# Floppy Eyelid Syndrome as an Indicator of the Presence of Glaucoma in Patients With Obstructive Sleep Apnea

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**Purpose:** The aim of the study was to investigate whether floppy eyelid syndrome (FES) could be an indicator of glaucoma in patients with obstructive sleep apnea (OSA).

**Materials and Methods:** A total of 152 patients were included: 75 patients with OSA and without FES; 52 patients with OSA and FES; and 25 non-OSA patients. The presence of FES was defined by easy upper eyelid eversion and tarsal papillary conjunctivitis. All the patients underwent a complete ophthalmologic examination to diagnose glaucoma; this included computerized perimetry and retinal fiber layer measurements with optical coherence tomography.

**Results:** The prevalence of glaucoma in OSA patients without FES was 5.33% (4/75). One patient had primary open-angle glaucoma and 3 had previously diagnosed glaucoma. The prevalence of glaucoma in OSA patients with FES was 23.07% (12/52). Six patients had normal-tension glaucoma, 5 had primary open-angle glaucoma and one patient had previously diagnosed glaucoma. None of the 25 patients without OSA had glaucoma. The difference in the prevalence of glaucoma between OSA patients without FES (5.3%) and OSA patients with FES (23.07%) was statistical significant ($P = 0.004$). When adjustments were made for age and body mass index, this significance remained ($P = 0.04$).

**Conclusions:** These data suggest that FES may offer a useful way to identify individuals with a greater probability of having glaucoma in the OSA population.

**Key Words:** glaucoma, floppy eyelid syndrome, obstructive sleep apnea

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Floppy eyelid syndrome (FES) is a frequently underdiagnosed disorder that is characterized by lax upper eyelids, which are easily distorted and everted with minimal traction; it is also associated with chronic papillary conjunctivitis of the upper palpebral conjunctiva.[1] Since its initial description by Culbertson and Ostler,[2] one of the most consistently reported associations of FES is with obstructive sleep apnea syndrome (OSA).[3,4] The prevalence of FES in the OSA population varies from 2%[1] to 32%,[4] according to the reported series. OSA is characterized by recurrent complete or partial upper airway obstructions during sleep. Each episode of apnea or hypopnea is associated with hypoxemia and hypercapnia and associated cardiorespiratory disturbances, with a high risk of cardiovascular and neurovascular complications.[5] The prevalence of OSA is estimated to be between 2% and 5% in middle-aged populations.[6] Ophthalmologic findings in patients with OSA include FES,[2,7] keratoconus,[8] papilledema,[9] optic neurophathy,[10] and glaucoma.[11–16] The prevalence of glaucoma in the OSA population varies from 2% to 9% according to the reported series.[11–16] Only 2 studies[3,7] have previously examined the association between FES and glaucoma. McNab[3] reported 1 in 8 patients (12.5%) with FES and OSA having normal-tension glaucoma (NTG). Robert et al[7] reported that 6 of 69 patients (8.7%) with sleep disorders screened for FES were treated for glaucoma. Unfortunately, they did not verify which of the glaucoma patients had OSA.

The aim of the present study was to determine whether the presence of FES is associated with a higher prevalence of glaucoma in OSA patients in order to determine whether FES could be an indicator of glaucoma in patients with OSA.

## MATERIALS AND METHODS

The study design was cross-sectional. The period for patient inclusion was September 2008 to December 2011. We included 152 patients who had been admitted for OSA evaluation at the Arnau de Vilanova University Hospital and Santa Maria Hospital, Lleida, Spain and who agreed to undergo an ophthalmologic examination to diagnose glaucoma. Seventy-five patients had OSA without FES; 52 patients had OSA and FES; and 25 patients did not have OSA. Two of the non-OSA patients had FES. All the patients were subjected to a complete ophthalmologic examination to diagnose glaucoma. They were examined by a glaucoma expert (MaJ.M.R.), who was blinded to the results of the sleep study, according to standard diagnostic criteria.

