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ORIGINAL RESEARCH

Corneal Topographic Changes and Intraocular Pressure in Different Stages of Obstructive Sleep Apnea: Case–Control Study

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Purpose: To evaluate corneal topographic parameters and intraocular pressure (IOP) in patients with different stages of obstructive sleep apnea (OSA) compared healthy controls, and to explore correlations between ocular findings and polysomnographic parameters.

Patients and Methods: This case–control study included 55 patients diagnosed with obstructive sleep apnea (OSA) and 22 healthy controls. All participants were recruited immediately after diagnosis and prior to the initiation of any treatment. OSA diagnosis was confirmed by full-night diagnostic polysomnography. Control subjects were screened clinically and underwent polysomnography to confirm the absence of OSA. Ophthalmic assessments were performed within the same week of polysomnography assessment and included measurement of intraocular pressure using Goldmann applanation tonometry and anterior segment imaging using Scheimpflug-based corneal tomography. Corneal pachymetry, anterior chamber parameters, keratometric indices, and ectasia-related indices were recorded and analyzed.

Results: No statistically significant differences were observed among groups regarding posterior corneal pachymetric parameters. Anterior chamber depth was significantly reduced in mild and severe OSA groups, and anterior chamber volume was lowest in the mild OSA group. Intraocular pressure was significantly higher in moderate and severe OSA groups compared with controls. Significant correlations were observed between arousal index and several corneal parameters, including pachymetry and progression indices.

Conclusion: Moderate and severe OSA were associated with increased intraocular pressure and changes in anterior chamber configuration. Arousal index showed significant correlations with corneal pachymetric and topographic parameters, suggesting a possible association between sleep fragmentation and corneal structural changes. Further studies of larger sample sizes are recommended to confirm these findings.

Keywords: corneal topography, intraocular pressure, obstructive sleep apnea, polysomnography

Introduction

Obstructive sleep apnea syndrome (OSAS) is a prevalent disease among Egyptians, with a reported total prevalence rate of 14% in previous studies.[1]

It mostly manifests with nocturnal symptoms, such as snoring, choking, or gasping during sleep, along with daytime symptoms including excessive sleepiness, fatigue, and cognitive changes.[2,3] It is defined as recurrent episodes of partial or complete airway obstruction during sleep, resulting in repetitive apneas and hypopneas. This condition is diagnosed when the apnea-hypopnea index (AHI)- defined as the number of apneas and hypopneas per hour of sleep is ≥5.[4]

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It is well proven that OSA is closely associated with a spectrum of systemic complications, including diabetes mellitus, obesity, hypertension, stroke, cardiovascular diseases, metabolic syndrome, and increased incidence of early death.[5,6]

Recently, increasing attention has been directed toward the ocular sequelae of OSA, including corneal ecstatic changes, glaucoma, dry eye, floppy eyelid syndrome, ischemic optic neuropathy, and retinal vascular diseases. However, many studies included relatively small samples and have reported conflicting results.[5]

Visual function mainly depends on the cornea, which is avascular and transparent structure, that relies mainly on oxygen uptake from the atmosphere and, to a lesser extent, from the aqueous humor. OSA may affect both routes of the corneal oxygen supply. Increased ocular surface exposure associated with dry eye cornea (indirect route) and systemic hypoxia during sleep apnea may impair oxygen delivery through aqueous humor (direct route). Hypoxia may subsequently effect the cornea through several mechanisms, including disruption of the epithelial barrier, endothelial dysfunction, and metabolic changes in the stroma.[7,8]

We aimed to study corneal topographic parameters and intraocular pressure (IOP) in patients with different stages of OSA compared with normal subjects, and to correlate these changes with polysomnography parameters, particularly the respiratory arousal index, which reflects the frequency of sleep fragmentation events and may play an important role in the pathophysiology of ocular changes in patients with obstructive sleep apnea.

