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Primary open-angle glaucoma in patients with obstructive sleep apnoea in a Colombian population: a cross-sectional study

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ABSTRACT

Objective Determine the prevalence, functional and structural alterations of primary open-angle glaucoma (POAG) in patients with obstructive sleep apnoea (OSA).

Design Cross-sectional.

Setting Tertiary hospital associated with specialised center in ophthalmologic images in Bogota, Colombia.

Participants 150 patients, for a sample of 300 eyes, 64 women (42.7%) and 84 men (57.3%) between 40 and 91 years old with a mean age of 66.8 (±12.1) years.

Interventions Visual acuity, biomicroscopy, intraocular pressure, indirect gonioscopy and direct ophthalmoscopy. Patients classified as glaucoma suspects underwent automated perimetry (AP) and optical coherence tomography of the optic nerve

Outcome measure The primary outcomes are the determination of prevalence of glaucoma suspects and POAG in patients with OSA. Secondary outcomes are the description of functional and structural alterations in computerised exams of patients with OSA.

Results The prevalence of glaucoma suspect was 12.6%, and for POAG was 17.3%. No alterations in the appearance of the optic nerve was seen in 74.6%, focal or diffuse thinning of the neuroretinal rim (16.6%) was the most frequently finding, followed by asymmetry of the disc>0.2 mm (8.6%) (p=0.005). In the AP, 41% showed arcuate, nasal step and paracentral focal defects. The mean retinal nerve fiber layer (RNFL) was normal (>80 µM) in 74% of the mild OSA group, 93.8% of the moderate group and 17.1% of the severe group. Similarly, normal (P5-90) ganglion cell complex (GCC) in 60%, 68% and 75%, respectively. Abnormal results in the mean RNFL was seen in 25.9%, 6.3% and 23.4% of the mild, moderate and severe groups, respectively. In the GCC, 39.7%, 33.3% and 25% of the patients in the aforementioned groups.

Conclusion It was possible to determine the relationship between structural changes in the optic nerve and the severity of OSA. No relationship with any of the other studied variables was identified.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Two-stage design for collecting data, based in complete clinical ophthalmological examination, followed by computerised exams to ensure diagnosis.
⇒ The collected data and results were verified by two glaucoma specialists.
⇒ The study reflects the situation in a tertiary hospital with a specialised ophthalmological centre in Bogota, Colombia. Generalisability of our study results needs to be verified
⇒ The main limitations of the study were the lack of a control group, sample size and that only one examination was performed, making it not possible to determine if the alterations found persisted or if the glaucoma progressed over time.

INTRODUCTION

Glaucoma is considered the second cause of irreversible blindness worldwide in people over 40 years of age, defined as optic neuropathy that generates loss of neural tissue in the optic nerve and, therefore, changes in the visual field (VF).[1] Its prevalence is estimated at 1.86%–7% in Hispanic, Asian and Native American ethnicities and 5.6% in African Americans.[1 2] It is estimated that by the year 2020, more than 80 million people will be affected, 11.2 million of whom will have bilateral blindness, with an annual incidence of 2.4 million cases.[3] It is a progressive and silent disease whose visual manifestations develop in advanced stages.[3]

Obstructive sleep apnoea (OSA) has been identified in some population study as a possible risk factor to glaucoma.[4 5] Its prevalence has been estimated at 27% but varies in different epidemiological studies.[6] OSA is understood as a disorder characterised by repetitive partial or complete obstruction of the upper airway during sleep, causing oxygen desaturation and becoming a risk factor for cardiovascular and neurological diseases.[7] The main symptoms are snoring, daytime sleep, difficulty concentrating and morning headache. Risk factors include obesity, gender, upper respiratory abnormalities, consumption and snoring.[8]

Regarding pathophysiology, upper airway obstruction favours hypoxia, hypercapnia, increased vascular resistance and sympathetic activation.[7 9 10] The relationship between glaucoma and OSA can be explained by the increase in intraocular pressure (IOP) at night due to a supine position,[6 11] which increases episcleral venous pressure,[6] and periods of hypoxia followed by oxidative stress that generate an increased intracranial pressure, which, in turn, decreases cerebral perfusion pressure, subsequently altering the vascular supply to the optic nerve.[7] Sympathetic system is important because, during apnoea episodes, the renin–angiotensin system is activated, leading to a sustained increase in blood pressure during sleep.[8] Dysregulation of optic nerve vascularisation secondary to repetitive hypoxia associated to an imbalance between vasodilator vascular stimuli (nitric oxide) and vasoconstrictors (endothelin) that generates variations in the ocular perfusion pressure and therefore susceptibility to ischaemia in the optic nerve head.[12] Additionally, episodes of apnoea and hypopnoea contribute to the development of complications with endothelial dysfunction, vascular remodelling and hypertension.[13]