None of the patients included had undergone any form of surgical intervention to the eyelid or had suffered from any type of ocular trauma or abnormality that could have affected eyelid function, nor did any of them have a history of ocular surgery in the previous 12 months. Patients with known neurological or psychiatric disorders were not included in the study. The protocol and informed consent were both approved by the Ethics Committee of our hospital, and informed consent was obtained in all cases.

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## Sleep Studies

Diagnosis of OSA was made on the basis of either conventional polysomnography or a cardiorespiratory sleep study. All the sleep studies were individually analyzed at each participating center by applying standard criteria.[17] The polysomnographies included the continuous recording of neurological variables: electroencephalogram (C3/A2 and C4/A1), electrooculogram, and electromyogram. Breathing variables were scored according to a flow tracing provided by a nasal cannula and thermistor. Thoracoabdominal motion was measured with thoracic and abdominal bands. Oxygen saturation was recorded with a finger-pulse oximeter. The cardiorespiratory sleep study included (as a minimum): a continuous recording from the nasal cannula, thoracoabdominal motion, oxygen saturation, and body position. Apnea was defined as an absence of airflow for at least 10 seconds, and hypopnea was defined as a clear (50%) airflow reduction for at least 10 seconds, with a drop in oxygen saturation of at least 4% on arousal. OSA was defined as the absence of airflow in the presence of chest or abdominal wall motion. The apnea-hypopnoea index (AHI) was calculated according to the average number of episodes of apnea plus hypopnea per hour of sleep or recording time. Sleep stages were scored as: normal, $AHI < 10^{-1}$; mild OSA, $10^{-1} \geq AHI < 20^{-1}$; moderate OSA, $20^{-1} \geq AHI < 30^{-1}$; and severe OSA, $AHI \geq 30^{-1}$.[17]

## Ophthalmic Examination

Eyelid examination was specifically conducted to evaluate eyelid laxity and to obtain an FES diagnosis. FES was defined as easily evertible lids and the presence of papillary conjunctivitis in the same upper eyelid, which is the clinical definition of FES.[1,2] Easy lid eversion was characterized by increased laxity in the upper lids that became easily distorted and everted with only minimal superolateral traction.

The eye examination included: best-corrected visual acuity with a recording of the refractive correction; slit-lamp biomicroscopy of the anterior segment; Goldmann applanation tonometry performed with the same tonometer; ultrasonic corneal pachymetry; gonioscopy; and visual field analysis with the Humphrey Field Analyzer (SITA-standard program, central 24-2 threshold test). Visual field tests were repeated 1 week later if significant fixation losses and/or false positives or negatives were detected to be >15%.[18] Patients with OSA also often tend to exhibit losses of attention during these tests. We also repeated the visual field test 4 months later in the case of patients suspected of suffering from glaucoma. After pupil dilatation, the morphology of the optic disc was assessed by stereoscopic slit-lamp biomicroscopy, using a fundus 3-mirror lens. The thickness of the retinal nerve fiber layer (RNFL) was assessed using optical coherence tomography (Stratus OCTm Carl Zeiss Ophthalmic Systems Inc.).

The following criteria were used to define glaucoma in our study, irrespective of the intraocular pressure (IOP): a normal-appearing anterior chamber angle on gonioscopy; glaucomatous optic disc damage with asymmetric cupping and thinning of the neuroretinal rim and/or optic disc hemorrhage and/or defects in RNFL thickness; glaucomatous visual field defects (Bjerrum and/or paracentral scotoma, nasal step, and altitudinal defects); and the progression of optic nerve damage and visual field defects. For the diagnosis of primary open-angle glaucoma (POAG), untreated IOPs had to remain at above 21 mm Hg and below 22 mm Hg for the diagnosis of NTG. If the patients had been diagnosed with glaucoma before the present study, we considered that they had previously diagnosed glaucoma (PDG).

## Statistical Analysis

All the data were expressed as mean ± SD. Statistical analyses of the demographic data, polysomnographic recordings, and ophthalmologic examinations were performed using the unpaired Student *t* test. The prevalence of glaucoma was calculated from the proportion of patients with evidence of glaucoma. We used the Mann-Whitney test to analyze data between groups. Right and left eyes were analyzed separately. The 95% confidence interval was used to compare the prevalence of glaucoma in our OSA patients with the prevalence of glaucoma in OSA reported in previous publications. Spearman Rank correlation was also used whenever appropriate. Each correlation was controlled for age and body mass index (BMI). $P < 0.05$ was considered statistically significant. The language and environment for the R Foundation for Statistical Computing (Vienna, Austria) was also used.

