Predictors of Excessive Daytime Sleepiness in Korean Snoring Patients

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ABSTRACT

Background : Excessive daytime sleepiness is one of the most common symptoms in snoring patients. However, the reason why some individuals complain of daytime sleepiness and others do not is unclear. In this study, we evaluated snoring individuals and examined several demographic and polysomnographic profiles in an attempt to identify predictors of excessive daytime sleepiness. **Methods** : The inclusion criteria for patients were the following: 1) patients who underwent an overnight polysomnograph, 2) patients with the chief complaint of snoring, and 3) patients who completed the Korean version of the Epworth sleepiness scale. We used the Epworth sleepiness scale to estimate excessive daytime sleepiness. We quantified correlations between the Epworth sleepiness scale and the demographic/polysomnographic parameters. We also analyzed the parameters affecting excessive daytime sleepiness using multiple linear regression analysis. **Results** : One hundred nineteen patients met the inclusion criteria for this study. Multiple regression analysis showed that young age was the only independent variable showing statistical significance for predicting excessive daytime sleepiness, and was well-correlated with the Epworth sleepiness scale. However, there were no polysomnographic parameters that were predictive. **Conclusions** : Clinicians need to be cautious when using the Epworth sleepiness scale for the diagnosis of obstructive sleep apnea and determining the response to treatment.

KEY WORDS : Age · Snoring · Daytime Sleepiness · Epworth Sleepiness Scale.

INTRODUCTION

Excessive daytime sleepiness (EDS) has been increasingly recognized as an important public health problem, with as many as 40% of the general population estimated to suffer from abnormal sleepiness at some stage in their lives.1 EDS is known to be a predisposing factor for motor-vehicle accidents, psychosocial morbidity, and poor quality of life.[1-2] It is also one of the most common symptoms in patients who snore or have obstructive sleep apnea (OSA).[3-4] The reason why some individuals with snore complain of EDS, while others do not, is unclear, and the mechanism underlying EDS is not determined. There have been several studies on the relationship between EDS and polysomnographic or demographic profiles in patients with OSA.[1) 4-11] However, the results from these studies have been not consistent, and there have been much controversy on the correlation between subjective EDS and objective polysomnographic findings.[1) 4-11] In some studies, nocturnal hypoxemia or high apnea-hypopnea index (AHI) has been proposed as the most important determinant of EDS in patients with OSA.[1) 5) 8) 11-12] However, other reports did not find such evidence.[6-7) 9) 13] Moreover, few studies have demonstrated that only obesity was a predictor of EDS, while one study has suggested snore was associated with EDS independent of the effect of an AHI.[4-5) 14]

The discrepant results among previous investigations may be due to several factors such as heterogeneity of study subjects or different rules for the scoring respiratory events. In addition, a majority of pre-existing studies shared a common methodological problem, which

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used Epworth sleepiness scale (ESS) to estimate EDS. