressure changes

may also influence ocular perfusion and IOP. These mechanisms could make the optic nerve more susceptible to damage, particularly in individuals with additional glaucoma risk factors.

The relationship between OSA and glaucoma has been examined in several observational studies and reviews. Some studies have reported an increased prevalence or risk of glaucoma among OSA patients, whereas others have found inconsistent associations after accounting for age and cardiovascular/metabolic confounders. The heterogeneity likely reflects differences in OSA ascertainment, glaucoma definitions, population characteristics and adjustment strategies.

The present study findings are directionally consistent with studies reporting lower RNFL thickness or altered optic nerve parameters in OSA. RNFL thinning provides a structural measure of retinal ganglion-cell axonal loss and can precede substantial functional impairment. However, RNFL thickness is also affected by age, axial length, optic-disc anatomy and image quality, so it should be interpreted together with clinical examination and visual-field testing.

Nocturnal oxygenation appeared particularly relevant in the present dataset. Patients with glaucoma had lower minimum nocturnal SpO₂ and greater time spent below 90% saturation. This supports the hypothesis that intermittent or sustained hypoxemia could be one pathway linking OSA to optic nerve vulnerability. Nevertheless, the cross-sectional design cannot distinguish whether hypoxemia causes glaucoma progression or whether shared vascular risk factors explain part of the association.

From a clinical perspective, the findings suggest that patients with severe OSA, particularly those with marked nocturnal desaturation or additional glaucoma risk factors, may benefit from awareness of ocular symptoms and appropriate ophthalmic evaluation. Conversely, ophthalmologists encountering patients with unexplained or high-risk optic neuropathy and symptoms suggestive of sleep-disordered breathing may consider referral for sleep assessment.

Future studies should use larger multicentre cohorts, include age- and sex-matched controls, assess nocturnal hypoxemia quantitatively and incorporate longitudinal follow-up. Adjustment for hypertension, diabetes, dyslipidemia, obesity, smoking, family history and ocular parameters will be important for determining whether OSA independently predicts glaucoma.

6. Limitations

- Single-centre hospital-based design limits generalizability.
- Cross-sectional design cannot establish causality or determine whether OSA accelerates glaucoma progression.
- The cohort of 100 limits precision for glaucoma subgroup analyses.
- Age, obesity, hypertension and other vascular risk factors may confound the observed association.
- Single-night polysomnography may not represent night-to-night variability in OSA severity.
- OCT measurements are influenced by image quality, segmentation and anatomical factors.

7. Conclusion

In this hospital-based cohort, increasing OSA severity was associated with a higher prevalence of glaucoma, higher IOP and lower RNFL thickness. Greater AHI and nocturnal oxygen desaturation were also associated with adverse ophthalmic parameters. These findings support further investigation of the lung–sleep–optic nerve relationship and suggest that severe OSA may represent an important population for targeted ophthalmic assessment. Confirmation requires prospective studies using verified patient-level data and appropriate adjustment for vascular and ocular confounders.

8. Clinical Implications

- Severe OSA and significant nocturnal hypoxemia may warrant greater awareness of potential ocular comorbidity.
- IOP alone should not be used to diagnose or exclude glaucoma; structural and functional optic nerve assessment is essential.
- OCT RNFL and visual-field testing can complement conventional ophthalmic evaluation in research studies.
- Future interdisciplinary protocols should include standardized sleep testing and standardized glaucoma definitions.

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