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The interaction between hypertension and obstructive sleep apnea on subjective daytime sleepiness

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Hypertension is one of the most common chronic cardiovascular diseases in adults while obstructive sleep apnea (OSA) is the most common type of sleep apnea. It was recently reported that the mean Epworth Sleepiness Scale (ESS) score, measuring subjective daytime sleepiness, was significantly higher in non-hypertensive subjects than the hypertensive counterparts with moderate to severe obstructive sleep apnea. In the current study, the authors investigated the interaction between hypertension and OSA on daytime sleepiness among 280 subjects recruited from a sleep study. OSA was evaluated with the Apnea-Hypopnea Index (AHI), and daytime sleepiness was measured with the ESS. Significantly higher mean ESS scores were found for subjects without than those with hypertension (11.3 vs 9.4, $P = 0.003$) but only a marginally significant difference was discerned for the ESS scores between subjects with AHI ≥15/h and AHI <15/h ($P = 0.075$). A significant interaction between hypertension and OSA status on daytime sleepiness was observed from the analysis of variance ($P = 0.02$). The adjusted mean ESS score for the group of normotensive subjects with moderate to severe OSA (13.11) was significantly higher than the other three groups, namely, normotensive subjects with mild OSA (9.35), hypertensive subjects with mild OSA (9.70), and hypertensive subjects with moderate to severe OSA to (9.43). In conclusion, subjective daytime sleepiness of normotensive subjects with moderate to severe OSA was significantly more severe than other subjects.

1 | INTRODUCTION

Hypertension is a condition in which the blood vessels have persistently raised pressure, thus placing them under increased stress.[1] Hypertension is one of the most common chronic cardiovascular diseases in adults.[2] More than one in five adults have raised blood pressure (BP), which underlie half of all deaths from stroke and heart disease.[1] It has recently been reported that, globally, both the numbers of individuals with Systolic BP levels of at least 110 to 115 mm Hg and of those with 140 mm Hg or higher have, alongside the estimated associated deaths, increased over the last 25 years.[3] Currently, complications from hypertension account for 9.4 million deaths worldwide yearly.[1]

Hypertension has been associated with short sleep duration,[4,5] sleep quality,[6] sleep disorder, and other types of sleep problems.[7] Wang et al[5] reported that short sleep durations were associated with elevated risks of not only hypertension in the overall population but also incident hypertension among subjects younger than 65 years. Guo et al[8] further reported that short sleep durations were associated with a higher risk for hypertension even on a longitudinal basis.[8] In addition, Lo et al[6] reported that subjects with hypertension had significantly worse sleep quality scores.

Obstructive sleep apnea (OSA) is the most common type of sleep apnea and is caused by complete or partial obstructions of the upper airway. It is characterized by repetitive episodes of shallow or paused breathing during sleep, despite the effort to breathe, and is usually associated with a reduction in blood oxygen saturation.[9] OSA is one of the major modifiable risk factors of hypertension[10] and has been associated with higher odds of displaying daytime sleepiness, as measured

by the Epworth Sleepiness Scale (ESS).[11] However, there have hitherto been few studies in the literature to examine the inter relationship among daytime sleepiness, hypertension, and OSA. Recently, in their study in Poland, Martynowicz and colleagues[12] reported that the mean ESS score was significantly higher in normotensive subjects than the hypertensive counterparts with moderate to severe OSA, defined as Apnea-Hypopnea Index (AHI) >15. To the best of our knowledge, no studies in Asia have been conducted to examine such differences; therefore, the aim of the current study was to examine the presence of any significant differences in daytime sleepiness between normal subjects and hypertensive ones with moderate to severe OSA.

2 | METHODS

This was an ancillary study of a randomized controlled trial (RCT) regarding the use of the ambulatory approach vs the hospital-based approach in managing clinic patients with suspected OSAS; the data collection was reported elsewhere.[13] OSAS was defined by an AHI ≥5 hours of sleep plus excessive daytime sleepiness or two of the following symptoms: choking or gasping during sleep, recurrent awakenings from sleep, unrefreshed sleep, daytime fatigue and impaired concentration.[14] In brief, patients with new referrals to the Respiratory Clinic of a teaching hospital in Hong Kong were recruited from September 25, 2013, to August 31, 2014.

2.1 | Setting

This study was conducted in a teaching hospital in Hong Kong.