# RESULTS

The demographic data for the 152 patients included in the study are presented in Table 1 and divided according to the presence of OSA and FES.

The prevalence of glaucoma in the 127 patients with OSA, including 75 patients without FES and 52 patients with FES, was 12.9%. The prevalence of glaucoma in the OSA patients without FES was 5.3% (4/75) with a 95% confidence interval of 1.5%-13.1%. One patient had POAG

**TABLE 1.** Categorization of the Patients Included in the Study on the Basis of Reported Clinical Findings

| | OSA Patients Without FES | OSA Patients With FES | *P* | Non-OSA Patients |
|---|---|---|---|---|
| N | 75 | 52 | | 25 |
| Age | 54.3 ± 9.25 | 63.66 ± 9.01 | 0.000 | 48.6 ± 11.15 |
| Sex | 76% | 84.6% | 0.145 | 72% |
| BMI | 31.4 ± 4.54 | 34.36 ± 6.03 | 0.011 | 29.2 ± 4.93 |
| AHI | 42.21 ± 23.6 | 45.73 ± 24.04 | 0.348 | 4.10 ± 1.61 |
| Glaucoma patients | 4/75 (5.33%) | 12/52 (23.07%) | 0.004 | 0% |
| | | | 0.04* | |

*After adjusting for age and BMI.
AHI indicates apnea-hypopnea index; BMI, body mass index (kg/m$^2$); FES, floppy eyelid syndrome; OSA, obstructive sleep apnea.

**TABLE 2.** Comparison Between the Prevalence of Glaucoma in OSA Patients According to the Presence of FES Observed in the Present Study and Those Previously Reported

| Prevalence of Glaucoma in the Present Study | Prevalence of Glaucoma in OSA Patients Published Previously | *P* |
|---|---|---|
| OSA patients without FES 5.3% (CI, 1.5%-13.1%) | 3.4%[16] | 0.299 |
| | 5.9%[13] | 0.924 |
| | 5.6%[15] | 0.990 |
| | 5.9%[14] | 0.987 |
| | 7.2%[11] | 0.610 |
| OSA patients with FES 23.07% (CI, 11.2%-34.6%) | 3.4%[16] | < 0.001 |
| | 5.9%[13] | < 0.001 |
| | 5.6%[15] | < 0.001 |
| | 5.9%[14] | < 0.001 |
| | 7.2%[11] | < 0.001 |

CI indicates confidence interval; FES, floppy eyelid syndrome; OSA, obstructive sleep apnea.

and 3 had PDG. The prevalence of glaucoma in patients with OSA and FES (12/52) was 23.07%, with a 95% confidence interval of 11.2%-34.9%. Six patients had NTG, 5 had POAG, and 1 patient had PDG. None of the 25 patients without OSA had glaucoma. A comparison between the prevalence of glaucoma in OSA patients according to the presence of FES observed in the present study and those previously reported is shown in Table 2. The difference in the prevalence of glaucoma between OSA patients without FES (5.3%) and OSA patients with FES (23.07%) was statistical significant ($P = 0.004$). When adjustments were made for age and BMI, this significance remained ($P = 0.04$). Glaucoma was associated with the presence of FES independently of BMI (Table 3). The demographic data and ophthalmologic findings for patients with OSA included in the study divided according to the presence or absence of glaucoma are presented in Table 4.

There was no association between AHI and the presence of glaucoma after adjusting for age and BMI; the adjusted coefficient was −0.079 and the *P*-value was 0.394. The Spearman correlation coefficient failed to show any correlation between the AHI and IOP, central corneal thickness, mean deviation of the visual field, and/or the average thickness of the RNFL (Table 5).

