

Research Article

Obstructive Sleep Apnoea and Glaucoma Connections: A Cross Sectional Study

Dr. Kali Shankar Das¹, Dr. Purban Ganguly², Dr. Sutanaya Saha^{3*}

¹Associate professor, Department of Ophthalmology, Regional institute of ophthalmology, Kolkata.

²Associate professor, Department of Ophthalmology, Jagannath Gupta institute of medical sciences and hospital.

^{3*}Senior Resident, Department of Ophthalmology, Regional institute of ophthalmology, kolkata.

Corresponding Author: Dr. Sutanaya Saha

Email: sutanaya.saha@gmail.com

Received: 28.08.26, Revised: 30.09.26, Accepted: 02.10.26

ABSTRACT

Introduction: Obstructive sleep apnoea (OSA) has been identified as a possible risk factor for glaucoma. Due to airway obstruction, there is decreased optic nerve perfusion pressure. This may lead to varied ophthalmic manifestations including glaucoma.

Aims: To find out positive co-relation between glaucoma parameters and OSA. **Methods:** 53 diagnosed OSA patients underwent comprehensive ophthalmic evaluation. The data was compared with an age-sex matched control.

Results: The study included 53 participants with OSA and 53 controls, with comparable mean age (52.4 ± 9.6 vs. 51.9 ± 8.8 years; $p = 0.74$) and male sex distribution (77.4% vs. 75.5%; $p = 0.81$). Among OSA participants, 34 (64.1%) had mild, 13 (24.5%) moderate, and 6 (11.4%) severe OSA ($p < 0.001$). Visual field defects were more frequent in OSA eyes (30 [28.3%] vs. 12 [11.3%]; $p = 0.002$). Mean IOP was higher in the OSA group (19.75 ± 3.53 vs. 17.2 ± 2.8 mmHg; $p = 0.01$), while mean RNFL thickness was lower (92.1 ± 8.89 vs. 95.6 ± 7.4 μ m; $p = 0.10$). RNFL thinning (34.0% vs. 13.2%; $p < 0.001$) and diffuse NRR thinning (22.6% vs. 5.7%; $p < 0.001$) were significantly more common in OSA. Hypertension, dyslipidemia, and diabetes were also significantly more prevalent in OSA (52.8% vs. 28.3%, $p = 0.01$; 41.5% vs. 18.9%, $p = 0.01$; and 30.2% vs. 13.2%, $p = 0.03$, respectively).

Conclusion: OSA was significantly associated with raised IOP, structural optic nerve abnormalities, and visual field defects, suggesting increased ocular vulnerability among affected individuals. The higher prevalence of hypertension, dyslipidemia, and diabetes further highlights the systemic burden accompanying OSA. Routine ophthalmic assessment, including IOP measurement, RNFL evaluation, optic nerve examination, and visual field testing, may therefore be beneficial in patients with OSA.

Keywords: Obstructive Sleep Apnoea (Osa), Glaucoma, Intraocular Pressure (Iop).

INTRODUCTION

Obstructive sleep apnoea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxia, sleep fragmentation, and marked fluctuations in intrathoracic pressure. It affects approximately 9–38% of the adult population, with higher prevalence among middle-aged and elderly individuals, males, and those with obesity and metabolic comorbidities [1]. OSA is increasingly recognized as a systemic disorder with multisystem consequences, including cardiovascular, cerebrovascular, metabolic, and neurocognitive complications. In recent years, growing attention has been directed toward its potential impact on ocular health, particularly its association with glaucomatous optic neuropathy.

Glaucoma is a progressive optic neuropathy characterized by structural damage to the optic nerve head and retinal nerve fiber layer (RNFL), accompanied by corresponding functional visual field loss. It is one of the leading causes of irreversible blindness worldwide. Elevated intraocular pressure (IOP) remains the most important modifiable risk factor; however, a significant proportion of patients develop glaucoma despite having normal IOP, highlighting the importance of non-pressure-dependent mechanisms such as vascular dysregulation, impaired autoregulation, and ischemia [2]. These mechanisms provide a plausible biological link between OSA and glaucoma.