Patients and Methods

This case-control study included 55 patients who were recruited based on previously established diagnosis of obstructive sleep apnea (OSA) confirmed by polysomnography performed prior to enrollment.and before the initiation of any treatment modalities. All consecutive patients were diagnosed with OSA who presented to Otorhinolaryngology clinics during the study were included.

The study cases were compared with 22 healthy subjects who were initially screened through detailed medical history to exclude symptoms suggestive of OSA. All control participants subsequently underwent full night polysomnography to confirm the absence of OSA. The study was conducted from January 2024 to June 2024.

Written informed consent was obtained from all participants after explaining the planned ophthalmological examinations. The study protocol was approved by the Sohag University Local Ethics Committee (Soh-Med-23-09-7PD), and the study was registered under the clinical trial identifier (NCT06347900). The present research adhered to the ethical principles of the Helsinki Declaration.

Data collection included medical history, demographic data, height and weight measurements, body mass index (kg/m^2), neck circumference (NC), and confirmation of diagnosis by polysomnography. Full-night laboratory diagnostic polysomnography (SOMNO screen TM plus PSG + sleep monitor, SOMNO medics GmbH, Germany) was performed by a sleep technician.

Moreover, the sleep study included electroencephalography readings with two frontal leads, two central leads, and two occipital leads; right and left eye leads; nasal pressure; nasal-oral airflow (thermal device); snore sensor; respiratory effort (thoracic and abdominal); oxygen saturation measured using pulse oximetry; submental electromyography; as well as left and right anterior tibialis electromyography.

The polysomnography findings were manually scored in accordance with the American Academy of Sleep Medicine Manual, which defines apnea as a cessation of airflow by ≥90% for a minimum of 10 seconds and hypopnea as a decline of airflow by ≥30% for a minimum of 10 seconds accompanied by a decrease in oxygen saturation levels of ≥4%. The total arousal index (AI) was calculated as the total number of arousals per hour of sleep.[9,10]

The study included cases whose diagnostic criteria matched OSA based on the Third Edition of the International Classification of Sleep Disorders. The cases were then grouped into mild (AHI 5–15), moderate (AHI 15–30), and severe (AHI >30) groups.[11]

To minimize the effect of obesity as a confounding factor, only patients with a body mass index (BMI) ≤33.20 were included in the study.

Ophthalmic Evaluation

All ophthalmic examinations were performed within the same week of the polysomnography assessment. Every patient underwent a comprehensive ophthalmologic examination, including uncorrected visual acuity (UCVA), best-corrected visual acuity (BCVA) using a Snellen chart, slit-lamp biomicroscopic examination of the anterior segment, and fundus examination. The same ophthalmologist performed all examinations for each patient.

Intraocular pressure (IOP) was measured using Goldmann applanation tonometry between 8:00 and 10:00 a.m., and values were corrected according to central corneal thickness using standard correction tables[12,13] A mydriatic fundus examination was also performed.

The Scheimpflug method (Pentacam, OCULUS Optikgeräte GmbH, Wetzlar, Germany) was utilized to obtain measurements of the anterior segment and corneal topography.

Measurements were recorded using the non-contact method in a dark room. Patients were asked to blink naturally twice and then keep their eyes open, and corneal topography was performed within four to eight seconds after blinking to minimize the effect of tear-film irregularity. Images from three successive scans were documented.[14,15]

Comparisons were made between the right eyes of the cases regarding aqueous depth (AD), anterior chamber volume (AV), corneal volume (CV), thinnest point of the cornea, apical corneal thickness (ACT), corneal thickness at the pupillary area, front and back corneal keratometric values at 3 mm (Ant-K1, Ant-K2, Post-K1, Post-K2, Ant-Km, Post-Km), anterior and posterior astigmatism, anterior and posterior R-min, anterior and posterior Q value, front elevation, back elevation, minimum progression index (Min PI), maximum progression index (Max PI), average progression index (Avg PI), and Ambrósio relational thickness maximum (ARTmax).