## DISCUSSION

The high prevalence of glaucoma detected in our patients with FES and OSA (23.07%) suggested that FES could be a strong indicator of glaucoma in patients with OSA. The association between OSA and glaucoma has been suggested in several previous reports.[11–16] However, the prevalence of glaucoma in OSA patients with FES as a possible indicator of glaucoma had previously received relatively little formal study[3,7]: McNab[3] studied a smaller sample than the one included in the present study and did not include OSA patients without FES as a control group. Robert et al[7] did not state which of their glaucoma patients had OSA. Other published studies have shown the prevalence of glaucoma in OSA without taking into account the presence of FES in the OSA population. Geyer et al[12] reported that the prevalence of glaucoma in OSA patients was similar to that found in the general white population[19,20]: a 2% prevalence of glaucoma was found in patients with OSA diagnosed by polysomnography (5/228).

**TABLE 3.** Correlational Study Between the Presence of Glaucoma and Age and BMI

| | Presence of Glaucoma |
|---|---|
| Age | |
| Correlation | 0.393 |
| *P* | 0.000 |
| BMI | |
| Correlation | 0.039 |
| *P* | 0.693 |

BMI indicates body mass index (kg/m$^2$).

**TABLE 4.** The Demographic Data and Ophthalmologic Findings for Patients With OSA Divided According to the Presence of Glaucoma

| | OSA Patients Without Glaucoma | OSA Patients With Glaucoma | *P* |
|---|---|---|---|
| N | 111 | 16 | |
| Age (y) | 56.2 ± 9.04 | 67.6 ± 8.30 | 0.001 |
| BMI | 32.31 ± 5.32 | 32.51 ± 4.21 | 0.691 |
| AHI | 44.17 ± 24.31 | 38.20 ± 18.00 | 0.507 |
| VA RE | 0.87 ± 0.15 | 0.80 ± 0.17 | 0.21 |
| VA LE | 0.85 ± 0.17 | 0.70 ± 0.3 | 0.09* |
| IOP RE | 15.41 ± 3.15 | 19.35 ± 5.01 | 0.004* |
| IOP LE | 14.92 ± 2.55 | 19.30 ± 5.34 | 0.000* |
| CCT RE | 544.12 ± 34.06 | 548.83 ± 27.99 | 0.649 |
| CCT LE | 544.09 ± 35.20 | 547.09 ± 29.80 | 0.788 |
| Average RNFL thickness RE | 99.36 ± 11.12 | 79.29 ± 14.74 | 0.000* |
| Average RNFL thickness LE | 96.07 ± 9.23 | 82.51 ± 13.38 | 0.001* |
| MD RE (dB) | −2.072 ± 2.20 | −6.75 ± 4.34 | 0.000* |
| MD LE (dB) | −2.20 ± 2.03 | −5.33 ± 3.48 | 0.003* |
| FES | 40/111 (36%) | 12/16 (75%) | 0.123* |

Data presented as mean ± SD.
*After adjusting for age.
AHI indicates apnea-hypopnea index; BMI, body mass index (kg/m$^2$); CCT, central corneal thickness; FES, floppy eyelid syndrome; IOP, intraocular pressure; LE, left eye; MD, visual field mean deviation; RE, right eye; RNFL, retinal nerve fiber layer; VA, visual acuity.

**TABLE 5.** Spearman Correlations Between Apnea-Hypopnea Index and Ophthalmologic Findings

| AHI Correlation | Adjusted Coefficient for AHI* | *P** |
|---|---|---|
| IOP RE | −0.233 | 0.546 |
| IOP LE | −0.144 | 0.712 |
| CCT RE | −0.487 | 0.184 |
| CCT LE | −0.436 | 0.241 |
| MD (dB) RE | −0.082 | 0.834 |
| MD (dB) LE | 0.277 | 0.470 |
| Average thickness RNFL RE | −0.151 | 0.698 |
| Average thickness RNFL LE | 0.028 | 0.943 |

*All the correlations were controlled for age and body mass index.
AHI indicates apnea-hypopnea index; CCT, central corneal thickness; IOP, intraocular pressure; LE, left eye; MD, mean deviation; RE, right eye; RNFL, retinal nerve fiber layer.