2.2 | Participants

All patients, aged between 18 and 80 with suspected OSAS, who were new referrals to the Respiratory Clinic of the teaching hospital from September 25, 2013, to August 31, 2014, were invited to join the study. They were then randomly assigned to one of the study groups and assessed using AHI, ESS and symptoms including choking or gasping during sleep, recurrent awakenings from sleep, unrefreshed sleep, daytime fatigue and impaired concentration.[13,14] These baseline data were used in the current paper for analysis.

2.3 | Study variables and measures

The ESS was used to measure the subjects' level of subjective daytime sleepiness. This scale consists of eight items that measure a subject's habitual likelihood of dozing or falling asleep in common situations. The ESS total score, ranging from 0 to 24, represents the sum of the scores for the individual items. Scores >10 are considered abnormal. Hypertension and other disease statuses of the subjects were extracted through the hospital database after getting the consents from the subjects. The AHI was measured with either overnight polysomnography (PSG) (Alice LE, Respironics) or the Embletta device (Medcare, Iceland). Anthropometric variables were measured, including the height (cm), weight (kg), neck circumference (cm), waist circumference (cm), and hip circumference (cm). Other variables were also assessed, including minimum the SaO_2 ($minio_2$), mean SaO_2 ($meano_2$), arousal index (arousal), duration of SaO_2 <90% (sao290), percentage of sleep/index time SaO_2 <90% (psao290), oxygen desaturation index (odi), duration of snoring (snore), percentage of sleep/index time snoring (psnore), total sleep time (tst), and sleep efficiency.

For the home sleep tests with the Embletta device which monitored airflow by a nasal pressure transducer, an apnea was defined as a decrease in airflow by 80% of baseline for ≥10 seconds while a hypopnea was defined as a decrease in airflow by 50% of baseline for ≥10 seconds associated with an oxygen desaturation ≥4%. The Embletta™ PDS AHI used for analysis was automatically analyzed by the Embletta™ PDS software with manual editing.[13]

For those who had undergone PSG, apnea was defined as cessation of airflow for >10 seconds with drop in the peak thermal sensor excursion by ≥90% of baseline whereas hypopnea as a reduction of nasal pressure airflow of ≥30% of baseline for >10 seconds plus an oxygen desaturation of ≥4%.[13]

2.4 | Statistical analysis

Descriptive statistics, including the mean, standard deviation, frequency, and percentage, were used to summarize the variables. Pearson's correlation coefficient was used to examine the association between the ESS score and the continuous variables. Independent sample *t* test or one-way analysis of variance (ANOVA) was used to examine the difference in the ESS scores across different groups of the categorical variables.[15] The ESS scores were first compared between hypertensive and normotensive patients using independent sample *t* test. This was followed by stratifying the data according to the different AHI statuses, namely (a) AHI <15/h vs AHI ≥15/h and (b) AHI <30/h vs AHI ≥30/h for comparison using two-way ANOVA with interactions. Multiple linear regression with backward elimination was used to examine the association between the ESS scores and hypertension status after adjusting for the AHI and other potential factors, defined as those with *P*-value <0.1 in correlation or ANOVA analysis. Statistical significance was set at 5%, and all the analyses were conducted using IBM SPSS Statistics 24 (IBM Corporation, Armonk, NY, USA).

2.5 | Ethical consideration

This study was approved by the Ethics Committees of the Chinese University of Hong Kong (CREC-2011.215-T) and registered at ClinicalTrials.gov (identifier: NCT01828216). Written informed consent was obtained from all subjects enrolled in this study.

3 | RESULTS

Initially, a total of 316 subjects were recruited for the study and randomized into the two groups. However, 11 subjects with