Ambrósio relational thickness maximum (ARTmax) values were not available for all eyes in the mild and moderate OSA groups. Specifically, data were available in 8 eyes in the mild group and 10 eyes in the moderate group. Due to the limited number of available observations in each subgroup, the mild and moderate groups were combined for the analysis to improve the stability of the statistical analysis.

Inclusion Criteria

The included cases were previously diagnosed with OSA at the sleep clinic of Sohag University Hospital following overnight polysomnography.

The participants' right eyes were assessed in all groups to avoid inter-eye correlation and ensure statistical independence of observations: 20 eyes (control group), 16 eyes (mild group), 16 eyes (moderate group), and 23 eyes (severe group).

Exclusion Criteria

Patients with a history of ocular surgery, contact lens use within the last 4 weeks, glaucoma or use of anti-glaucoma medications, corneal opacities, or dense cataract preventing adequate ocular examination were excluded. Patients with refractive errors greater than +3.00 D hyperopia or −6.00 D myopia were also excluded.

Moreover, exclusion covered cases with cardiovascular disease, hypertension, diabetes mellitus, or any systemic diseases that could affect the biomechanics of the cornea, such as Sjögren's disease, liver diseases, chronic renal failure, chronic obstructive pulmonary disease (COPD), rheumatic disorders, and oncological diseases. Cases with central, complex, or mixed sleep apnea were also excluded.

Statistical Analysis

Data were statistically analyzed using SPSS version 26 (developed by IBM Inc., located in Illinois, the United States). Quantitative variables were given in the form of mean and standard deviation (SD) and analyzed between the groups using the ANOVA (F) test, followed by a post hoc analysis (Tukey). Additionally, qualitative variables were given in the form of frequencies and percentages (%) and analyzed by the Chi-square test. A correlation among many variables was conducted by the Pearson moment correlation formula for linear relationships of normally distributed variables.

Results

The study included 55 patients diagnosed with obstructive sleep apnea by otolaryngology and pulmonary specialists. The study cases were compared with 22 normal subjects without obstructive sleep apnea who served as a control group.

Patients in the severe OSA group were the oldest among all groups, and the majority were males. Uncorrected visual acuity (UCVA) and best-corrected visual acuity (BCVA) were better in control and mild OSA groups (Table 1).

Cylinder refractive error was higher in the severe and moderate OSA groups. Mean keratometry was higher in the severe OSA group compared with the mild OSA group (Table 2).

There were no statistically significant differences in posterior corneal pachymetric parameters among the studied groups (Table 3).

Anterior chamber volume was lowest in the mild OSA group. Anterior chamber depth was shallower in the mild and severe OSA groups compared with the control and moderate groups (Table 4).

The minimum progression index was highest in the moderate and severe OSA groups, while the maximum progression index was highest in the severe OSA groups. Corneal back elevation was also highest in the severe OSA group. Intraocular pressure was higher in the moderate and severe OSA groups. Ambrósio relational thickness maximum (ARTmax) showed higher values in the mild and moderate groups compared with the severe group, although the difference was not statistically significant (Table 5 and Table 6).

Obstructive apnea index, respiratory distress index, and desaturation index showed higher values in the severe OSA group, whereas the arousal index was highest in the moderate OSA group (Table 7).

Table 1 Demographic Data and Visual Acuity (UCVA and BCVA) Among the Control Subjects and Different Stages of Obstructive Sleep Apneas Syndrome (OSAS)