Kadyan et al[16] reported that the prevalence of open-angle glaucoma in OSA patients (3/89, 3.4%) was similar to that in a normal population (2%). In 2007, Sergi et al[13] reported 3 of 51 OSA patients (5.9%) with NTG; this suggested that the prevalence of NTG in OSA patients was higher than would normally be expected in a white population of the same age and that OSA may be an important risk factor for NTG. A recent study by Chambe et al[15] showed a prevalence of glaucoma in OSA patients diagnosed by overnight respiratory polygraphy of 5.6% (5/89). In 2010, Lin et al[14] reported a prevalence of 5.7% of NTG among patients with OSA (12/209) but no glaucoma patients among 38 non-OSA patients. Monjon et al[11] found a 7.2% prevalence of glaucoma among 69 patients with OSA who underwent a polysomnographic evaluation (5/69), which suggested a strong association between glaucoma and OSA. Our study confirmed the previously reported high correlation between glaucoma and OSA, with a 12.9% prevalence of glaucoma in all OSA patients. It is possible than this prevalence was higher than that reported in previous publications because of the large number of FES patients included in the present study. When we excluded the patients with FES, the prevalence of glaucoma in OSA patients was 5.3%, and this prevalence was not significantly different from that of glaucoma in patients with OSA that had been published previously. When we included only OSA patients with FES, the rate of prevalence reached 23%, and this was significantly higher than the prevalence of glaucoma in OSA patients reported in previous publications. These data supported the hypothesis that FES could be an important indicator of glaucoma in OSA patients. It is quite likely that age and BMI are also associated with the presence of glaucoma.[21,22] The observed differences in the prevalence of glaucoma between patients with OSA but without FES and those with both OSA and FES were statistically significant after adjusting for age and BMI. Glaucoma was associated with the presence of FES independently of BMI. Although age was a partial confounding factor in the relation between glaucoma and FES, its effect did not prevent these differences from being statistically significant. These results suggested that FES could be a risk factor for glaucoma in patients with OSA. Further, the presence of glaucoma in patients with OSA in the current study was not related to the severity of OSA defined by their AHI.

Therefore, FES could be useful for identifying individuals with more probability of having glaucoma amongst the OSA population. More studies are necessary to corroborate these results and to study whether FES could be an independent risk factor for glaucoma in patients without OSA.

Owing to the observational character of our study, the results obtained do not allow us to draw any conclusions about direct causal relationships between glaucoma, OSA, and FES.

Several reports have proposed a vascular etiology in glaucoma.[11,13] In patients with OSA, prolonged repetitive periods of apnea during sleep are accompanied by transient hypoxia and increased vascular resistance. It has been suggested that this episodic vascular impairment may compromise optic nerve head perfusion and oxygenation, causing glaucomatous optic neuropathy. The proposed link between OSA and FES has had quite a strong influence on the ischemia-reperfusion theory.[23] Hypoxic ischemia may contribute to optic nerve damage but would not necessarily be expected to do so by increasing IOP. We cannot, therefore, exclude the possibility of another factor also influencing glaucoma in OSA. In obese patients, increases in IOP during positional changes, in which the neck is compressed, have also been observed.[24] Both FES and OSA have been independently associated with obesity. Moreover, it has also been observed that uvula tissue from patients with OSA demonstrates a loss of elastic fibers.[25] Schlötzer-Schrehardt et al[26] reported a pattern of elastic fiber depletion in the tarsal connective tissue in FES. Culbertson and Ostler[2] hypothesized that a subtle form of underlying generalized connective tissue alteration could be responsible for eyelid laxity in FES. It could be possible that the elastic fiber depletion described in FES and OSA could indicate some of the characteristics present in other ocular structures, such as lamina cribosa or/and trabecular meshwork. These changes could increase the risk of glaucoma in OSA patients affected by FES. Further studies are necessary to corroborate this hypothesis.

In conclusion, FES may be a predictive factor for the presence of glaucoma in patients with OSA. Given the high prevalence of glaucoma in patients with OSA and FES observed in our study, we advise screening FES patients for glaucoma.

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