Original Article

Prevalence of Primary Open-Angle Glaucoma in Patients with Obstructive Sleep Apnea Syndrome in Thailand

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Objective: To determine the prevalence of primary open-angle glaucoma (POAG) among patients with obstructive sleep apnea syndrome and to identify factors associated with intraocular pressure (IOP).

Materials and Methods: Eighty-eight patients with obstructive sleep apnea (OSA) and 88 control subjects were enrolled. All subjects underwent eye examination including visual acuity, IOP, gonioscopy, optic disc, and visual field evaluation within one week of enrollment.

Results: Three of the 88 patients with OSA (3.4%) and only one (1.1%) in the matched group had POAG, indicating no statistically significant difference in both groups (p=0.621). All patients with OSA diagnosed with POAG had severe OSA. OSA did not significantly influence the risk factor of POAG (OR 3.07, 95% CI 0.31 to 30.11, p=0.312). However, the presence of mean oxygen saturation of less than 88% or oxygen desaturation index (ODI) greater than 30 events per hour correlated with an elevation of IOP (p=0.026, 0.010). Based on apnea-hypopnea index (AHI) and ODI, there was medium correlation with IOP (Pearson correlation 0.342, 95% CI 0.143 to 0.5147, p=0.0011 and Pearson correlation 0.317, 95% CI 0.116 to 0.494, p=0.0025).

Conclusion: The prevalence of POAG was 3.4% in patients with OSA when compared with 1.1% in control group. There was positive correlation between AHI or ODI with the IOP. Moreover, the presence of mean oxygen saturation of less than 88% or ODI of more than 30 events per hour correlated with an elevated IOP.

Keywords: Glaucoma, Obstructive sleep apnea, Prevalence, Primary open-angle glaucoma, Sleep apnea

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Primary open-angle glaucoma (POAG) is a multifactorial optic neuropathy that affects 1.7% to 7.0% of the population[1-4]. The prevalence of POAG in Thai population is estimated to be approximately 6% with age above 50 years[5]. It is characterized by progressive retinal ganglion cell death and associated glaucomatous visual field loss. The various factors that may be involved in the etiology of POAG include abnormal ocular blood flow, vasospasm, systemic hypotension, abnormal coagulopathy, cardiac and cerebral diseases, and autoimmune diseases[6].

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete collapse of the upper airway during sleeping. The reduction of airflow leads to hypoxia and hypercapnia, which induces oxidative stress and consequently promotes the systemic inflammation, sympathetic activation, and endothelial dysfunction. It has been proposed that these effects are associated with multiple cardiovascular consequences including hypertension, coronary artery disease, stroke, pulmonary hypertension, and congestive heart failure[7-9]. Moreover, OSA typically causes cyclic intermittent hypoxemia and arousal that result in sleep fragmentation and poor sleep quality. Patients often have excessive daytime sleepiness that increases the risks of work or vehicle accidents. The prevalence of OSA worldwide is estimated to be 9.0% to 24.0% in middle-aged western population and 6.3% to 15.4% in Thai population[10,11]. The prevalence typically increases with ages.

OSA is one of the common causes of hypoxemia, which may compromise optic nerve perfusion and oxygenation, and subsequently cause optic neuropathy. Previous studies have demonstrated OSA to be associated with eye diseases, such as

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Table 1. Inclusion and exclusion criteria for patient eligibility

Inclusion criteria	Exclusion criteria
OSAS subjects - Age 30 to 70 years, and newly diagnosed OSAS(23)	- Unable to perform an accurate IOP measurement - Unable to produce a reliable VF test - Known allergy to any topical anesthesia or fluorescein - Previous intraocular surgery - Active ocular infection or inflammation
Control subjects - Matched age ±2 years - Same gender - Similar comorbidities including type II diabetes, hypertension, myopia, migraine, peripheral vascular disease - No OSA symptoms and ESS score of <10	

OSAS=obstructive sleep apnea syndrome; ESS=Epworth Sleepiness Scale; IOP=intraocular pressure; VF=visual field

floppy eyelid syndrome and non-arteritic ischemic optic neuropathy[12]. Several studies have reported an increased prevalence of glaucoma among OSA patients[13-15] and adequate treatment of OSA will also have a better control glaucoma[16]. In contrast, some study reported that patients with OSA do not have higher risks of glaucoma compared with patients without OSA[17]. However, the risk of POAG among OSA patients remains a controversial issue[18]. The aims of the present study were to determine the prevalence of POAG among OSA patients and to identify factors associated with intraocular pressure (IOP).