	Control Group (n=20)	Mild OSAS Group (n=16)	Moderate OSAS Group (n=16)	Severe OSAS Group. (n=23)	P value
Age (years)	33.6 ± 6.02	38 ± 4.1	32.3 ± 8.5	44 ± 7.11	<0.001*
	P1	0.504	0.975	<0.001*	
	P2		0.459	0.208	
	P3			0.002*	
Male sex	47.83%	17.39%	26.09%	91.3%	0.030*
	P1	0.964	0.117	0.042*	
	P2		0.467	0.529	
	P3			0.963	
UCVA (Log MAR)	1 ± 0	0.8 ± 0.15	0.7 ± 0.29	0.7 ± 0.2	<0.001*
	P1	0.16	<0.001*	<0.001*	
	P2		0.323	0.973	
	P3			0.325	
BCVA (Log MAR)	1 ± 0	0.9 ± 0.1	0.8 ± 0.18	0.8 ± 0.18	<0.001*
	P1	0.159	0.006*	<0.001*	
	P2		0.704	0.999	
	P3			0.611	

Notes: *Significant as p value ≤0.05, P1: P value compared to control group, P2: P value compared to mild group, P3: P value compared to moderate group.
Abbreviations: UCVA, Uncorrected Visual Acuity; BCVA, Best corrected visual acuity; OSAS, Obstructive sleep apnea syndrome.

Table 2 Comparison of Refractive Errors and Anterior Corneal Topographic Parameters Between Control Subjects and Patients with Different Stages of Obstructive Sleep Apnea Syndrome (OSAS)

	Control Group (n=20)	Mild OSAS Group (n=16)	Moderate OSAS Group (n=16)	Severe OSAS group (n=23)	P value
Spherical error (D)	0.1 ± 0.17	0.5 ± 0.98	0.1 ± 0.88	−0.1 ± 1.28	0.669
Cylindrical error (D)	0 ± 0.06	−0.5 ± 0.37	−0.8 ± 0.41	−0.7 ± 0.66	<0.001*
	P1	0.14	0.002*	<0.001*	
	P2		0.50	0.95	
	P3			0.62	
K1 (D)	43 ± 1.33	42.9 ± 1.46	42.2 ± 1.46	42.9 ± 1.51	0.723
K2 (D)	44.5 ± 1.41	44 ± 1.5	42.4 ± 1.45	43.8 ± 1.83	0.073
K max (D)	43.7 ± 1.29	42.2 ± 1.63	42.3± 1.67	43.3 ± 1.59	0.049*
	P1	1	1	1	
	P2		0.132	0.043*	
	P3			1	
Corneal astigmatism (D)	1.5 ± 0.88	0.7 ± 0.29	0.1 ± 1.55	1 ± 0.97	0.023*
	P1	0.294	0.02*	0.34	
	P2		0.74	0.23	
	P3			0.91	
R-min (mm)	7.4 ± 0.27	7.3 ± 0.31	7.4 ± 0.18	7.3 ± 0.2	0.515
Q value	−0.3 ± 0.13	−0.1 ± 0.2	−0.4 ± 0.66	−0.1 ± 0.41	0.179

Notes: *Significant as p value ≤0.05, P1: P value compared to control group, P2: P value compared to mild group, P3: P value compared to moderate group,
Abbreviations: CF, cornea front; OSAS, Obstructive sleep apnea syndrome.

Table 3 Posterior Corneal Topographic and Pachymetry Parameters in Control Subjects and Obstructive Sleep Apnea Groups (Mild, Moderate, and Severe)

	Control Group (n=20)	Mild OSAS Group (n=16)	Moderate OSAS Group (n=16)	Sever OSAS group (n=23)	P value
K1 (D)	−6.2 ± 0.2	−6.1 ± 0.36	−6.3 ± 0.17	−6.1 ± 0.33	0.394
K2 (D)	−6.5 ± 0.26	−6.3 ± 0.25	−6.5 ± 0.22	−6.5 ± 0.3	0.348
Km (D)	−6.3 ± 0.22	−6.2 ± 0.3	−6.4 ± 0.2	−6.3 ± 0.19	0.363
K max (D)	45.9 ± 1.68	46.1 ± 1.9	45.5 ± 1.12	46.4 ± 1.27	0.575
Astigmatism	0.3 ± 0.16	0.2 ± 0.1	0.3 ± 0.1	0.4 ± 0.22	0.250
R-min (mm)	5.9 ± 0.27	6 ± 0.36	5.9 ± 0.22	5.9 ± 0.25	0.895
Q value	−0.5 ± 1.19	0 ± 0.31	0.1 ± 0.31	0.1 ± 0.44	0.116
Pachymetry at corneal apex (um)	545.4 ± 42.02	530.7 ± 22.78	506.3 ± 65.57	528.6 ± 20.37	0.143
Pachymetry at thinnest location (um)	540.8 ± 41.85	532.5 ± 21.34	506 ± 68.9	521.1 ± 19.81	0.170
Pachymetry at pupil center (um)	543.9 ± 42.15	535 ± 22.65	505.2 ± 67.31	526.4 ± 20.47	0.146

