Sleep Breath
DOI 10.1007/s11325-014-0978-9

ORIGINAL ARTICLE

# Changes of visual field and optic nerve fiber layer in patients with OSAS

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Received: 31 January 2014 / Revised: 25 March 2014 / Accepted: 27 March 2014


**Abstract**
*Background* The prevalence of primary open-angle glaucoma (POAG) increases in obstructive sleep apnea syndrome (OSAS). OSAS could increase cerebrospinal fluid pressure (CSFP) and binocular papilledema.
*Methods* In this cross-sectional study, intraocular pressure (IOP), CSFP, mean deviation (MD), pattern standard deviation (PSD), optic disc indices, and retinal nerve fiber layer (RNFL) were compared among four groups with different extents of OSAS. Regression analysis was performed to correlate MD, PSD, and RNFL to polysomnography (PSG) index. For subgroups with severe OSAS, IOP and CSFP were compared. The prevalence of POAG was calculated.
*Results* The severe OSAS had a significantly higher CSFP than the other three groups ($p$=0.002, 0.036, and 0.017). Both moderate and severe groups showed significantly higher IOP than control group ($p$=0.022 and 0.001). MD was correlated with average oxygen saturation (MSaO2) ($p$=0.001). PSD was correlated with oxygen desaturation index (ODI) ($p$=0.004). Significant differences were found in nasal RNFL and inferior RNFL among the four groups ($p$=0.013, $p$=0.004). Nasal RNFL correlated with the ODI ($p$=0.048). For severe group, compared to normal RNFL group, CSFP was significantly lower in the thinned RNFL group ($p$=0.039) and higher in the thickened RNFL group ($p$=0.034). Totally, the prevalence of POAG was 5.49 %.
*Conclusions* OSAS had a high prevalence of POAG. Visual field was damaged and the RNFL was thinned. Due to diverse CSFP, RNFL changed differently in the patients with severe OSAS.

**Keywords** Obstructive sleep apnea-hypopnea syndrome · Retinal nerve fiber layer · Intraocular pressure · Orbital cerebrospinal fluid pressure · Visual field

## Introduction

Obstructive sleep apnea syndrome (OSAS) is one of the most common sleep disorders. It is characterized by recurrent upper airway obstruction, intermittent hypoxia, and symptoms of triggered awakening during sleep and daytime sleepiness. Alternating tissue hypoxia and awakening could stimulate the sympathetic nervous system, thereby causing a series of cascade reactions [1]. Meanwhile, tissue hypoxia could also increase blood pressure and reduce the vascular reaction to vasorelaxation factors, leading to autoregulatory dysfunction of the vascular tissues [2] and endothelial dysfunction [3]. As an independent risk factor, OSAS may increase the incidence of hypertension and cardiovascular and cerebrovascular disease through the above-mentioned mechanisms [4–6]. OSAS may also have some influence on the eye. OSAS is present in 90–100 % of patients with floppy eyelids [7–9], and its prevalence is significantly increased in patients with non-arteritic ischemic optic neuropathy [10]. Further, the prevalence of primary open-angle glaucoma (POAG) is increased in patients with OSAS [11, 12], and OSAS might cause an increase in cerebrospinal fluid pressure (CSFP) and binocular papilledema in these patients [13, 14].

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Published online: 08 May 2014

The aim of this study was to evaluate the effect of OSAS on the visual field and the thickness of the retinal nerve fiber layer (RNFL) and to analyze the relevant factors in such changes. Meanwhile, the prevalence of POAG in patients with OSAS was observed.

## Material and methods

In this study, 135 patients receiving polysomnography (PSG) transferred from the sleep monitoring center to the Department of Ophthalmology from March to August in 2013 were consecutively enrolled. Among them, 32 were normal without OSAS and 103 were newly diagnosed with OSAS. We excluded 16 patients (4 patients without OSAS), including 3 patients who had undergone eye surgery, 2 patients who were unable to complete the ocular examinations because of severe cataract, 2 patients with high myopia, 7 patients with diabetes, and 2 patients with a history of cerebral infarction. Finally, 91 patients with OSAS and 28 patients without OSAS were enrolled in the analysis. The Medical Ethics Committee of the Beijing Anzhen Hospital approved the study protocol. All participants provided written informed consent, according to the Declaration of Helsinki.