Materials and Methods

Study design

The present study design was a cross-sectional study between August 2015 and May 2019. The OSA-hypopnea syndrome patients were selected from the sleep clinic of the Otolaryngology Outpatient Department, at Songklanagarind Hospital. The control group were randomly selected from the normal population who did not have the symptoms suspected OSA and the Epworth Sleepiness Scale score of less than 10[19,20]. All recruited subjects met the inclusion and exclusion criteria that are summarized in Table 1. Participants were sent for detailed ophthalmic examinations within one week after enrollment at the eye clinic, Songklanagarind Hospital. The study protocol was approved by the Human Research Ethics Committee, Faculty of Medicine, Prince of Songkla University, and an informed consent was obtained from each patient.

Sleep studies

The diagnosis of OSA was made by an attended type I polysomnography (Compumedics E series, Compumedics Ltd., Abbotsford, Australia). It comprised of electroencephalography, electrooculography, chin and leg electromyography, electrocardiography, thermistors, and nasal pressure transducer for oronasal airflow, thoracic and abdominal belts for respiratory efforts, pulse oximetry for oxyhemoglobin level, tracheal microphone for snoring, and sensors for sleeping position. Additionally, these parameters' recordings were scored manually by using standard criteria[21]. Apnea-hypopnea index (AHI) was used to determine the severity of OSA, as follow, mild for 5 to less than 15, moderate for 15 to 30, and severe for more than 30[22,23].

Ophthalmologic examination

The ophthalmic examinations were performed by a single experienced glaucoma specialist (Kiddee W). Recruited patients underwent baseline assessment of best-corrected visual acuity (BCVA), IOP measured by calibrated Goldmann applanation tonometer, gonioscopy, dilated fundoscopy with a 78-diopter lens, and Humphrey 24-2 perimetry (Humphrey visual field, Carl Zeiss Meditecinc., Dublin, CA).

A single drop of tetracaine hydrochloride 0.5% (Alcon Laboratories Fort Worth, TX) was used before the IOP measurement. Three IOP measurements were performed on each eye, and the averaged values were used for the analyses. All IOP measurements were done in the morning between 8 a.m. and 9 a.m.. Glaucoma was diagnosed with the typical glaucomatous optic neuropathy, the correlated glaucomatous visual field defects, open-angle on the gonioscopy, regardless of IOP level[24-26].

Statistical analysis

The prevalence was calculated from the proportion of patients with evidence of glaucoma. Values are presented as mean and standard deviations (SD) and percentages. Statistical significance was set as p-value less than 0.05. The 95% confidence

Table 2. Characteristics of the study population

Variables	OSAS (n=88); n (%)	Control (n=88); n (%)	p-value
Age (year); mean±SD	51.3±11.0	51±10.8	0.868
Sex			1.000
Male	55 (62.5)	55 (62.5)	
Female	33 (37.5)	33 (37.5)	
Comorbidities			0.613
Hypertension	30 (34.1)	30 (34.1)	
Diabetes	3 (3.4)	3 (3.4)	
Myopia	1 (1.1)	0 (0.0)	
Allergic rhinitis	7 (8.0)	4 (4.5)	
Asthma	3 (3.4)	2 (2.3)	
Dyslipidemia	8 (9.1)	7 (8.0)	
Other	6 (6.8)	2 (2.3)	
Frequent Alcohol drinking	40 (45.5)	45 (51.1)	0.546
Daily Smoking	8 (9.1)	19 (21.6)	0.036
ESS scores; median (IQR)	9 (6, 12)	5 (3, 7)	<0.001
BMI (kg/m^2); mean±SD	29.3±6.8	23.7±3.5	<0.001
Neck circumference (cm); mean±SD	38.5±5.6	34±4.9	<0.001
Waist circumference (cm); mean±SD	98.2±16.0	83±12.1	<0.001
Systolic BP (mmHg); mean±SD	134.0±15.5	138.9±10.8	0.675
Diastolic BP (mmHg); mean±SD	80.8±12.5	78.3±12.4	0.199

ESS=Epworth sleepiness scale; BMI=body mass index; IQR=interquartile range; SD=standard deviation; OSAS=obstructive sleep apnea syndrome; BP=blood pressure; kg/m^2=kilogram per square meter; mmHg=millimeters of mercury; cm=centimeter

interval (CI) was used to compare the prevalence of glaucoma in our patients with OSA with the expected in the general population. The factors between groups were compared by using Mann-Whitney U test for ordinal variables and using unpaired t-test for continuous variables. Fisher's exact test was used to compare the dichotomized variables of POAG diagnosis. Statistical analysis was carried out by using R software (version 3.6.2).