Due to the complexity and controversy in the association between tOSA and POAG,[9 10] is very important to be able to categorise the frequency of cases. Furthermore, by identifying the risk factor for OSA,[14] a protocol that allows patients to be referred to ophthalmological consultation in a timely manner could be created in order to avoid visual sequelae. Additionally, patients requiring medical treatment in order to prevent optic nerve damage and avoid blindness can be timely identified.[15]

The objective of this study is to characterise the prevalence of primary open-angle glaucoma, as well as its functional and structural alterations, in patients with OSA.

MATERIALS AND METHODS

We included all the legal adult patients who were listed in Bogota's *Hospital Militar Central* database as diagnosed with OSA (by polysomnography) between January 2013 and December 2019. These patients' medical histories were used to determine demographic data such as age and ethnicity as well as family history, arterial hypertension (high blood pressure) and diabetes mellitus histories. The findings in polysomnography determined the severity of OSA using the Apnoea/Hipopnea Index AHI (sum of apnoea and hypopnea events divided by total hours of sleep) to categorise it into one of three groups: mild (index 6–15), moderate (16–30) and severe (>30).[16]

Exclusion criteria include psychiatric or neurological disorders, optic neuropathy, anterior ischaemic optic neuropathy, heavy smoking (20 cigarettes per day), alcohol or psychoactive substance abuse, any condition affecting the VF (such as intracranial or ocular lesions), coexisting retinal disease, sequelae of trauma and/or eye inflammation, congenital ocular anatomical alterations, intraocular surgery (with the exception of cataract surgery), history of hypertensive crisis, history of prolonged steroid use, uveitis, bronchial asthma, interstitial lung diseases, heart disease, cirrhosis and chronic renal failure. Additionally, patients with closed-angle glaucoma or suspected closure by gonioscopy and patients who did not sign the informed consent in the study were excluded.

Two clinical days were carried out. On the first day, two glaucoma specialists in the hospital's Department of Ophthalmology, performed a complete ophthalmological examination, including visual acuity (VA) using the Snellen chart, the anterior segment with a slit lamp, intraocular pressure (IOP) measurement using a Goldmann tonometer and an assessment of indirect gonioscopy employing a Posner lens. The ophthalmoscopy was performed with a 90D lens (Volk Optical Inc) to determine alterations such as focal or diffuse thinning of the neuroretinal rim, optic disc haemorrhages, defects in the retinal nerve fiber layer (RNFL) and disc asymmetry greater than 0.2 mm.

Glaucoma suspects were selected if they showed the following conditions as open angles by gonioscopy, optic nerve excavations greater than 0.6 mm and asymmetry of optic nerve excavations greater than 0.2 mm. Associated, IOP greater than or equal to 22 mm Hg and IOP within normal limits linked to thinning of the neuroretinal rim. Additionally, thinning of RNFL, optic nerve vessels causing 'bayoneting' flexion or deformation, peripapillary haemorrhages and loss of the inferior > superior > nasal > temporal ISNT rule on RNFL.

On the second day, glaucoma suspects were invited to the *Instituto de Macula y Retina Oftalmocenter* in Bogota where automated perimetry (AP) (Humphrey Field Analyzer model 750i, Carl Zeiss-Meditec, Dublin, California, USA, SITA Standard program) and optic nerve optical coherence tomography (OCT) (spectral domain with the Cirrus OCT team) exams were performed. In the AP, the mean deviation (MD) defined as the average difference from normal expected value in the patient age group and alterations in the VF (superior arcuate defect, inferior arcuate defect, generalised decrease in sensitivity, nasal step, focal defects and paracentral and normal defects) were determined. The OCT measured the mean RNFL and ganglion cell complex (GCC), as well as analysed the optic disc. Subsequently, two glaucoma specialists participating in the study analysed the examinations.