*PSG* Every patient was monitored by using a portable sleep monitor (Embletta, PDS model 2601–1 X10Xact Trace/ Embletta, USA); all patients were reminded the cautions during the sleep monitoring by the same technician in the afternoon before the patient underwent sleep monitoring. All monitoring results were scored by the same experienced technicians who were blinded to the basic characteristics of the patients. The apnea/hypopnea index (AHI) of the patients was evaluated by using the American Academy of Sleep Medicine (AASM) standards [15]. The patients were diagnosed and classified according to AHI as follows: those without OSAS (AHI≤5), those with mild OSAS (AHI 5–15), those with moderate OSAS (AHI 15–30), and those with severe OSAS (AHI≥ 30). The following variables were assessed: the average oxygen saturation (MSaO2), which reflects the average blood oxygen saturation of the patient at night; the oxygen desaturation index (ODI), which reflects the frequency in which the arterial oxygen saturation fell more than 4 % in 1 h; and the TS90%, which reflects the percent of the time that the arterial oxygen saturation is less than 90 % of the total sleep time.

Each patient underwent comprehensive ocular examinations that was completed from 9:00 am to 12:00 am. The ocular examinations included the following assessments: diopter, best-corrected visual acuity, reaction of pupil to light, slit-lamp examination, central corneal thickness (CCT), intraocular pressure with Goldmann applanation tonometry, and mydriatic fundus photography. The values of the right eye were selected for statistical analysis.

SITA-standard 30-2 automated perimetry was performed (Humphrey Visual Field Analyzer II; Carl Zeiss Meditec, Inc., Dublin, CA) and was used to examine each patient. If the fixation loss, the false positive rate, and the false negative rate were less than 15 %, the results of the visual field were considered to be credible. The mean deviation (MD) and the pattern standard deviation (PSD) were subsequently recorded and were included in the final statistical analysis. If the test results were incredible, perimetry was performed again after 1 week.

*OCT examination* OCT examination was performed on all patients using 3D OCT-1000 (Topcon Corporation, Japan). The patients were given Mydrin-P before examination and were instructed to gaze at an object prior to scanning in the built-in optic nerve mode. The optic disc area, cup area, rim area, cup:disc ratio (C/D), and RNFL thickness in the four quadrants (superior, nasal, inferior, and temple) were recorded. All examinations were completed by the same examiner who was blinded to the patients' respiratory and sleep states.

*Orbital cerebrospinal fluid pressure (CSFP) speculation* We calculated the CSFP level based on the BMI, diastolic blood pressure, and age of the patient by using the method for speculating CSFP after lumbar puncture, as previously described [16].

The diagnosis for glaucoma is the following. A pathologic optic disk was defined as optic nerve cupping and rim thinning (cup-disk ratio ≥0.6 or cup-disk asymmetry ≥0.3) or partial or complete notching, and/or rim hemorrhages. Pathologic VF results were defined as an abnormal glaucoma hemifield test on at least two fields plus a cluster of three or more points in a location typical for glaucoma, all of which were depressed on the pattern deviation plot at a $P<5$ % level.

Data analysis was conducted by using SPSS software version 19.0 (SPSS, Inc, Chicago, IL, USA). Continuous measurement values were presented as mean±standard deviation (SD) and are expressed as percentages. The differences in measurement categorical data values between groups were determined using one-way ANOVA, and the differences between subgroups were performed with the LSD test. Categorical data were studied by using the $\chi^2$ test. CCT was corrected by using covariance analysis before comparing intraocular pressure (IOP) among groups. Multiple stepwise regression analysis was performed to correlate visual field index and RNFL to PSG index (MSaO2, ODI, TS90%). $P$ values <0.05 were considered statistically significant.