Results

One hundred seventy-six patients entered and completed the present study. Mean age, gender, and comorbidities were similar in both groups. The mean body mass index (BMI) was significantly higher in OSA group with 29.3±6.8 kilogram per square meter (kg/m²) compared with control group 23.7±3.5 kg/m². In addition, neck circumference and waist circumference were higher in the OSA group. The systolic and diastolic pressures were similar between groups (Table 2).

The prevalence of POAG in obstructive sleep apnea syndrome (OSAS) patients was 3.4% (3 in 88). Only 1.1% (1 in 88) of control subjects had POAG (Table 3). There was no association between the presence of POAG and OSA (odd ratio 3.07, CI 0.31 to 30.11, p=0.312) in univariate analysis. After adjusting confounders including smoking, BMI, and neck circumference, patients with OSA did not have significant higher odds of POAG with patients who did not have OSA in multivariate analysis (odd ratio 3.88, CI 0.26 to 57.09, p=0.302) (Table 4).

Table 3. Comparison between subjects with and without OSA

	OSAS (n=88); mean±SD	Control (n=88); mean±SD	p-value
BCVA (log MAR)	0.16±0.09	0.12±0.11	0.353
IOP (mmHg)	14.7±2.7	14.9±2.6	0.611
C:D ratio	0.3±0.2	0.3±0.1	0.580
Diagnosis of POAG, n (%)	3 (3.4)	1 (1.1)	0.621

SD=standard deviation; BCVA=best-corrected visual acuity; log MAR=logarithm of minimal angle of resolution; IOP=intraocular pressure; C:D ratio=cup to disc ratio; POAG=primary open-angle glaucoma; OSAS=obstructive sleep apnea syndrome

Table 4. Unadjusted and adjusted odds ratios for POAG among case vs control groups

	Odds ratio (95% confidence interval)	
	Unadjusted	Adjusted for smoking, BMI, and neck circumference
Control	1.0	1.0
OSAS	3.07 (0.31 to 30.11)	3.88 (0.26 to 57.09)
p-value	0.312	0.302

OSAS=obstructive sleep apnea syndrome; BMI=body mass

The three patients in the OSA group who were diagnosed with POAG, had severe OSA. However, the severity of OSA did not show statistically significant influence with glaucoma, or elevation of IOP and CD ratio (Table 5). Moreover, the authors found that the mean oxygen saturation and lowest oxygen saturation were significantly lower in severe OSA than mild or moderate OSA. This is in contrast with oxygen desaturation index (ODI), which was significantly higher in severe group.

Based on AHI and ODI, there were medium correlation with the IOP (Pearson correlation 0.342, 95% CI 0.143 to 0.5147, p=0.0011 and Pearson correlation 0.317, 95% CI 0.116 to 0.494; p=0.0025) (Figure 1). Additionally, the mean oxygen saturation of less than 88% or ODI of more than 30 were the significant factors related with the increasing of IOP (p=0.026, p=0.010) (Table 6).

Table 5. Studied parameters according to the different severity in OSAS groups

	Mild OSAS (n=11); mean±SD	Moderate OSAS (n=24); mean±SD	Severe OSAS (n=53); mean±SD	p-value
BCVA (log MAR)	0.13±0.15	0.17±0.12	0.13±0.13	0.178
IOP (mmHg)	13.7±3.0	14.3±1.9	15.1±2.9	0.235
C:D ratio	0.3±0.1	0.3±0.1	0.3±0.2	0.150
Lowest oxygen saturation (%)	85.7±7.8	80.5±10.2	74.1±12.4	0.002
Mean oxygen saturation (%)	96.1±0.8	94.9±1.7	91.5±5.3	<0.001
ODI (events/hour)	6.2±9.0	15.6±7.7	44.2±28.7	<0.001

SD=standard deviation; OSAS=obstructive sleep apnea syndrome; BCVA=best-corrected visual acuity; log MAR=logarithm of minimal angle of resolution; IOP=intraocular pressure; C:D ratio=cup to disc ratio; ODI=oxygen desaturation index

Table 6. Comparison of eye parameters in OSA patients with mean oxygen saturation (<88 vs ≥ 88%) and oxygen desaturation index (≤ 30 vs >30 events/hour)