Several pathophysiological processes in OSA may contribute to glaucomatous damage. Recurrent nocturnal hypoxia and reoxygenation lead to oxidative stress, endothelial

dysfunction, and systemic inflammation, all of which can adversely affect ocular perfusion [3]. In addition, repeated apnoeic episodes cause fluctuations in blood pressure and reductions in ocular perfusion pressure, potentially compromising blood flow to the optic nerve head. Increased sympathetic activity and altered nitric oxide metabolism further impair vascular autoregulation, making the optic nerve more vulnerable to ischemic injury [4]. These mechanisms are particularly relevant in normal-tension glaucoma, where vascular factors play a dominant role.

Beyond vascular effects, mechanical factors may also link OSA to glaucomatous damage. Episodes of hypoxia-induced cerebral vasodilation and changes in intrathoracic pressure can lead to transient increases in intracranial pressure, potentially altering the translaminal pressure gradient across the lamina cribrosa [5]. An imbalance between intraocular pressure and intracranial pressure may contribute to deformation of the lamina cribrosa and axonal damage, thereby accelerating glaucomatous optic neuropathy. Additionally, supine sleep posture and nocturnal rostral fluid shifts in OSA patients may cause transient elevations in IOP during sleep, further stressing the optic nerve [6].

Advances in ocular imaging have enabled detailed evaluation of glaucoma-related structural parameters. Optical coherence tomography (OCT) provides high-resolution, quantitative assessment of RNFL thickness, ganglion cell–inner plexiform layer (GCIPL), and optic nerve head morphology. Structural damage detected by OCT often precedes detectable visual field loss, making it a valuable tool for early diagnosis and monitoring of glaucoma [7]. Several studies have reported reduced RNFL and GCIPL thickness in patients with OSA, even in the absence of clinically diagnosed glaucoma, suggesting subclinical optic nerve damage related to sleep-disordered breathing [8].

Functional assessment of glaucoma relies primarily on standard automated perimetry, which detects characteristic patterns of visual field loss corresponding to structural damage. In OSA patients, visual field defects such as generalized depression, arcuate scotomas, and nasal steps have been described, raising concern that OSA may contribute not only to structural optic nerve changes but also to functional impairment [9]. The relationship between OSA severity, measured by the apnoea–hypopnoea index, and the extent of

visual field loss remains an area of active investigation, with some studies demonstrating a dose–response relationship.

Despite increasing evidence linking OSA and glaucoma, the association remains incompletely understood and at times controversial. Variability in study design, diagnostic criteria for OSA and glaucoma, and differences in population characteristics have contributed to inconsistent findings across studies. Furthermore, many patients with OSA remain undiagnosed, and ocular manifestations may be overlooked in routine clinical practice. Understanding the relationship between OSA and glaucoma-related structural and functional parameters is therefore of significant clinical importance, as early identification and management of OSA may represent a modifiable risk factor in the prevention or progression of glaucomatous damage [10].

To find out positive co-relation between glaucoma parameters and OSA. Methods: 53 diagnosed OSA patients underwent comprehensive ophthalmic evaluation. The data was compared with an age-sex matched control.

MATERIALS AND METHODS

Study Design: Hospital-based cross-sectional observational study.

Study Setting and Duration: 1 year

Sample Size: 53 patients were included in this study.

Study Population: Adult patients (≥ 18 years) diagnosed with obstructive sleep apnoea (OSA).

Inclusion Criteria: Patients with OSA confirmed by polysomnography (AHI ≥ 5 events/hour).

Exclusion Criteria: Known glaucoma, previous ocular surgery, secondary glaucoma, retinal or neurological diseases affecting vision.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS software version 27.0 (SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as frequencies and percentages. The unpaired t-test was used to compare continuous variables between independent groups, and the paired t-test was applied for within-group comparisons. Categorical variables were analyzed using the Chi-square test or Fisher's exact test as appropriate. A p-value of <0.05 was considered statistically significant

RESULT

Table 1. Demographic Characteristics of the Study Population

Parameter	OSA Group (n = 53)	Control Group (n = 53)	p-value
Mean age (years)	52.4 ± 9.6	51.9 ± 8.8	0.74
Male sex, n (%)	41 (77.4%)	40 (75.5%)	0.81