The data collected were grouped into four groups for analysis. The first group analysed the demographic data and risk factors (glaucoma family history, history of arterial hypertension or diabetes mellitus). The second group analysed the findings in the clinical history and ophthalmological examination (AHI, VA, basal IOP, gonioscopy, optic nerve excavation and suspicious changes in the optic nerve). The third group evaluated the results of the VF (MD value classified as mild ≤6.00 dB, moderate MD −6.00 to −12.00 dB and severe ≥12.00dB) and the campimetry alterations. In the fourth group, the OCT results were categorised as normal (>80 µM), suspicious (70–79 µM) or abnormal (<70 µM) for RNFL and normal

(P5-90%), suspicious (P1-5%) or abnormal (p<5%) for GCC.

The diagnosis of glaucoma suspect was assigned to patients that showed either risk factors linked to developing glaucoma or clinical signs suggesting the disease but without functional alterations in the VF or structural alterations in the OCT. Patients were classified as having POAG when they showed functional alterations in the VF and structural alterations in the OCT.

For the statistical analysis, SPSS V.23 software was used. A χ^2 test to determine the association between variables and the severity of OSA. A Shapiro-Wilk test to identify if the sample data followed a normal distribution, Levene's test to specify if the continuous variables had the same variance and analysis of variance test to compare the means of the continuous variables. Using their respective confidence intervals, we established the prevalence of glaucoma suspects as well as POAG with OSA. The frequency of each visual alteration in the patients was determined, and the findings of the OCT and VF were described.

Regarding bias control, all clinical records of the patients diagnosed with OSA met the inclusion and exclusion criteria proposed. The personnel in charge were trained to carry out the evaluation; the same person was in charge of a specific function. Data collected and results were supervised and analysed by two glaucoma specialists. The data collected were grouped into four groups for analysis. It was used the same diagnostic equipment, which was properly calibrated and had the corresponding technological updates.

Patient and public involvement

No patients were involved in the design, recruitment or conduct of the study. The results were informed directly to each participant of the study but were not disseminated in the others study participants.

RESULTS

The study included 150 patients (300 eyes). A total of 42.7% women and 57.3% men between 40 and 91 years old with a mean age of 66.8 (±12.1). All 150 patients were Hispanic, 1.3% had a family history of glaucoma, 64.7% high blood pressure and 20.7% diabetes mellitus (table 1).

The AHI was categorised as 62% mild OSA, 17.3% as moderate and 20.7% as severe. In total, 90 eyes were glaucoma suspects, which 52 eyes were POAG, for a prevalence of 17.3% for POAG and 12.6% for glaucoma suspect.

- *VA:* the most prevalent were between 20/20 and 20/60 in 94.6% (p=0.057). The patients with good visual acuities were in the mild severity group (20/20, 28.5%; 20/25, 23.7%; 20/30, 19.4%; 20/40, 15.1%), followed by the lowest VA in the moderate group (20/50, 11.5%; 20/150, 1.9%) and the severe group (20/60, 4.8%; 20/70, 3.2%; 20/80, 4.8%) (table 2).

Table 1 Demographic variables

Demographic variables		n (%)
Sex	1. Female	64 (42.7)
	2. Male	86 (57.3)
Ethnicity	1. Hispanic	150 (100)
	2. Afro descendant	0
	3. Indigenous	0
Family history	1. Yes	2 (1.3)
	2. No	148 (98.6)
Hypertension	1. Yes	97 (64.7)
	2. No	53 (35.3)
Diabetes mellitus	1. Yes	31 (20.7)
	2. No	119 (79.3)

- *IOP:* the mean was 13 mm Hg (± 2.6), 13.3 mm Hg (± 0.2) for mild cases, 13.3 mm Hg (± 0.3) for moderate cases and 12.5 mm Hg (± 0.3) for severe cases (p=0.107). Regarding the excavation of the optic nerve, the mean was 0.4 mm (±0.2) (p=0.953) (tables 3 and 4).
- *Optic nerve changes:* 74.6% of the eyes showed no alterations, and 16.6% have a focal or diffuse thinning of the neuroretinal rim. These findings were more prevalent in the mild OSA group (21.5%), followed by the moderate OSA group (15.4%) and the severe OSA group (3.2%). Disc asymmetry (>0.2 mm) was observed in 8.6%, with a higher prevalence in the severe OSA group (16.1%), followed by the mild OSA group (8.2%) and finally the moderate group (7.7%) (p=0.005) (table 2).
- *Campimetry alterations:* 54.4% were normal with no significant differences between the groups (mild OSA 55.2%), moderate (56.3%) and severe (50.0%)). The second alteration was superior arcuate defect in 14.4%, more commonly found in the mild OSA group (12.1%). The next alternation was focal defects in 13.3%, mainly in the mild group (13.8%), with an equal percentage in the moderate and severe groups (12.5%). The nasal step defect was present in 12.2%, most frequently in the moderate OSA group (18.8%), followed by the mild group (13.8%) and severe group (6.3%). To a lesser extent, a paracentral defect was seen in 3.3%—only in the mild OSA group (5.2%)—and an inferior arcuate defect was seen in 2.2% in the moderate (6.3%) and mild (1.7%) OSA groups (p=0.583) (table 5).
- The decrease in the MD was mild (≤6.00 dB) in 83.3%, distributed as follows: moderate OSA (56.3%), mild OSA (55.2%) and severe OSA (50.0%). The moderate decrease in MD (−6.00 and −12.00 dB) occurred in 13.3%, distributed as follows: severe OSA (18.8%), mild OSA (12.1%) and moderate OSA (6.3%). The severe decrease in MD (≥12.00 dB) was 4.4%,