## Results

All 119 OSAS patients included in the study were divided into 4 groups according to the AHI (measured by using the PSG

**Table 1** The basic characteristics and OSAS-related index of the patients

| | Control | Mild | Moderate | Severe | *P* value |
|---|---|---|---|---|---|
| Eye (*n*) | 28 | 24 | 31 | 36 | |
| Age (years) | 55.4±11.6 | 54.5±10.1 | 57.3±12.8 | 50.8±13.3 | 0.172 |
| Male (%) | 58.3 | 71.0 | 80.6 | 71.4 | 0.322 |
| IOP (mmHg) | 14.4±2.4 | 15.8±2.3 | 16.1±2.9 | 16.7±3.0 | 0.010* |
| CCT (μm) | 0.510±0.024 | 0.486±0.111 | 0.514±0.031 | 0.510±0.057 | 0.075 |
| Diopter (D) | 0.21±1.07 | −0.39±1.19 | −0.17±1.33 | −0.28±1.29 | 0.896 |
| HBP (%) | 50.0 | 46.2 | 62.0 | 54.2 | 0.229 |
| AHI | 1.4±1.0 | 9.8±3.0 | 25.6±6.2 | 49.7±16.7 | <0.001** |
| MSaO2 (%) | 97.8±0.5 | 95.4±1.2 | 95.3±1.3 | 92.3±2.5 | <0.001** |
| BMI (kg/m$^2$) | 26.1±2.0 | 27.3±4.3 | 28.4±3.5 | 29.7±4.1 | 0.02* |
| CSFP (mmHg) | 14.7±3.2 | 15.5±3.2 | 15.4±3. | 17.7±4.7 | 0.012* |

* indicates $p<0.05$; **indicates $p<0.01$

test): control group, 28 patients (AHI≤5); mild group, 24 patients (AHI 5–15); moderate group, 31 patients (AHI 15–30); and severe group, 36 patients (AHI≥30). The basic characteristics and the OSAS-related index of the patients are shown in Table 1. No significant differences in age, sex ratio, diopter, and CCT were found among groups. However, a significant difference was found in the BMI among groups ($p=0.02$). The BMI in the moderate and severe OSAS groups were significantly higher than that in the control group ($p=0.014$ and $p<0.001$, respectively), while the BMI in the severe OSAS group was significantly higher than that in the mild OSAS group ($p=0.014$). Further, a significant difference was observed in the orbital CSFP among the groups ($p=0.012$), with the severe OSAS group having a significantly higher CSFP than the other three groups ($p=0.002$, 0.036, and 0.017). The intraocular pressure (IOP) was also significantly different among the groups ($p=0.010$), with the moderate and severe groups showing a significantly higher IOP than the control group ($p=0.022$ and 0.001). However, IOP was not significantly different between the moderate and severe OSAS groups ($p=0.313$). No significant correlation was found between the IOP and the AHI ($p=0.05$).

*Analysis of visual field* The MD and PSD of the visual field in each group are listed in Table 2. The MD and PSD were significantly different among the groups ($p=0.047$ and 0.007). No significant difference was found in the MD between the control group and the mild OSAS group or between the control and moderate OSAS groups ($p=0.163$ and 0.576).

**Table 2** MD and PSD (db)

| | Control | Mild | Moderate | Severe | *P* value |
|---|---|---|---|---|---|
| MD | −1.57±1.58 | −3.01±3.46 | −2.11±1.65 | −4.02±5.69 | 0.047 |
| PSD | 1.99±0.54 | 2.69±1.89 | 2.31±1.01 | 3.63±3.04 | 0.007 |