Table 2. Distribution of OSA Severity

OSA Severity	OSA Group, n (%) (n = 53)	Control Group, n (%) (n = 53)	p-value
Mild OSA	34 (64.1%)	0 (0.0%)	<0.001
Moderate OSA	13 (24.5%)	0 (0.0%)	
Severe OSA	6 (11.4%)	0 (0.0%)	
Total	53 (100%)	53 (100%)	

Table 3. Comparison of Visual Field Defects between OSA and Control Groups (Eye-Level Analysis)

Parameter	OSA Group (n = 106 eyes)	Control Group (n = 106 eyes)	p-value
Visual field defects, n (%)	30 (28.3%)	12 (11.3%)	0.002

Table 4. Comparison of Mean RNFL Thickness and IOP between OSA and Control Groups

Parameter	OSA Group (n = 53)	Control Group (n = 53)	p-value
Mean RNFL thickness (µm)	92.1 ± 8.89	95.6 ± 7.4	0.1
Mean IOP (mmHg)	19.75 ± 3.53	17.2 ± 2.8	0.01

Table 5. Comparison of RNFL and Neuroretinal Rim Parameters between OSA and Control Groups

Parameter	OSA Group, n (%) (n = 106 eyes)	Control Group, n (%) (n = 106 eyes)	p-value
RNFL thinning	36 (34.0%)	14 (13.2%)	<0.001
Focal NRR thinning	28 (26.4%)	20 (18.9%)	0.189
Diffuse NRR thinning	24 (22.6%)	6 (5.7%)	<0.001

Table 6. Comparison of Ocular Findings between OSA and Control Groups

Ocular Parameter	OSA Group, n (%) (n = 106 eyes)	Control Group, n (%) (n = 106 eyes)	p-value
Raised IOP (>21 mmHg)	32 (30.2%)	12 (11.3%)	0.001
RNFL thinning	36 (34.0%)	14 (13.2%)	<0.001
Focal NRR thinning	28 (26.4%)	20 (18.9%)	0.189
Diffuse NRR thinning	24 (22.6%)	6 (5.7%)	<0.001
Visual field defects	30 (28.3%)	12 (11.3%)	0.002

Table 7. Comparison of Systemic Comorbidities between OSA and Control Groups

Systemic Comorbidity	OSA Group n (%) (n = 53)	Control Group n (%) (n = 53)	p-value
Hypertension	28 (52.8%)	15 (28.3%)	0.01
Dyslipidemia	22 (41.5%)	10 (18.9%)	0.01
Diabetes mellitus	16 (30.2%)	7 (13.2%)	0.03
Asthma	10 (18.9%)	4 (7.5%)	0.09