Notes: P1: P value compared to control group, P2: P value compared to mild group, P3: P value compared to moderate group,
Abbreviation: OSAS, Obstructive sleep apneas syndrome.

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Table 4 Comparison of Corneal Volume and Anterior Chamber Volume Parameters Among Control Subjects and Obstructive Sleep Apnea Syndrome Groups of Different Severity

	Control Group (n=20)	Mild OSAS Group (n=16)	Moderate OSAS Group (n=16)	Sever OSAS group (n=23)	P value
Corneal volume (mm3)	60.5 ± 5.25	58.1 ± 2.22	59.1 ± 5.92	58.5 ± 3.91	0.503
Anterior chamber volume (mm3)	176.4 ± 30.46	112.4 ± 57.54	176.8 ± 51.57	145.4 ± 31.16	0.002*
	P1	0.003*	1	0.053	
	P2		0.021*	0.232	
	P3			0.271	
AC depth (mm)	3.1 ± 0.27	2.6 ± 0.21	3.1 ± 0.37	2.8 ± 0.29	<0.001*
	P1	0.001*	0.998	0.004*	
	P2		0.008*	0.429	
	P3			0.052	

Notes: *Significant as p value ≤0.05, P1: P value compared to control group, P2: P value compared to mild group, P3: P value,
Abbreviations: AC depth, Anterior chamber depth; OSAS, Obstructive sleep apnea syndrome.

Table 5 Comparison of Corneal Tomographic Indices (Progression Index, Front and Back Elevation, and Intra Ocular Pressure) Between Control Subjects and Obstructive Sleep Apnea Groups

	Control Group (n=20)	Mild OSAS Group (n=16)	Moderate OSAS Group (n=16)	Sever OSAS Group (n=23)	P value
Min PI	0.7 ± 0.12	0.5 ± 0.16	1.3 ± 0.65	0.7 ± 0.23	<0.001*
	P1	0.584	<0.001*	0.978	
	P2		<0.001*	0.409	
	P3			<0.001*	
Max PI	1.2 ± 0.13	1.2 ± 0.29	1.6 ± 0.74	13.3 ± 21.48	0.033*
	P1	1	1	0.039*	
	P2		1	0.245	
	P3			0.273	
Avg PI	1 ± 0.1	0.9 ± 0.2	1.4 ± 0.58	1.1 ± 0.46	0.061
Front elevation (um)	1.1 ± 2.47	−0.7 ± 2.25	−4.3 ± 9.91	0.8 ± 4.73	0.094
Back elevation (um)	5.6 ± 3.8	7.8 ± 8.52	10.8 ± 3.31	14.2 ± 8.15	0.001*
	P1	0.886	0.326	<0.001*	
	P2		0.852	0.155	
	P3			0.669	
IOP (mm hg)	13.7 ± 1.25	16.1 ± 3.07	19.1 ± 4.75	17.4 ± 2.65	<0.001*
	P1	0.229	<0.001*	<0.001*	
	P2		0.187	0.685	
	P3			0.484	

Notes: *Significant as p value ≤0.05, P1: P value compared to control group, P2: P value compared to mild group, P3: P value compared to moderate group,
Abbreviations: IOP, Intraocular pressure; PI, Progression index; AVG PI, Average progression index; Min PI, Minimum progression index; Max PI, Maximum progression index.