Table 2 Visual acuity and suspicious changes in the optic nerve

Variable		Obstructive sleep apnoea severity (AHI)			Total (n=300)	P value
		Mild (n=186)	Moderate (n=52)	Severe (n=62)	n (%)	
Visual acuity	20/20	53 (28.5%)	14 (26.9%)	13 (21.0%)	80 (26.6)	0.031
	20/25	44 (23.7%)	11 (21.1%)	6 (9.7%)	61 (20.3)	
	20/30	36 (19.4%)	10 (19.2%)	22 (35.5%)	68 (22.6)	
	20/40	28 (15.1%)	7 (13.5%)	9 (14.7%)	44 (14.6)	
	20/50	15 (8.1%)	6 (11.5%)	3 (4.8%)	24 (8)	
	20/60	2 (1.1%)	2 (3.8%)	3 (4.8%)	7 (2.3)	
	20/70	3 (1.6%)	0	2 (3.2%)	5 (1.6)	
	20/80	1 (0.5%)	0	3 (4.8%)	4 (1.3)	
	20/100	2 (1.1%)	0	0	2 (0.6)	
	20/150	0	1 (1.9%)	1 (1.6%)	2 (0.6)	
	20/200	2 (1.1%)	0	0	2 (0.6)	
Suspected changes in the optic nerve	1. Focal or diffuse thinning of the neuroretinal rim	40 (21.5%)	8 (15.4%)	2 (3.2%)	50 (16.6)	0.005
	2. Disc haemorrage	0	0	0	0	
	3. RNFL alterations	0	0	0	0	
	4. Disc asymmetry >0.2	12 (8.2%)	4 (7.7%)	10 (16.1%)	26 (8.6)	
	No alterations	134 (72.0%)	40 (76.9%)	50 (80.6%)	224 (74.6)	

distributed mostly in severe OSA (12.5%) versus mild OSA (3.4%) (p=0.305).

- *OCT of the optic nerve:* the mean RNFL was normal in 74% of the mild OSA group, 93.8% of the moderate OSA group and 17.1% of the severe OSA patients. Similarly, the GCC was normal in 60%, 68% and 75% of the respective OSA severity groups. Abnormalities in the mean RNFL were seen in 25.9%, 6.3% and 23.4% of the mild, moderate and severe groups, respectively (p=0.081). Regarding GCC, abnormalities were found in 39.7%, 33.3% and 25% for these same groups (p=0.218) (table 5).

DISCUSSION

OSA has been considered a possible risk factor for developing glaucoma. According to the pathophysiology of OSA, a vascular theory has been postulated that relates to the increase in vascular resistance secondary to hypoxia. This would cause dysregulation of optic nerve head vascularisation.[7 12] There is also a mechanical theory that the increase of IOP caused by supine position and an increase of episcleral venous pressure linked to obesity could lead to this condition.[6 11] According to these theories, functional and structural changes would be generated in the optic nerve, leading to glaucoma.

Table 3 Intraocular pressure and optic nerve excavation

Variable	Median	SD
Age (years)	66.8	12.1
Intraocular pressure (mm Hg)	13	2.6
Optic nerve excavation (mm)	0.4	0.2

This study found the prevalence of POAG in patients with OSA was 17.3%, and glaucoma suspect was 12.6%. These prevalence values are similar to those of studies such as that of Bagabas *et al*,[17] which found a 16% prevalence, but were much lower than those found in studies such as Friedlander *et al*[18] and Wozniak *et al*.[19] In various meta-analyses, a link between OSA and glaucoma[7 20] has been identified; Shi *et al*[21] revealed a significant relationship between the prevalence of glaucoma and OSA in case–control studies (OR=1.96) and cross-sectional studies (OR=1.41).