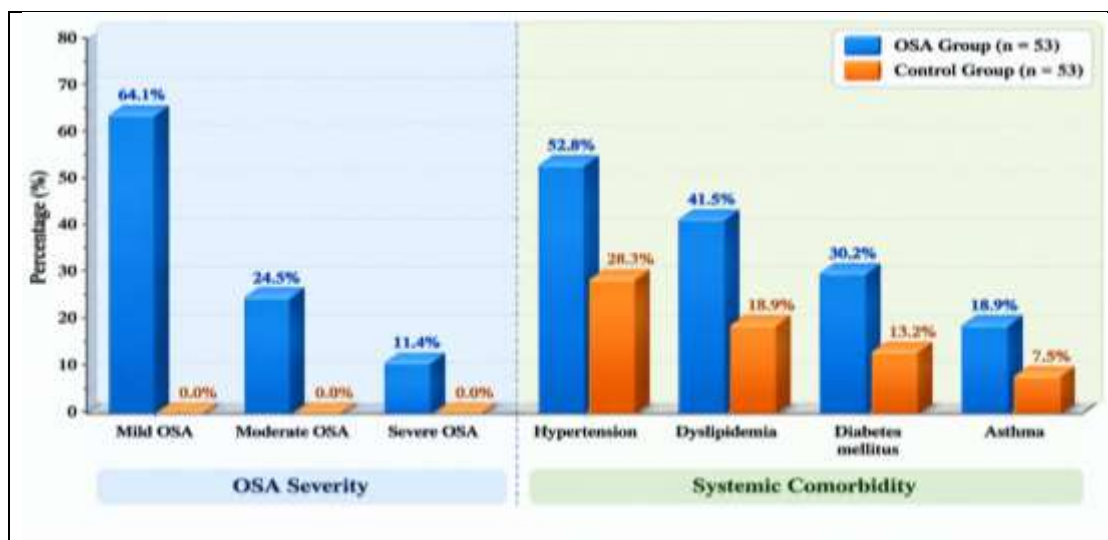


Figure: 1. Comparison of OSA Severity and Systemic Comorbidities between the OSA and Control Groups



Figure: 2. Comparison of Ocular Findings between the OSA and Control Groups

The demographic characteristics of the study population are presented in Table 1. The mean age was comparable between the OSA and control groups, being 52.4 ± 9.6 years in the OSA group and 51.9 ± 8.8 years in the control group. This difference was not statistically significant ($p = 0.74$), indicating that the two groups were well matched with respect to age. Similarly, males constituted the majority of participants in both groups, with 41 (77.4%) males in the OSA group compared with 40 (75.5%) in the control group. The difference in sex distribution was also statistically non-significant ($p = 0.81$). Thus, there was no significant baseline difference between the OSA and control groups with regard to either age or sex.

Table 2 demonstrates the distribution of OSA severity among the study participants. Of the 53 participants in the OSA group, 34 (64.1%) had mild OSA, 13 (24.5%) had moderate OSA, and 6 (11.4%) had severe OSA. Thus, mild OSA represented the largest proportion of affected participants, followed by moderate and severe OSA. As expected, none of the 53 participants (0.0%) in the control group had mild, moderate, or severe OSA. The overall difference in OSA severity distribution between the two groups was highly statistically significant ($p < 0.001$). The findings confirm that the OSA group consisted of participants across the spectrum of OSA severity, whereas the control group did not have OSA.

Table 3 compares the frequency of visual field defects between the OSA and control groups at the level of individual eyes, with each group contributing 106 eyes. Visual field defects were observed in 30 eyes (28.3%) in the OSA group compared with 12 eyes (11.3%) in the control group, representing a statistically significant difference ($p = 0.002$). Mean RNFL thickness and mean IOP are reported at the participant level in Table 4, so that both eyes of the same individual are not counted as independent observations; the corresponding results are described below.

Table 4 presents the comparison of mean RNFL thickness and IOP between the two groups, with 53 participants in each group. Mean RNFL thickness was $92.1 \pm 8.89 \mu\text{m}$ in the OSA group and $95.6 \pm 7.4 \mu\text{m}$ in the control group. Although the mean RNFL thickness was numerically lower in the OSA group, the difference was reported as statistically non-significant ($p = 0.10$). Mean IOP, however, was higher in the OSA group ($19.75 \pm 3.53 \text{ mmHg}$) compared with the control group ($17.2 \pm 2.8 \text{ mmHg}$), and this difference was statistically significant ($p = 0.01$). Therefore, the findings indicate a significant difference in IOP between the groups, while the difference in mean RNFL thickness did not reach statistical significance according to the values reported in this table.

Table 5 compares structural optic nerve parameters between the OSA and control groups, based on 106 eyes in each group. RNFL thinning was observed in 36 eyes (34.0%) in the OSA group compared with 14 eyes (13.2%) in the control group, and this difference was highly statistically significant ($p < 0.001$). Focal neuroretinal rim (NRR) thinning was present in 28 eyes (26.4%) in the OSA group and 20 eyes (18.9%) in the control group; however, this difference was not statistically significant ($p = 0.189$). Diffuse NRR thinning was observed considerably more frequently in the OSA group, affecting 24 eyes (22.6%), compared with 6 eyes (5.7%) in the control group, with a highly significant difference ($p < 0.001$). These findings indicate that RNFL thinning and diffuse NRR thinning were significantly more frequent among eyes in the OSA group, whereas focal NRR thinning did not differ significantly between the two groups.