Table 6 Comparison of ART Max According to Severity of Obstructive Sleep Apneas Syndrome (OSAS)

	Control Group (n=20)	Mild & Moderate OSAS Group (n=32)	Sever OSAS Group (n=23)	P value
ART max	420.4 ± 62.28	475.5 ± 98.29	395.6 ± 134.69	0.511

Abbreviations: ART max, The Ambrósio relational thickness maximum; OSAS, Obstructive sleep apnea syndrome.

Table 7 Comparison of Sleep Study Parameters (Apnea Indices, Oxygen Saturation, Heart Rate and Arousal Index) Among Different Stages of Obstructive Sleep Apnea Syndrome

	Mild OSAS Group (n=16)	Moderate OSAS Group (n=16)	Sever OSAS Group (n=23)	P value	Post hoc
Obstructive apnea	2.4 ± 2.46	13.3 ± 3.81	52.2 ± 22.5	<0.001*	P1 = 0.659 P2 <0.001* P3 = 0.002*
Central apnea index (events/hour)	0 ± 0	0.9 ± 0.29	0.9 ± 0.97	0.065	
Hypopnea index (events/hour)	4 ± 3.89	5.1 ± 0.75	10.7 ± 10.94	0.236	
RDI (events/hour)	6.5 ± 3.36	19.2 ± 2.77	63.5 ± 24.09	<0.001*	P1 = 0.608 P2 <0.001* P3 = 0.001*
Max HR (beat/min)	94.3 ± 22.81	103.5 ± 2.89	106.9 ± 13.76	0.253	
MIN HR (beat/min)	65.7 ± 14.54	57 ± 10.39	61.8 ± 9.35	0.473	
Desaturation index (events/hour)	3.7 ± 4.11	22.6 ± 10.39	37.7 ± 20.84	0.001*	P1 = 0.245 P2 =0.001* P3 = 0.29
Min O saturation (%)	92.3 ± 1.03	87 ± 4.62	85.9 ± 5.72	0.035*	P1 =0.254 P2=0.027* P3 =0.912
Avg. O saturation (%)	93.7 ± 4.59	95.5 ± 0.58	86.5 ± 16.24	0.335	
Arousal index (events/hour)	3.4 ± 3.87	8.3 ± 4.16	2.6 ± 1.55	0.003*	P1 =0.032* P2 =0.862 P3 =0.002*

Notes: *Significant as p value ≤0.05, P1: P value between mild group and moderate group, P2: P value between mild group and severe group, P3: P value between moderate group and severe group,

Abbreviations: RDI, Respiratory distress index; HR, heart rate; OSAS, Obstructive sleep apnea syndrome.

There was a significant negative correlation between arousal index and pachymetry at the corneal apex, the thinnest location, and the pupil center. The arousal index was also significantly correlated with average, minimum, and maximum progression indices, intraocular pressure, and corneal back elevation. In addition, respiratory distress index was significantly correlated with average and minimum progression indices. A significant positive correlation was observed between central apnea index and intraocular pressure (Table 8).

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Table 8 Correlation Between Polysomnography Parameters and Different Corneal Ectasia Indices

		R	p-value
Pachymetry at corneal Apex (um)	Arousal index	−0.681	<0.001*
Pachymetry at thinnest location (um)	Arousal index	−0.707	<0.001*
Pachymetry pupil centre (um)	Arousal index	−0.707	<0.001*
Average PI	Arousal index	0.501	0.021*
Min PI	Arousal index	0.697	<0.001*
Max PI	Arousal index	0.590**	0.005*
IOP (mm Hg)	Arousal index	0.699	<0.001*
Back elevation (um)	Obstructive apnea	0.561	0.001*
Average PI	RDI	0.627	0.0002*
Min PI	RDI	0.64	0.014
IOP (mm Hg)	Central apnea index	0.405	0.026*

Note: *Significant as p value ≤0.05.
Abbreviations: IOP, Intraocular pressure; PI, Progression index; AVG PI, Average progression index; Min PI, Minimum progression index; Max PI, Maximum progression index; RDI, Respiratory distress index.