A high number of patients have visual acuities between 20/20 and 20/60, indicating no mayor compromise of the VA in the population studied. No statistical relationship was found regarding the severity of OSA and visual acuities, most likely indicating that OSA does not increase loss of vision.

IOP is recognised as the only modifiable risk factor associated with developing glaucoma, so timely identification would allow for rapid treatment in order to prevent the progression of glaucoma. Various studies have identified a relationship between OSA and IOP values, finding positive correlation with the supine position (Moghimi *et al*,[22] Sergio *et al*[23] and Yee *et al*).[11] This is contrary to studies described by Nowak *et al*[24] and Shalaby *et al*.[25] In studies such as that of Carnero *et al*,[26] patients with OSA experience upper limits of IOP. Unlike the aforementioned, in our study, IOP occurred within normal, with no evidence of a correlation with the severity of OSA.

Table 4 Obstructive sleep apnoea severity, intraocular pressure and optic nerve excavation

Obstructive sleep apnoea severity (AHI)		Intraocular pressure (mm Hg)	Optic nerve excavation (mm)	Total n
Mild	**Median**	13.3	0.4	186
	SD	0.2	0.0	
Moderate	**Median**	13.3	0.4	52
	SD	0.3	0.0	
Severe	**Median**	12.5	0.4	62
	SD	0.3	0,0	
ANOVA p value		0.107	0.953	

ANOVA, analysis of variance.

The excavation with greater sizes was reported in patients with glaucoma and no relation to OSA severity. Suspicious changes in the optic nerve were the only variable in which a statistical significance was found regarding the severity of OSA. This demonstrates how OSA can influence the optic nerve anatomy by favouring the development of optic neuropathy and, to some extent, the onset of POAG. A higher percentage of these changes was found in the severe OSA group. It was possible to identify that focal or diffuse thinning of the neuroretinal rim in the mild OSA group as a possible indicator of early and focal damage of the optic nerve. Additionally,

Table 5 Functional and structural findings in the visual field and optic nerve OCT

Variable		Obstructive sleep apnoea severity (AHI)			Total (n=90)	P value
		Mild (n=58)	Moderate (n=16)	Severe (n=16)	n (%)	
Visual field	1. Superior arcuate defect	7 (12.1%)	1 (6.3%)	5 (3.1%)	13 (14.4)	0.583
	2. Inferior arcuate defect	1 (1.7%)	1 (6.3%)	0	2 (2.2)	
	3. General diminished sensitivity	0	0	0	0	
	4. Nasal step	7 (12.1%)	3 (18.8%)	1 (6.3%)	11 (12.2)	
	5. Focal defects	8 (13.8%)	2 (12.5%)	2 (12.5%)	12 (13.3)	
	6. Paracentral defects	3 (5.2%)	0	0	3 (3.3)	
	7. Normal	32 (55.2%)	9 (56.3%)	8 (50.0%)	49 (54.4)	
Mean deviation	1. MD ≤6.00 dB	49 (84.5%)	15 (93.8%)	11 (68.8%)	75 (83.3)	0.305
	2. MD −6.00 a-12.00 dB	7 (12.1%)	1 (6.3%)	3 (18.8%)	11 (12.2)	
	3. MD ≥12.00 dB	2 (3.4%)	0	2 (12.5%)	4 (4.4)	
Mean Retinal nerve fib layer	1. Normal >80 µm	43 (74.1%)	15 (93.8%)	12 (17.1%)	70 (77.7)	0.081
	2. Suspicious 70–79 µm	12 (20.7%)	0	1 (6.3%)	13 (14.4)	
	3. Abnormal <70 µm	3 (5.2%)	1 (6.3%)	3 (18.8%)	7 (7.7)	
Ganglion cell complex	1. Normal (green)	35 (60.3%)	11 (68.8%)	12 (75.0%)	58 (64.4)	0.218
	2. Suspicious (yellow)	11 (19.0%)	4 (25.0%)	0	15 (16.6)	
	3. Abnormal (red)	12 (20.7%)	1 (6.3%)	4 (25.0%)	17 (18.9)	
Primary open angle glaucoma	1. Yes	35 (60.3%)	9 (56.3%)	8 (50.0%)	52 (57.8)	0.752
	2. No	23 (39.7%)	7 (43.8%)	8 (50.0%)	38 (42.2)	
Glaucoma suspects	1. Yes	23 (39.7%)	7 (43.8%)	8 (50.0%)	38 (42.2)	0.752
	2. No	35 (60.3%)	9 (56.3%)	8 (50.0%)	52 (57.8)	