The comparative ocular findings are summarized in Table 6. Raised IOP ($>21 \text{ mmHg}$) was detected in 32 eyes (30.2%) in the OSA group compared with 12 eyes (11.3%) in the control group, and the difference was statistically significant ($p = 0.001$). RNFL

thinning was found in 36 eyes (34.0%) in the OSA group and 14 eyes (13.2%) in the control group, demonstrating a highly significant difference ($p < 0.001$). Focal NRR thinning was present in 28 eyes (26.4%) in the OSA group versus 20 eyes (18.9%) in the control group, although the difference was not statistically significant ($p = 0.189$). Diffuse NRR thinning occurred in 24 eyes (22.6%) in the OSA group compared with only 6 eyes (5.7%) in the control group, with a highly significant difference ($p < 0.001$). Similarly, visual field defects were observed in 30 eyes (28.3%) in the OSA group compared with 12 eyes (11.3%) in the control group ($p = 0.002$). Overall, raised IOP, RNFL thinning, diffuse NRR thinning, and visual field defects were significantly more frequent in the OSA group, whereas focal NRR thinning showed no statistically significant difference.

Table 7 compares the prevalence of selected systemic comorbidities between the OSA and control groups, with 53 participants in each group. Hypertension was present in 28 participants (52.8%) in the OSA group compared with 15 (28.3%) in the control group, representing a statistically significant difference ($p = 0.01$). Dyslipidemia was also more frequent among participants with OSA, being observed in 22 (41.5%) compared with 10 (18.9%) in the control group ($p = 0.01$). Diabetes mellitus was reported in 16 participants (30.2%) in the OSA group versus 7 (13.2%) in the control group, and this difference was statistically significant ($p = 0.03$). Asthma was present in 10 participants (18.9%) in the OSA group and 4 (7.5%) in the control group; however, this difference did not reach statistical significance ($p = 0.09$). Thus, hypertension, dyslipidemia, and diabetes mellitus were significantly more prevalent in the OSA group, whereas the observed difference in asthma prevalence was not statistically significant.

DISCUSSION

The present study demonstrated that the OSA and control groups were broadly comparable in terms of baseline demographic characteristics. The mean age was 52.4 ± 9.6 years in the OSA group and 51.9 ± 8.8 years in the control group ($p = 0.74$), while males constituted 77.4% and 75.5% of the respective groups ($p = 0.81$). The absence of significant differences suggests that age and sex were unlikely to account for the observed ocular differences. This comparability is important because age and sex can influence

both the prevalence of OSA and the development of optic nerve disease. A previous cross-sectional study of OSA patients also reported a predominantly male population, although demographic differences between OSA and non-OSA participants were more pronounced in that cohort. [11] The close matching of the present groups therefore strengthens the interpretation of the subsequent ocular comparisons.

In the present study, 64.1% of participants with OSA had mild disease, 24.5% had moderate OSA, and 11.4% had severe OSA, while none of the controls had OSA ($p < 0.001$). The predominance of mild OSA in the present cohort differs from some ophthalmic studies that have recruited predominantly moderate-to-severe OSA cases. For example, Bendel et al. investigated 100 patients with moderate-to-severe OSA and reported glaucoma in 27% of participants. [12] More recent evidence suggests that the ocular consequences of OSA may vary according to disease severity and the presence of accompanying vascular risk factors. A large systematic review and meta-analysis found an overall increased risk of glaucoma in patients with OSA, with adjusted analyses continuing to demonstrate an association after accounting for important confounders. [13] Thus, although the present study contained a greater proportion of mild cases, the occurrence of ocular abnormalities across the OSA cohort remains consistent with the broader literature.

A major finding of the present study was the significantly greater frequency of visual field defects, which were detected in 30 eyes (28.3%) in the OSA group compared with 12 eyes (11.3%) in controls ($p = 0.002$). This finding supports the concept that OSA may be associated with functional visual pathway or optic nerve abnormalities. Earlier work by Mojon and colleagues described visual field abnormalities in patients with sleep apnoea, providing early clinical evidence that nocturnal respiratory disturbance may be associated with visual field changes. [14] The higher frequency of visual field defects in the present OSA group may therefore reflect the combined effects of intermittent hypoxia, vascular dysregulation, and altered nocturnal ocular perfusion. Nevertheless, visual field defects are not specific for glaucoma, and this finding should be interpreted together with the structural parameters.