	Mean oxygen saturation (%); mean±SD			Oxygen desaturation index (events/hour); mean±SD		
	<88 (n=8)	≥88 (n=80)	p-value	≤30 (n=53)	>30 (n=35)	p-value
BCVA (log MAR)	0.16±0.20	0.10±0.14	0.153	0.15±0.12	0.13±0.11	0.874
IOP (mmHg)	16.6±2.5	14.5±2.6	0.033	14.1±2.1	15.6±3.2	0.010
C:D ratio	0.4±0.1	0.3±0.2	0.167	0.3±0.2	0.3±0.1	0.438

SD=standard deviation; BCVA=best-corrected visual acuity; log MAR=logarithm of minimal angle of resolution; IOP=intraocular pressure; C:D ratio=cup to disc ratio

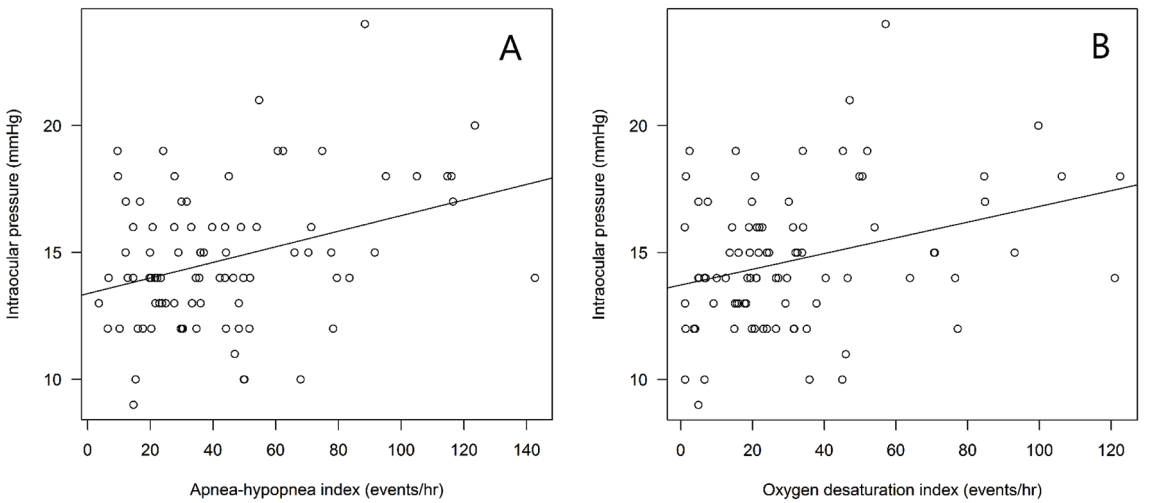

Figure 1. The association between intraocular pressure (IOP) and apnea-hypopnea index (A) that showed the medium relationship (Pearson correlation 0.342, 95% CI 0.143 to 0.5147; p=0.0011), and (B) showed the medium association between IOP and oxygen desaturation index (Pearson correlation 0.317, 95% CI 0.116 to 0.494; p=0.0025).

Discussion

Glaucoma is the second most common cause of unilateral blindness. The prevalence of POAG increases with age in both gender[1-5]. In the present study, the authors defined OSAS patients diagnosed with type I polysomnography using the standard diagnostic criteria and selected matched controls with the same age, gender, and comorbidities for reducing the confounder bias. The author found that there was no significant difference in POAG prevalence between OSAS patients and the matched groups (3.4% versus 1.1%). These findings were similar to Geyer et al that reported 2.0%[18]. However, they contrasted with Mojon et al and Bendel et al, which reported as high as 7.2% to 27.0%[13,14]. Furthermore, these studies could not conclude about a direct causal

relationship between glaucoma and OSA because they could not exclude a third factor influencing both diseases in their studies.

Although the pathophysiologic mechanism of glaucoma is not fully understood, IOP elevation has been proven to be the main cause of glaucoma. Elevated IOP may compromise the retinal ganglion cell. OSA is characterized by the recurrent hypoxia during sleep, which is thought to be one of the mechanisms of optic nerve damage. In the literatures, it remains unclear whether glaucoma is directly associated with OSA. Some study demonstrated that repetitive and prolonged apneas may impair optic nerve blood flow autoregulation and affect the ganglion cell loss[13]. Kargi et al found diffuse reduction of retinal nerve fiber layer (RNFL) thickness in patients with sleep apnea compared with controls, and the severity of OSA strongly correlated with the decrease in RNFL[27]. This finding was supported by the study of Lin et al showed that OSA is associated with an increased risk of subsequent open-angle glaucoma within 5-year period[28]. On the contrary, the present study showed that patients with OSA did not have significant odds of glaucoma when compared with patients without OSA, which was similar to the studies of Aptel et al and Girkin et al[17,29].