However, MD was significantly lower in the severe OSAS group than in the control and moderate OSAS groups ($p=0.009$ and 0.036) but it was not statistically different between the severe and mild OSAS groups ($p=0.298$). The severe OSAS group had a significantly higher PSD than the control group and the mild OSAS group ($p<0.001$ and 0.041), while no significant difference was found in the PSD between the severe and moderate OSAS groups ($p=0.135$). The PSD was significantly higher in the moderate OSAS group than in the control group ($p=0.039$), but the PSD in the moderate group was not significantly different compared with the mild OSAS group ($p=0.517$). A significant negative correlation between the AHI and MD ($r=-0.206$, $p=0.024$) and a significant positive correlation between the PSD and AHI ($r=0.323$, $p<0.001$) were found. Multiple stepwise regression analysis was performed for the MD and PSD with the MSaO2 and ODI. The analysis revealed that the MD was significantly correlated with the MSaO2 ($R^2=0.152$, $p=0.001$), but not with the ODI ($p=0.521$), and that the PSD was significantly correlated with the ODI ($R^2=0.113$, $p=0.004$), but not with the MSaO2 ($p=0.902$).

*RNFL thickness* The optic nerve-related indicators in each group were listed in Table 3. Significant differences were found in the nasal RNFL and the inferior RNFL among the groups ($p=0.013$, $p=0.004$). Compared to the control group, the nasal RNFL thickness in the mild, moderate, and severe OSAS groups were significantly thinner ($p=0.011$, 0.009, and 0004), while no significant difference was found between each two groups of patients with OSAS ($p=0.936$, 0.912, and 0.836). The thickness of the nasal RNFL was not significantly correlated with either the AHI or MSaO2 ($p=0.086$ and 0.717), but was significantly correlated with the ODI ($p=0.048$). The thickness of the inferior RNFL in the mild and moderate OSAS groups were significantly thinner than that in the control group ($p=0.049$, $p<0.001$); in the severe OSAS group, this value was not significantly different from that in

**Table 3** Optic nerve-related indicators

| | Control | Mild | Moderate | Severe | *P* value |
|---|---|---|---|---|---|
| Disc area | 2.52±0.42 | 2.36±0.49 | 2.43±0.42 | 2.64±0.48 | 0.106 |
| Cup area | 1.52±0.31 | 0.79±0.62 | 0.69±0.49 | 0.71±0.43 | 0.374 |
| Rim area | 1.52±0.31 | 1.61±0.57 | 1.73±0.36 | 1.88±0.48 | 0.009** |
| C/D | 0.33±0.14 | 0.24±0.17 | 0.28±0.16 | 0.28±0.15 | 0.292 |
| Superior RNFL | 125±12 | 115±18 | 120±22 | 117±21 | 0.165 |
| Nasal RNFL | 91±11 | 80±18 | 80±16 | 79±17 | 0.013* |
| Inferior RNFL | 137±8 | 127±19 | 119±23 | 128±22 | 0.004** |
| Temple RNFL | 81±13 | 78±18 | 80±17 | 82±12 | 0.798 |

* indicates $p<0.05$; **indicates $p<0.01$

the control group ($p=0.055$), but it was obviously thicker than that in the moderate OSAS group ($p=0.047$).

*CSFP distribution of severe OSAS patients* The severe OSAS patients were divided into 3 groups according to changes in the inferior RNFL: normal RNFL group ($n=16$), thinned RNFL group ($n=7$), and thickened RNFL group ($n=13$). The basic characteristics of the subgroup patients with severe OSAS are shown in Table 4. No significant difference was found in BMI ($p=0.053$) and IOP ($p=0.330$) among the three groups, whereas the CSFP was significantly different ($p=0.002$). Compared to the normal RNFL group, the CSFP was significantly lower in the thinned RNFL group ($p=0.039$) and was significantly higher in the thickened RNFL group ($p=0.034$).

*Prevalence of POAG* POAG was found in five patients (5.49 %). The basic information of the OSAS patients combined with POAG is listed in Table 5.