The present study also showed a numerically lower mean RNFL thickness in the OSA group

($92.1 \pm 8.89 \mu\text{m}$) compared with controls ($95.6 \pm 7.4 \mu\text{m}$); however, at the participant level (Table 4), this difference did not reach statistical significance ($p = 0.10$). Although the direction of the finding was consistent with a detrimental effect of OSA on RNFL thickness, the categorical measure of RNFL thinning (34.0% vs. 13.2%; $p < 0.001$) was a more sensitive indicator of structural change in this cohort than the continuous mean RNFL thickness comparison. Previous OCT-based research has similarly suggested that OSA may be associated with reduced retinal nerve fiber layer thickness. [8] The biological explanation may involve repeated nocturnal hypoxemia, oxidative stress, impaired autoregulation, and reduced perfusion of the optic nerve head, all of which could increase susceptibility to retinal ganglion-cell axonal injury.

Mean IOP was consistently higher in the OSA group, measuring $19.75 \pm 3.53 \text{ mmHg}$, compared with $17.2 \pm 2.8 \text{ mmHg}$ in controls. This difference was statistically significant at the participant level ($p = 0.01$; Table 4). This finding is compatible with studies suggesting that OSA can influence nocturnal IOP and ocular perfusion. Liu et al. demonstrated nocturnal elevation of IOP in patients with OSA, supporting the possibility that repeated nighttime physiological changes may influence ocular pressure. [15] In another study, patients with OSA had higher IOP in at least one eye compared with individuals without OSA, although the relationship between OSA and glaucoma itself was not always statistically significant. [11] Conversely, some investigations have failed to demonstrate a consistent association between OSA severity and IOP, indicating that the relationship is probably multifactorial rather than simply dependent on apnoea severity. [12] The significant increase in mean IOP in the present study nevertheless provides an additional potential mechanism through which OSA could contribute to optic nerve vulnerability.

The frequency of RNFL thinning provides further structural support for the ocular findings. In the present study, RNFL thinning was observed in 36 eyes (34.0%) in the OSA group compared with 14 eyes (13.2%) in controls ($p < 0.001$). This difference is substantial and agrees with previous OCT studies reporting thinner RNFL measurements among individuals with OSA. [8] The finding is also consistent with the broader systematic-review evidence demonstrating an association between OSA and ocular structural changes

characteristic of glaucomatous disease. Intermittent hypoxia may cause repeated episodes of oxidative and vascular stress, while nocturnal changes in blood pressure and ocular perfusion could further compromise the optic nerve head. These mechanisms could explain why structural RNFL abnormalities were considerably more frequent in the OSA group. With regard to the neuroretinal rim, focal NRR thinning occurred in 28 eyes (26.4%) in the OSA group and 20 eyes (18.9%) in controls, but the difference was not statistically significant ($p = 0.189$). This result suggests that focal rim changes alone may not reliably distinguish OSA-associated ocular abnormalities from those occurring in controls. Interestingly, a Colombian cross-sectional study examining OSA and optic nerve findings reported focal or diffuse neuroretinal rim thinning in a proportion of eyes, but the distribution varied according to OSA severity. [16] In contrast, diffuse NRR thinning in the present study was significantly more frequent in the OSA group, occurring in 24 eyes (22.6%) compared with 6 eyes (5.7%) in controls ($p < 0.001$). The greater frequency of diffuse rather than focal changes may indicate a more generalized susceptibility of the optic nerve to chronic hypoxic or vascular stress in OSA. However, these findings should not by themselves be interpreted as diagnostic evidence of glaucoma.