TABLE 1 Demographic information of the 280 subjects

	Mean (SD)/n (%)
Age (y)	51.7 (11.9)
Gender	
Female	76 (27.1%)
Male	204 (72.9%)
Height (cm)	164.7 (8.8)
Weight (kg)	74.9 (15.0)
Body mass index (BMI)	27.6 (4.8)
Neck circumference (cm)	39.1 (3.7)
Waist circumference (cm)	96.0 (11.4)
Hip circumference (cm)	101.6 (8.7)
Group	
Home—Embletta	150 (53.6%)
Hospital—PSG	130 (46.4%)
Smoking status	
Non-smoker	200 (71.4%)
Current smoker	40 (14.2%)
Ex-smoker	39 (13.9%)
Drinking	
Non-drinker	151 (53.9%)
Current drinker	5 (1.8%)
Ex-drinker	52 (18.6%)
Apnea-Hypopnea Index (AHI)	
<15	107 (38.2%)
≥15	173 (61.8%)
Congestive heart failure (CHF)	
No	269 (96.1%)
Yes	11 (3.9%)
Diabetes	
No	233 (83.2%)
Yes	47 (16.8%)
Hypertension	
No	144 (51.4%)
Yes	136 (48.6%)
Cerebrovascular disease (CVA)	
No	273 (97.5%)
Yes	7 (2.5%)
Ischemic heart diseases (IHD)	
No	263 (93.9%)
Yes	17 (6.1%)
Lipid	
No	227 (81.1%)
Yes	53 (18.9%)

TABLE 2 Mean ESS score by categorical variables

	ESS score	Test statistic[a]	*P*-value
Gender			
Male (n = 204)	10.4 (5.3)	$t = -0.11$	0.913
Female (n = 76)	10.3 (6.2)		
Group			
Home—Embletta (n = 150)	11.0 (5.6)	$t = 2.196$	0.029
Hospital—PSG (n = 130)	9.60 (5.3)		
Smoking status			
Non-smoker (n = 200)	10.2 (5.5)	$F = 1.318$	0.269
Current smoker (n = 40)	10.2 (5.3)		
Ex-smoker (n = 39)	11.7 (6.0)		
Drinking			
Non-drinker(n = 151)	10.0 (5.4)	$F = 0.763$	0.467
Current drinker (n = 5)	9.4 (3.0)		
Ex-drinker (n = 52)	11.0 (5.6)		
AHI			
<15 (n = 107)	10.8 (5.7)	$t = 1.79$	0.075
≥15 (n = 173)	9.6 (5.2)		
Congestive heart failure (CHF)			
No (n = 269)	10.4 (5.6)	$t = -0.673$	0.501
Yes (n = 11)	9.3 (4.2)		
Diabetes			
No (n = 233)	10.5 (5.5)	$t = -1.175$	0.241
Yes (n = 47)	9.5 (5.6)		
Hypertension			
No (n = 144)	11.3 (5.2)	$t = -2.96$	0.003
Yes (n = 136)	9.4 (5.7)		
Cerebrovascular disease (CVA)			
No (n = 273)	10.5 (5.5)	$t = -1.990$	0.048
Yes (n = 7)	6.3 (5.2)		
Ischemic heart diseases (IHD)			
No (n = 263)	10.4 (5.5)	$t = -0.785$	0.433
Yes (n = 17)	9.4 (5.9)		
Lipid			
No (n = 227)	10.6 (5.6)	$t = -1.349$	0.178
Yes (n = 53)	9.5 (5.2)		

[a]t is the test statistic computed using Independent Samples t-test and F is the test statistic computed using One-way Analysis of Variance.

incomplete ESS scores and 25 subjects with incomplete AHI were excluded in the current analysis. Table 1 shows the demographic information of the 280 subjects. Table 2 shows the ESS scores by demographic variables. Significant differences were noted between subjects with and without hypertension. Table 3 shows the Pearson's correlation coefficient between the ESS scores and other variables in the study; only minino2, snore, psnore, and sleep efficiency were statistically significant.

TABLE 3 Correlation between ESS score and other variables (n = 280)

	Age	Height (cm)	Weight (kg)	BMI	Neck circumference (cm)	Waist circumference (cm)	Hip circumference (cm)
r	−0.17	0.072	0.036	0.007	0.067	−0.020	0.064
P-value	0.004	0.231	0.549	0.910	0.265	0.741	0.288
	AHI	**Minio$_2$**	**Meano**	**Arousal**	**SaO290**	**PSaO290**	**ODI**	**Snore**	**PSnore**	**TST**	**Sleepeff**
r[a]	0.071	−0.162	−0.052	−0.014	0.086	0.072	0.116	0.184	0.206	0.116	0.182
P-value	0.235	0.007	0.388	0.872	0.151	0.239	0.053	0.004	0.001	0.083	0.006

AHI, Apnea-Hypopnea Index; arousal, arousal index (only available for patients with full polysomnography in hospital); meano, mean of the SaO_2; minio, minimum of the SaO_2; ODI, oxygen desaturation index; PSaO290, percentage of sleep/index time SaO2 <90%; PSnore, percentage of sleep/index time snoring; SaO290, duration of SaO2 <90%; Snore, duration of snoring; Sleepeff, sleep efficiency; TST, total sleep time.