## Discussion

The prevalence of POAG is reported to be 2 to 47.6 % [9, 11, 17, 18]. Although Girkin et al. [19] did not find that OSAS was an independent risk factor for POAG after correcting for diabetes, high blood pressure, and other confounding factors, their study may have been affected by information bias because it was a large-scale retrospective study in which the patients were divided into groups based only on the disease code. Our study revealed that the prevalence of POAG in OSAS patients (5.49 %) is far higher than that in the normal Chinese population (1.2 %) [20] and is similar to the reported prevalence of POAG in Taiwanese patients with OSAS (5.7 %) by Lin PW et al. [12]. After CCT correction, the intraocular pressure of OSAS patients still showed a significant increase in this study. Although the correlation between the intraocular pressure and AHI did not reach statistical difference, the intraocular pressure showed a notable tendency to be increased in moderate and severe OSAS patients, which is similar to the results previously reported by Moghimi et al. [21]. In contrast, Karakucuk et al. did not observe increased intraocular pressure in OSAS patients [17]. This may be because the CCT-induced error of the intraocular pressure measurements could not be excluded in the study by Karakucuk et al. because the CCT was not measured.

Meanwhile, the MD of OSAS patients was significantly lower than that of the control group, while the PSD values were significantly higher than those in the control group. Moreover, the MD was significantly correlated with both the

**Table 4** The basic characteristics of the subgroup patients with severe OSAS

| | Normal | Thinned group | Thickened group | *P* value |
|---|---|---|---|---|
| Eye (*n*) | 16 | 7 | 13 | |
| Age (years) | 50.5±13.7 | 60.7±16.1 | 45.9±8.0 | 0.052 |
| Male (%) | 81.3 | 92.3 | 71.4 | 1.000 |
| IOP (mmHg) | 17.5±3.1 | 16.9±3.1 | 15.8±2.8 | 0.330 |
| CCT (μm) | 0.530±0.035 | 0.504±0.031 | 0.532±0.023 | 0.211 |
| Diopter (D) | −0.50±1.38 | −0.39±1.45 | −0.21±0.91 | 0.824 |
| HBP (%) | 50.0 | 62.0 | 54.2 | 1.000 |
| AHI | 44.5±25.1 | 44.8±22.8 | 49.0±16.6 | 0.856 |
| MSaO2 (%) | 90.8±8.9 | 90.6±5.4 | 92.0±2.6 | 0.873 |
| BMI (kg/m$^2$) | 29.5±4.6 | 26.9±1.8 | 31.5±3.8 | 0.053 |
| CSFP (mmHg) | 17.2±4.5 | 13.3±2.2 | 20.5±4.1 | 0.002** |

* indicates $p<0.05$; **indicates $p<0.01$

**Table 5** Basic information of the OSAS patients combined with POAG

| | Sex | Age | IOP | CSFP | OSAS group |
|---|---|---|---|---|---|
| Patient 1 | Male | 73 | 18/17 | 11.05 | Severe |
| Patient 2 | Male | 41 | 20/21 | 25.18 | Severe |
| Patient 3 | Male | 79 | 14/15 | 9.45 | Severe |
| Patient 4 | Female | 52 | 16/12 | 15.43 | Moderate |
| Patient 5 | Male | 65 | 19/18 | 12.92 | Mild |

AHI and $MSaO_2$, while the PSD was significantly correlated with both the AHI and ODI. Therefore, we speculate that the level of average visual field impairment is related to the average nocturnal blood oxygen level and that the level of partial visual field impairment is related to the sharp fluctuation frequency of nocturnal blood oxygen in OSAS patients. These findings are similar to those reported by Tsang et.al [22]. In contrast, Casas et al. [23] reported that moderate and severe OSAS patients show a significant change only in the MD and not in the PSD. However, because the age-matched population selected as the control group did not receive PSG monitoring in their study, the OSAS condition was not definitely determined, which could have had an effect on the results.