Regarding to the severe OSA, the IOP tends to be higher than mild and moderate groups. Moreover, the present study showed that the elevation in both AHI and ODI had medium correlation with the rising of IOP. These were relevant to the study of Mojon et al reporting a positive correlation between respiratory disturbance index (RDI) and the IOP[13]. Recently, the measurement of the number of apnea and hypopnea events per hour, which is called AHI, and RDI as the sum of the AHI and respiratory effort related arousal index, are used to grade OSA. However, AHI or RDI does not always reflect the degree of hypoxemia or the duration of the respiratory events. In which, the severe OSA may not have severe hypoxemia. Thus, it is just a quantitative parameter. The studies of Geyer et al and Bengel et al reported that they did not find any correlation between AHI and IOP[14,18]. The parameters that reflect the severity of hypoxemia have not been studied yet. Interestingly, another parameter used for monitoring the quality of sleep-related breathing events is ODI. ODI is the average number of desaturation episodes that occur within an hour of sleep. Desaturation episodes are generally described as a decrease in the arterial oxygen saturation of 3.0% or less. This parameter reflects the respiratory events associated with desaturation. Moreover, the mean oxygen saturation is defined as the average of blood oxygen during sleep. These parameters typically measured as a part of diagnostic sleep studies, which better reflect the severity of hypoxemia during sleep. Furthermore, the present study found the mean oxygen saturation of less than 88.0% or ODI of more than 30 events per hour had a relationship with the IOP elevation. It might be the good predictors for glaucoma development.

The present study showed that the prevalence of POAG among OSA patients was similar to the normal population and there was no evidence of the association between OSA and POAG. Thus, the routine screening of glaucoma in OSA patients might not be necessary, unless in some OSA patients were having the mean oxygen desaturation of less than 88.0% or ODI of more than 30 events per hour. On the other hand, the screening of OSA in the glaucoma patients may be beneficial in those having significant OSA symptoms. The limitations of the present study were small sample size and the matched control subjects did not have sleep study to rule out a case of asymptomatic OSA as well. Further studies with longer follow-up period after OSA diagnosis are needed to clarify whether the OSA is an etiology of glaucoma.

Conclusion

The prevalence of POAG was 3.4% in OSA patients compared with 1.1% in the control group. The present study did not provide evidence of association between OSA and POAG. However, there was positive correlation between AHI or ODI with the IOP. Additionally, the presence of mean oxygen saturation of less than 88% or ODI of more than 30 events per hour correlated with an elevation of IOP.

What is already known on this topic?

OSA is one of the common causes of hypoxemia, which may compromise optic nerve perfusion and oxygenation, and subsequently cause optic neuropathy. Previous studies have demonstrated OSA to be associated with eye diseases, such as floppy eyelid syndrome and non-arteritic ischemic optic neuropathy. However, the risk of POAG among OSA patients remains controversial.

What this study adds?

The prevalence of POAG in patients with OSA was investigated in 88 OSA patients and 88 matched controls. Although no evidence supported an association between OSA and POAG, OSA screening

in glaucoma patients may be beneficial for those who have significant OSA symptoms because mean oxygen saturation of less than 88% or an ODI of more than 30 events per hour had a relationship with increased IOP, both of which may be good predictors for development of glaucoma.

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Conflicts of interest

The authors declare no conflict of interest.