Discussion

Obstructive sleep apnea (OSA) has become a common disorder in recent years, particularly with the increasing prevalence of obesity among younger populations.[16] The close relationship between hypoxia resulting from apneic episodes and various ocular complications has been widely discussed. In the present study, we attempted to highlight a potential contributing factor, namely the respiratory arousal index, which may play a role in ocular changes observed in OSA patients.

In the current study, patients in the severe OSA group were significantly older than those in the control and moderate groups, which is consistent with previous studies demonstrating a higher prevalence of sleep-disordered breathing among elderly individuals. However, age may represent a potential confounding factor that could influence ocular parameters. Therefore, the findings of this study should be interpreted with caution considering these differences.[17] The mechanisms by which aging increases the risk of severe OSA require further investigation; however, age-related changes in airway anatomy and increased collapsibility may contribute to disease severity.[18]

A higher proportion of males was observed in the severe OSA group compared with the control group, although no statistically significant difference was detected among the OSA subgroups. This finding is consistent with epidemiological data showing a slightly higher prevalence of OSA in males than females in Egypt, with reported incidence rates of 3–7% and 2–5%, respectively, among adults aged 30–60 years.[19]

The higher frequency of males in severe OSA may be attributed to increased upper airway collapsibility, longer airway length, and greater soft tissue volume in the lateral pharyngeal walls, all of which increase susceptibility to airway collapse.[20]

Visual acuity was significantly lower in the moderate and severe OSA groups compared with controls, which may be related to the higher cylindrical refractive error observed in these groups. However, no statistically significant differences were observed between groups regarding anterior and posterior keratometric values, corneal astigmatism, R-min, and Q-value. These findings are consistent with those reported by Irem Isik et al, who found no significant differences in corneal topographic parameters between OSA patients and controls.[21]

Although pachymetry values at the corneal apex, thinnest location, and pupil center were lower in OSA groups, the differences were not statistically significant. Similar findings have been reported in some studies that demonstrated

thinning of central corneal thickness,;[21,22] whereas other studies did not detect significant changes in corneal thickness.[23] These variations may be attributed to differences in sample size, disease severity, or measurement techniques.

Intraocular pressure was higher in all OSA groups compared with controls, reaching statistical significance in moderate and severe OSA. These findings are consistent with previous reports demonstrating a positive correlation between OSA severity and intraocular pressure.[24]

The observed elevation in IOP in our study was associated with a reduction in anterior chamber volume and depth. Although anterior chamber angle was not directly measured, these findings may have implications for aqueous humor dynamics.

The relationship between OSA and glaucoma has been widely investigated; however, the type of glaucoma associated with OSA remains controversial. Some studies have reported a higher prevalence of normal tension glaucoma in OSA patients.[25] Whereas others have demonstrated an increased association with open-angle glaucoma,[26] an additional reports have described an elevated risk of primary angle closure glaucoma.[27]

In contrast, other investigators have suggested that OSA may not be a significant risk factor for glaucoma and may even be associated with transient reductions in intraocular pressure during apneic episodes.[28,29]

Inflammation plays an important role in the physiological response to hypoxic stress experienced during sleep apnea. Hypoxia-inducible factors and inflammatory mediators such as nuclear factor-kappa B (NF-κB) regulate the transcription of genes involved in metabolic adaptation and vascular responses, including phosphoglycerate kinase and vascular endothelial growth factor.[30]

In this study, we considered the possible contribution of both open-angle and angle-closure mechanisms in OSA-related ocular changes. The reduction in anterior chamber depth and volume observed in our patients may suggest a potential predisposition to angle crowding, although the anterior chamber angle was not directly measured. Vasodilation of iris vessels secondary to recurrent hypoxemia may also contribute to these structural changes.