Raised IOP (>21 mmHg) was also significantly more common in the OSA group, occurring in 32 eyes (30.2%) compared with 12 eyes (11.3%) in controls ($p = 0.001$). This finding complements the significantly higher mean IOP observed in the OSA group. Previous studies have produced mixed results regarding the relationship between OSA and IOP, but evidence of nocturnal IOP elevation provides a plausible physiological link. [15] The coexistence of raised IOP, RNFL thinning, diffuse NRR thinning, and visual field defects in the present OSA group is particularly noteworthy because these parameters represent different components of glaucomatous assessment—pressure-related risk, structural damage, and functional impairment. A systematic review of OSA and glaucoma likewise concluded that OSA was associated with a higher risk of glaucoma and more severe glaucoma-related ocular findings, although substantial heterogeneity between studies was noted. [10]

The systemic findings further support the recognized association between OSA and cardiometabolic disease. Hypertension was

present in 28 participants (52.8%) in the OSA group compared with 15 (28.3%) in controls ($p = 0.01$). This observation is consistent with evidence from a large meta-analysis demonstrating a significant association between OSA and hypertension. [17] The mechanism is thought to involve recurrent intermittent hypoxia, sympathetic activation, endothelial dysfunction, and altered autonomic regulation. The prevalence in the present study was lower than that reported in some OSA cohorts, but the direction of association was similar.

Similarly, dyslipidemia was significantly more common in the OSA group, affecting 22 participants (41.5%) compared with 10 (18.9%) controls ($p = 0.01$). These findings are in agreement with a large meta-regression analysis showing that OSA is associated with an adverse lipid profile, including higher total cholesterol, LDL cholesterol, and triglycerides and lower HDL cholesterol. [18] Intermittent hypoxia, sleep fragmentation, sympathetic activation, insulin resistance, and obesity-related metabolic dysfunction may all contribute to this association. The present results therefore reinforce the importance of assessing cardiovascular and metabolic risk factors in individuals with OSA.

Diabetes mellitus was also significantly more frequent in the OSA group, occurring in 16 participants (30.2%) compared with 7 (13.2%) in controls ($p = 0.03$). This finding is consistent with contemporary epidemiological evidence showing increased cardiometabolic comorbidity among individuals with OSA. A large population-based study found significantly increased risks of diabetes, dyslipidemia, and hypertension among patients with OSA, even after adjustment for age and socioeconomic factors. [19] More recent clinical data similarly demonstrate a substantial burden of diabetes and other metabolic diseases among patients with OSA. [20] The present association may reflect shared mechanisms such as obesity, insulin resistance, chronic intermittent hypoxia, and systemic inflammation.

In contrast, asthma was present in 10 participants (18.9%) in the OSA group compared with 4 (7.5%) in controls, but the difference was not statistically significant ($p = 0.09$). Although the observed proportion was higher in the OSA group, the sample size may have limited the ability to demonstrate a statistically significant difference. Contemporary large-scale data indicate that asthma and other respiratory conditions can

coexist with OSA, but the strength of association varies across populations and may be influenced by age, obesity, smoking, and other comorbid conditions. [20] Therefore, the present finding suggests a possible trend toward greater asthma prevalence but does not provide sufficient evidence for a significant association in this cohort.

Overall, the findings of the present study indicate that OSA was associated with a greater burden of several ocular abnormalities, particularly raised IOP, RNFL thinning, diffuse NRR thinning, and visual field defects, together with a higher prevalence of hypertension, dyslipidemia, and diabetes mellitus. These findings are broadly consistent with the growing literature linking OSA with both ocular and systemic vascular/metabolic abnormalities. However, the results should be interpreted as associations rather than proof of causation. Differences in OSA severity, diagnostic criteria, sample size, comorbidities, and adjustment for confounding factors may explain some of the variability between individual studies. Longitudinal studies incorporating detailed polysomnographic parameters, nocturnal oxygen saturation, OCT progression, visual-field progression, and treatment response would be useful to clarify whether effective treatment of OSA can prevent or slow ocular structural and functional deterioration.

CONCLUSION

The present study demonstrates a significant association between obstructive sleep apnoea (OSA) and ocular abnormalities, particularly raised intraocular pressure, retinal nerve fiber layer (RNFL) thinning, diffuse neuroretinal rim thinning, and visual field defects. Compared with controls, participants with OSA had higher mean IOP, greater frequencies of raised IOP, RNFL thinning, diffuse NRR thinning, and visual field abnormalities, suggesting increased optic nerve vulnerability in OSA. Although focal NRR thinning did not differ significantly between groups, the overall structural and functional findings support a potential relationship between OSA and glaucomatous optic nerve changes. OSA participants also showed significantly higher frequencies of hypertension, dyslipidemia, and diabetes mellitus, highlighting the substantial systemic cardiometabolic burden associated with the condition. Age and sex distributions were comparable between groups, reducing the likelihood that these demographic factors explained the observed differences. Overall, the findings emphasize the importance of

recognizing ocular involvement in OSA and support regular ophthalmic evaluation, particularly assessment of IOP, RNFL, optic nerve morphology, and visual fields, in affected individuals.