References

1. Friedman DS, Wolfs RC, O'Colmain BJ, Klein BE, Taylor HR, West S, et al. Prevalence of open-angle glaucoma among adults in the United States. Arch Ophthalmol 2004;122:532-8.
2. Leske MC, Connell AM, Schachat AP, Hyman L. The Barbados Eye Study. Prevalence of open angle glaucoma. Arch Ophthalmol 1994;112:821-9.
3. Wensor MD, McCarty CA, Stanislavsky YL, Livingston PM, Taylor HR. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalmology 1998;105:733-9.
4. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. Ophthalmology 1996;103:1661-9.
5. Bourne RR, Sukudom P, Foster PJ, Tantisevi V, Jitapunkul S, Lee PS, et al. Prevalence of glaucoma in Thailand: a population based survey in Rom Klao District, Bangkok. Br J Ophthalmol 2003;87:1069-74.
6. Moore D, Harris A, Wudunn D, Kheradiya N, Siesky B. Dysfunctional regulation of ocular blood flow: A risk factor for glaucoma? Clin Ophthalmol 2008;2:849-61.
7. Terán-Santos J, Jiménez-Gómez A, Cordero-Guevara J. The association between sleep apnea and the risk of traffic accidents. Cooperative Group Burgos-Santander. N Engl J Med 1999;340:847-51.
8. Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. Sleep 2008;31:1071-8.
9. Marshall NS, Wong KK, Liu PY, Cullen SR, Knuiman MW, Grunstein RR. Sleep apnea as an independent risk factor for all-cause mortality: the Busselton Health Study. Sleep 2008;31:1079-85.
10. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230-5.
11. Neruntarat C, Chantapant S. Prevalence of sleep apnea in HRH Princess Maha Chakri Srinthorn Medical Center, Thailand. Sleep Breath 2011;15:641-8.
12. Huon LK, Liu SY, Camacho M, Guilleminault C. The association between ophthalmologic diseases and obstructive sleep apnea: a systematic review and meta-analysis. Sleep Breath 2016;20:1145-54.
13. Mojon DS, Hess CW, Goldblum D, Fleischhauer J, Koerner F, Bassetti C, et al. High prevalence of glaucoma in patients with sleep apnea syndrome. Ophthalmology 1999;106:1009-12.
14. Bendel RE, Kaplan J, Heckman M, Fredrickson PA, Lin SC. Prevalence of glaucoma in patients with obstructive sleep apnoea--a cross-sectional case-series. Eye (Lond) 2008;22:1105-9.
15. Boyle-Walker M, Semes LP, Clay OJ, Liu L, Fuhr P. Sleep apnea syndrome represents a risk for glaucoma in a veterans' affairs population. ISRN Ophthalmol 2011;2011:920767.
16. Hashim SP, Al Mansouri FA, Farouk M, Al Hashemi AA, Singh R. Prevalence of glaucoma in patients with moderate to severe obstructive sleep apnea: ocular morbidity and outcomes in a 3 year follow-up study. Eye (Lond) 2014;28:1304-9.
17. Aptel F, Chiquet C, Tamisier R, Sapene M, Martin F, Stach B, et al. Association between glaucoma and sleep apnea in a large French multicenter prospective cohort. Sleep Med 2014;15:576-81.
18. Geyer O, Cohen N, Segev E, Rath EZ, Melamud L, Peled R, et al. The prevalence of glaucoma in patients with sleep apnea syndrome: same as in the general population. Am J Ophthalmol 2003;136:1093-6.
19. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14:540-5.
20. Banhiran W, Assanasen P, Nopmaneejumruslers C, Metheetrairut C. Epworth sleepiness scale in obstructive sleep disordered breathing: the reliability and validity of the Thai version. Sleep Breath 2011;15:571-7.
21. Berry RB, Brooks R, Gamaldo CE, Harding SM, Lloyd RM, Quan SF, et al. The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications. Version 2.4. Darien, IL: American Academy of Sleep Medicine; 2017.
22. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K, et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: An American Academy of Sleep Medicine Clinical Practice Guideline. J Clin Sleep Med 2017;13:479-504.
23. American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
24. American Academy of Ophthalmology. Primary open-angle glaucoma suspect: preferred practice pattern

[Internet]. San Francisco: American Academy of Ophthalmology; 2010 [cited 2014 Dec 3]. Available from: https://www.aao.org/ppp.
25. Foster PJ, Buhrmann R, Quigley HA, Johnson GJ. The definition and classification of glaucoma in prevalence surveys. Br J Ophthalmol 2002;86:238-42.
26. Canadian Ophthalmological Society Glaucoma Clinical Practice Guideline Expert Committee. Canadian Ophthalmological Society evidence-based clinical practice guidelines for the management of glaucoma in the adult eye. Can J Ophthalmol 2009;44 Suppl 1:S7-93.
27. Kargi SH, Altin R, Koksal M, Kart L, Cinar F, Ugurbas SH, et al. Retinal nerve fibre layer measurements are reduced in patients with obstructive sleep apnoea syndrome. Eye (Lond) 2005;19:575-9.
28. Lin CC, Hu CC, Ho JD, Chiu HW, Lin HC. Obstructive sleep apnea and increased risk of glaucoma: a population-based matched-cohort study. Ophthalmology 2013;120:1559-64.
29. Girkin CA, McGwin G Jr, McNeal SF, Owsley C. Is there an association between pre-existing sleep apnoea and the development of glaucoma? Br J Ophthalmol 2006;90:679-81.