asymmetry >0.2 mm was identified in patients with severe OSA, indicating greater damage by a diffuse thinning of the neuroretinal rim, from which it could be inferred that the progression of optic nerve damage is related to the severity of OSA. This is similar to the findings of Tsang *et al*,[27] a case–control study that demonstrated an incidence of suspicious optic nerve changes four times higher in patients with moderate or severe OSA. Another study by Uslu *et al*[28] suggests an increased optic nerve excavation in patients with OSA, and it could be considered an indicator of neural damage in the early diagnosis of OSA. In contrast to previous results, Lin *et al*[29] showed that the optic nerve parameters did not differ between OSA severity groups, nor did it differ from the general population described by Salzgeber *et al*.[30]

As for the functional changes in the VF, authors such as Tsang *et al*[27] found abnormalities in MD and pattern SD correlated with OSA severity index,[23] indicating a direct relationship between these pathologies. In contrast, this study identified that most of the patients did not show functional alterations, which is similar to the findings of Swaminathan *et al*[31] and Salzgeber *et al*.[30]

According to VF numerical parameters, a high percentage of patients showed a mild decrease in the MD that predominated in the moderate OSA group. Meanwhile, a moderate to severe decrease was seen in the severe OSA group. This indicates that the greater the severity of OSA, the more compromised the VF; this signifies a possible link between these pathologies, although it could not be statistically demonstrated in this study.

It has been described that the first VF manifestations in patients with POAG are generalised depression, enlargement of the blind spot, Seidel's scotoma or nasal step.[32] Subsequently, paracentral defects that may join with the blind spot, as well as temporary steps, and superior or inferior arcuate defects. Finally, progressive damage can lead to peripheral constriction of the VF, leaving central or temporal vision islands.[32] Comparing this with what was observed in this study, the functional alterations described are commonly identified in intermediate or advanced stages of POAG, which occurred more frequently in the mild OSA group; therefore, it could be inferred that mild stages of OSA may be related to greater VF alterations.

The OCT showed that most of the patients were within normal ranges, patients with abnormal results showed an increased severity of structural nerve damage as the analysis went from mild and moderate to severe OSA. This is supported by meta-analyses that demonstrated a link between moderate and severe OSA with significant thinning of RNFL thickness—mainly in the upper, lower and nasal quadrants.[13 33 34] Additionally, Fan *et al*[35] described greater progression of structural changes (RNFL thickness) in patients with mild OSA, obtaining an 8.448 risk of structural progression in patients with severe OSA. This differs from the studies of Nowak *et al*,[24] Salzgeber *et al*[30] and Kara *et al*.[36] Moreover, Abdullayev *et al*[37] found no alteration of RNFL thickness in patients with OSA that correlated to its severity but described a decrease in GCC in patients with mild severity. In contrast, Uslu *et al* found no alterations in the GCC but a decrease in the peripapillary RNFL thickness.[28]

The general population with POAG, structural changes has been observed as the first manifestation of glaucoma–before a compromised VF,[32] which correspond to the decrease of the GCC in the initial stages with a subsequent decrease in the mean RNFL. When comparing these characteristics of the general population with POAG to the results of this study, it can be deduced that most of the patients with OSA and glaucoma first showed an alteration in the GCC, followed by alterations in the mean RNFL. Some of the patients experienced these alterations before VF alterations; therefore, it can be inferred that the development of glaucoma would manifest itself in the same way in patients with OSA as in the general population with glaucoma.

The main limitations of the study were the lack of a control group and that only one examination was performed. As such, it was not possible to determine if the alterations found persisted or if the glaucoma progressed over time. Finally, due to the sample size, it could not be determined whether OSA correlated with the development of POAG.

In conclusion, this study found a prevalence of glaucoma suspects of 12.6% versus a POAG prevalence of 17.3% in patients diagnosed with OSA. The study demonstrated a relationship between the structural changes of the optic nerve and the severity of OSA, suggesting that OSA might influence the anatomy of the optic nerve and favour the development of optic neuropathy and, to some extent, the onset of POAG. No relationship was identified with any of the other variables that were studied. Future research, studies and follow-ups are recommended for patients with OSA to rule out glaucoma as one of its multiple systemic manifestations.

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