[a]Pearson's correlation coefficient.

Table 4 shows the ESS scores by using two different cutoff points for the AHI, namely (a) <15/h vs ≥15/h and (b) <30/h vs ≥30/h Regardless of the AHI cutoff, the ESS scores were significantly higher for the normotensive subjects than the hypertensive subjects in the higher AHI group. Likewise, the interaction terms were significant regardless of the AHI cutoff.

Table 5 shows the results of multiple linear regression, in which all variables with *P*-value <0.1 in univariate analysis were considered, including the age, hypertension status, AHI, minio2, psnore, and sleep efficiency. Although the occurrence of snoring was also significant, its correlation with the psnore was 0.96 and only the latter was included to avoid multi-collinearity. After backward selection, only age, psnore, hypertension, AHI, and interaction term between hypertension and AHI were retained in the model. The adjusted mean ESS for normotensive subjects with AHI ≥15/h was 13.11 (95% CI: 11.82, 14.39), significantly higher than the other three groups, namely, normotensive subjects with AHI <15/h (9.35; 95% CI: 8.00, 10.70), hypertensive subjects with AHI <15/h (9.70; 95% CI: 7.84, 11.57), and hypertensive subjects with AHI ≥15/h (9.43; 95% CI: 8.31, 10.55) (Table 6).

4 | DISCUSSION

In this study, we found that, among subjects with moderate to severe OSA (ie, AHI >15/h), the ESS scores of normotensive subjects were significantly higher than hypertensive subjects. Conflict results have been reported in the literature. In their study in Poland, Martynowicz and colleagues[12] reported that the mean ESS score was significantly higher in normotensive subjects than the hypertensive counterparts with moderate to severe OSA, defined as AHI >15/h In a sleep study involving 275 male employees, Harada et al[16] observed that the ESS scores of those with hypertension were lower than those without hypertension, despite statistical insignificance. In examining 149 workers with OSA, Okabayashi et al[17] found that the mean ESS scores of those subjects with hypertension were lower compared with those without hypertension regardless of whether they were overweight. However, Kapur et al[18] reported that the association of sleep-disordered breathing with hypertension is stronger in individuals who report daytime sleepiness than in those who do not. Therefore, more studies should be conducted to examine the association.

The mechanism underlyingsuch differences is still unclear. Previous studies have focused mainly on the association between OSA and daytime sleepiness or hypertension. Kim and colleagues[11] reported, based on 1492 subjects recruited from the community, that no significant differences were noted in the ESS scores between subjects with and without OSA whereas Chung[19] reported that the mean ESS score of OSA patients was significantly higher than that of healthy subjects. Multiple findings, that highlight a bi-directional association between OSA and hypertension,[20] have been noted in previous population-based studies: A significantly positive correlation

TABLE 4 ESS by hypertensive status stratified by AHI (n = 280)

	Non-hypertensive (n = 144)	Hypertensive (n = 136)	*P*-value[a]	Variable	*P*-value[b]
AHI <15 (n = 107)	9.5 (5.3)	9.9 (5.0)	0.694	AHI	0.038
AHI ≥15 (n = 173)	13.0 (5.0)	9.2 (5.9)	<0.001	HPT	0.012
P-value[a]	<0.001	0.514		Interaction	0.02
AHI <30 (n = 188)	10.6 (5.0)	9.9 (5.3)	0.359	AHI	0.279
AHI ≥30 (n = 92)	13.4 (5.4)	8.6 (6.1)	<0.001	HPT	<0.001
P-value[a]	0.005	0.194		Interaction	0.004
Overall	11.3 (5.3)	9.4 (5.6)	0.003		

AHI, Apnea-Hypopnea Index; HPT, hypertension.
[a]*P*-value was computed based on independent samples *t* test.
[b]*P*-value was computed based on two-way analysis of variance (ANOVA) with interaction.