Regarding the optic nerve structure, the nasal RNFL thickness of the OSAS patients was significantly thinner than control people. Furthermore, the thinning extent of the nasal RNFL was significantly correlated only with the ODI, i.e., the extent of damage of the nasal nerve fiber layer was related to the sharp fluctuation frequency of the average nocturnal blood oxygen level in OSAS patients. This is similar to the results reported by Casas et al. [23]. However, Lin et al. [12] found that both the average and the superior RNFLs of severe OSAS patients were significantly thinner than were those in the control and mild OSAS groups.

The average, superior, inferior, and temporal RNFLs were significantly thinner in patients with moderate to severe OSAS compared to both control and mild OSAS patients. Intriguingly, in this study, we found that the inferior RNFL was thinner in patients with mild to moderate OSAS, whereas the value did not show a significant change in the severe OSAS and control groups. Some OSAS patients may have concomitant increased CSFP and papilledema [13, 14]. Lee [14] speculated that OSAS causes an increase in CSFP through the following mechanisms: (a) an intermittent hypoxic episode induces abnormal vascular autoregulation, which damages the optic nerve; (b) OSAS may induce hypertension and arteriosclerosis, thereby further damaging the vascular autoregulation function of the optic nerve; (c) optic nerve injury is induced by long-term hypoxia; and (d) abnormal optic nerve vascular autoregulation is caused by an OSAS-induced balance disturbance between the vasorelaxation factors and vasoconstriction factors of the patients. Regarding the pathogenesis of glaucoma, Wang Ningli recently proposed the concept of the trans-laminar cribrosa pressure difference and suggested that optic nerve fiber layer damage is caused by the disturbance in the pressure balance between the two sides of the lamina cribrosa (LC). Although the intraocular pressure is within the normal range statistically, especially for glaucoma patients with normal intraocular pressure, the pressure posterior to the LC drops (CSFP is decreased), thus damaging the pressure balance between the two sides and causing continuous backward pressure to be exerted on the LC. Finally, a change in the LC structure and apoptosis of nerve fibers passing through the LC pore were shown to induce glaucoma [24]. Therefore, it is hypothesized that different RNFL changes may be caused by the different CSFP conditions of severe OSAS patients. In this study, severe OSAS patients were divided into the following groups according to the extent of RNFL changes: no RNFL change, thinned RNFL, and thickened RNFL. Although BMI values have been shown to be positively correlated with CSFP [25, 26], no significant difference was found in the BMI among the three groups and the intraocular pressure levels were similar in the three groups. The CSFP in the thinned RNFL group was significantly lower than that of the normal RNFL group, whereas the CSFP was significantly higher in the thickened RNFL group than in the normal RNFL group. In contrast, increased CSFP had an opposite effect on the optic nerve, resulting in subclinical thickening of the RNFL. From another perspective, our results confirmed the theory of the trans-laminar cribrosa pressure difference proposed by Wang Ningli. Although the extent of hypoxia is the same in severe OSAS patients, different CSFPs cause a disturbance in the balance between the intraocular pressure and CSFP. The RNFL is thicker when the CSFP exceeds intraocular pressure and vice versa.

In summary, changes in intraocular pressure, CSFP, and the optic nerve blood supply as well as visual field and RNFL changes are caused by the condition of long-term intermittent hypoxia and sympathetic adjustment disorder in OSAS patients. Joint treatment with positive pressure ventilation and weight loss is proposed for overweight patients with severe OSAS because CSFP changes may result in further RNFL damage if weight loss is not combined with positive pressure ventilation and other methods for hypoxia relief.

The major shortcoming of this study is the limited sample of patients. Further, the present standard for the CSFP measurement is lumbar puncture, and several risks are associated with this invasive procedure. Therefore, only the previously inferred formula based on the Chinese population was used to evaluate the CSFP level [16]. In addition, this study had a selection bias because the subjects were undergoing sleep monitoring; thus, the results may not represent the condition of all OSAS patients. Finally, the exact timing of the development of OSAS

was not known, and this course may affect the changes in the patient's eye.

**Conflicts of interest** No authors have conflict of interest in this study.

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