REFERENCES

1. Senaratna CV, et al. Prevalence of obstructive sleep apnea in the general population. *Sleep Med Rev.* 2017; 34:70-81.
2. Weinreb RN, et al. The pathophysiology and treatment of glaucoma. *JAMA.* 2014;311(18):1901-11.
3. Lavie L. Oxidative stress in obstructive sleep apnea and intermittent hypoxia. *Free Radic Biol Med.* 2015;86:1-11.
4. Kargi SH, et al. Ocular blood flow and glaucoma in obstructive sleep apnea syndrome. *J Glaucoma.* 2005;14(6):461-5.
5. Berdahl JP, et al. Intracranial pressure and glaucoma. *Curr Opin Ophthalmol.* 2012;23(2):106-11.
6. Liu JHK, et al. Nocturnal elevation of intraocular pressure in obstructive sleep apnea. *Invest Ophthalmol Vis Sci.* 2011;52(6):4226-31.
7. Leung CK, et al. Retinal nerve fiber layer imaging with optical coherence tomography. *Ophthalmology.* 2010;117(12):2337-44.
8. Lin PW, Friedman M, Lin HC, Chang HW, Wilson MT, Lin MC. Decreased retinal nerve fiber layer thickness in patients with obstructive sleep apnea. *Graefes Arch Clin Exp Ophthalmol.* 2011;249(9):1285-1290.
9. Mojon DS, et al. Visual field defects in sleep apnea syndrome. *Ophthalmology.* 1999;106(5):1009-13.
10. Shi Y, et al. Association between obstructive sleep apnea and glaucoma: a meta-analysis. *PLoS One.* 2015;10(2):e0115625.
11. Goyal JL, Khanduri R, Sinha R, et al. Prevalence of glaucoma in patients with obstructive sleep apnea. *J Glaucoma.* 2020;29(7):535-540.
12. Bendel RE, Kaplan J, Heckman M, Fredrickson PA, Lin SC. Prevalence of glaucoma in patients with obstructive sleep apnoea. *Eye (Lond).* 2008;22(9):1105-1109.
13. Sun C, et al. Obstructive sleep apnoea and glaucoma: a systematic review and meta-analysis. *Eye (Lond).* 2023;37:2475-2485.

14. Mojon DS, Mathis J, Zulauf M, Koerner F, Hess T, Jaggi C, et al. Optic neuropathy associated with sleep apnea syndrome. *Ophthalmology*. 1998;105(5):874-877.
15. Liu JHK, Weinreb RN, Zhang X, Friedmann DS. Nocturnal elevation of intraocular pressure in obstructive sleep apnea. *Invest Ophthalmol Vis Sci*. 2011;52(6):4226-4231.
16. Caramori M, et al. Primary open-angle glaucoma in patients with obstructive sleep apnoea in a Colombian population: a cross-sectional study. *BMJ Open Ophthalmol*. 2023;8:e001347.
17. Hou H, Zhao Y, Yu W, et al. Association of obstructive sleep apnea with hypertension: a systematic review and meta-analysis. *J Glob Health*. 2018;8(1):010405.
18. Nadeem R, Singh M, Nida M, et al. Effect of obstructive sleep apnea hypopnea syndrome on lipid profile: a meta-regression analysis. *J Clin Sleep Med*. 2014;10(5):475-489.
19. Seo MY, et al. Obstructive sleep apnea and medical comorbidities in the Asian population: evidence from a nationwide healthcare system data in South Korea. *Sci Rep*. 2024;14:21406.
20. Tao Y, Dong X, Tu H, Fang X, Shao Y. Symptom and comorbidity burden in hypertensive patients with obstructive sleep apnea. *Front Endocrinol (Lausanne)*. 2024;15:1361466.