TABLE 5 Multiple regression analysis for Epworth Sleepiness Scale (ESS) score

Variable	β (95% CI)	*P*-value
Intercept	11.84 (8.45, 15.23)	<0.001
Age	−0.06 (−0.12, −0.01)	0.026
PSnore	0.05 (0.02, 0.09)	0.005
HPT (yes as ref)	3.67 (1.97, 5.37)	<0.001
AHI (≥15 as ref)	0.27 (−1.91, 2.45)	0.806
HPT *AHI	−4.03 (−6.87, −1.19)	0.006
R^2 = 14.5%		

AHI, Apnea-Hypopnea Index; HPT, hypertension.

TABLE 6 Adjusted mean (95% CI) of Epworth Sleepiness Scale (ESS) score from the regression model

Variable	Non-hypertensive	Hypertensive
AHI <15	9.35 (8.00, 10.70)	9.70 (7.84, 11.57)
AHI ≥15	13.11 (11.82, 14.39)	9.43 (8.31, 10.55)

AHI, Apnea-Hypopnea Index.

was identified between the AHI and BP[21]; a higher prevalence of OSA was observed in hypertensive individuals[22]; and a higher prevalence of hypertension was reported for OSA patients.[23] However, studies about the interrelationship between OSA, daytime sleepiness, and hypertension are still limited. Montemurro et al[24] reported subjects with severe OSA but without daytime sleepiness had higher very low frequency heart rate variability than those with daytime sleepiness and they suggested the difference could be due to the alertness-inducing effects of excessive sympathetic nervous system activity.

Nevertheless, results from several studies may provide insights about the difference in the ESS scores between non-hypertensive and hypertensive subjects with moderate to severe sleep apnea. Lombardi et al[25] reported that patients with excessive daytime sleepiness, when compared to those without, had significantly lower baroreflex sensitivity and significantly higher low-to-high frequency power ratio of heart rate variability during the different stages of nocturnal sleep. In addition, Kapur and colleagues[18] reported that daytime sleepiness could be an effect modifier of the association between sleep-disordered breathing with hypertension. Furthermore, Feng and colleagues[26] reported that, among severe OSA patients with comparable AHI, the use of the ESS might identify a subset of individuals with OSA at higher risk of hypertension. This demonstrated the potential utility of excessive daytime sleepiness as an indicator for blood pressure profile in patients with OSA.

Other researches, relevant to the interrelationship between OSA, daytime sleepiness, and hypertension, focused mainly on the effectiveness of using continuous positive airway pressure (CPAP) in reducing BP or hypertensive events of patients with OSA but no daytime sleepiness. A RCT of 35 non-sleepy, hypertensive patients with OSA, reported no overall difference in the mean 24-hour ambulatory BP between the subjects in the CPAP and sham treatment group (Robinson et al).[27] In a multicenter RCT with 753 subjects, patients with OSA without daytime sleepiness, the prescription of CPAP compared with usual care, did not result in a statistically significant reduction in the incidence of hypertension.[28] However, from an individual patient data meta-analysis, it showed patients' BP was significant lower for those whose CPAP usage was more than 4 hours per night, similar finding.[29]

There are some limitations to this study. Firstly, the baseline data from the RCT were collected through convenient sampling, due to which selection bias might have introduced; therefore, the demographic information of the subjects was compared to ensure the comparability between the two groups (ie, with or without hypertension). Secondly, the sample size of only 280 subjects was not appreciably large, although adequate statistical power could be attained as the usual rule of thumb for the sample size of regression was 15 subjects per independent variable.[15] In addition, daytime sleepiness was measured subjectively using the ESS but the scale has been validated and widely accepted in clinical practice. Lastly, the AHI of the home sleep test was based on recording time instead of true sleep time as the denominator and this would underestimate the true AHI. Nevertheless, we have previously shown that

the AHI based on the Embletta PDS correlated closely with that obtained by PSG ($r = 0.979$, $P < 0.001$) in the Chinese population in Hong Kong, with a high sensitivity at AHI ≥5/h (sensitivity 0.924 and specificity 0.857) and high specificity at AHI ≥20/h (sensitivity 0.853 and specificity 0.957) in comparisons against the AHI based on PSG.[30] An additional analysis was conducted by the two groups, and the pattern of the ESS scores is the same (Table S1).

In conclusion, subjective daytime sleepiness among normotensive subjects was significantly more severe than that in hypertensive subjects with moderate to severe OSA. Further studies are recommended to explore the mechanisms and the response to CPAP in lowering BP among sleepy and non-sleepy patients with OSA.

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CONFLICT OF INTEREST

None.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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