Sustained or recurrent hypoxia may lead to oxidative stress and inflammatory activation, which have been implicated in trabecular meshwork dysfunction and impaired aqueous outflow, potentially contributing to elevated intraocular pressure.[31]

Indices suggestive of early ectatic changes, including progression indices and back elevation, were higher in moderate and severe OSA groups. These findings agree with previous studies reporting an association between OSA and corneal ectatic changes. Hypoxia-induced activation of matrix metalloproteinases may contribute to degradation of stromal collagen and extracellular matrix components, thereby affecting corneal biomechanics.[32,33]

In the present study, corneal topographic changes were also correlated with polysomnography parameters. To the best of our knowledge, few studies have evaluated such correlations in detail. We observed a significant negative association between respiratory arousal index and pachymetry at the corneal apex, thinnest location, and pupil center, along with significant positive correlations between arousal index and progression indices.

The respiratory arousal index reflects the number of cortical arousals per hour of sleep and represents an important component of sleep fragmentation.[27] Recurrent arousals are associated with sympathetic activation and catecholamine release, which may contribute to systemic and ocular physiological changes.[34]

The cornea is richly innervated and contains adrenergic receptors that may respond to sympathetic stimulation.[35] Experimental observations have also suggested that catecholamines may influence intraocular pressure.[36]

So, it could be catecholamine surge during repeated respiratory arousal index is associated with increased catecholamine release from sympathetic innervation of the cornea, which can influence the development of corneal ectasia and lead to elevation of IOP in patients having OSA.

There is a moderate positive correlation between (respiratory distress index (RSI) and obstructive apnea) and average pachymetric progression index, minimum progression index, and corneal back elevation. It could be attributed to the hypoxia-inflammation theory discussed previously, which we precisely discussed.

A moderate positive correlation exists between IOP elevation and central apnea index (0.405). To the best of our knowledge, this is the first to be described.

This study has several limitations. First, the sample size was relatively small, which may limit the generalizability of the findings. Second, the study was conducted at a single center. Third, intraocular pressure was measured only during morning hours, and diurnal variation was not assessed. Finally, age differences between groups may have influenced some anterior segment parameters. Further studies of larger sample sizes are recommended to confirm these findings.

This study has several limitations that should be acknowledged. First, there were statistically significant differences in age among the study groups, particularly in the severe OSA group, which may act as a potential confounding factor. Second, although an upper limit for body mass index (BMI) was applied to minimize the effect of obesity, detailed BMI analysis across the study groups was not performed. Third, the ophthalmologist performing the examinations was not blinded to the OSA status of the participants, which may introduce observer bias; however, the use of objective measurement techniques may have minimized this effect.

Also, Ambrósio relational thickness maximum (ARTmax) data were not available for all eyes, particularly in the mild and moderate OSA groups. This resulted in a limited number of observations in each subgroup, necessitating the combination of these groups for this parameter, which may have reduced the ability to detect subgroup-specific differences.

Additionally, some variables demonstrated relatively large standard deviations, likely reflecting variability within the sample or the presence of outliers, and therefore these findings should be interpreted with caution. Finally, the relatively small sample size may limit the generalizability of the results.

In conclusion, intraocular pressure was higher in moderate and severe OSA groups, and anterior chamber depth was significantly shallower in mild and severe OSA patients. A significant negative correlation was observed between the respiratory arousal index and corneal pachymetry, suggesting a potential role of sleep fragmentation in corneal structural changes. Further studies with larger sample sizes are recommended to better clarify the underlying mechanisms and confirm these findings.

Data Sharing Statement

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

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This paper has not been presented in a meeting. We clarified that the paper presents novel scientific content, which totally differs from our previous publications in the journal about the same issue.

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Disclosure

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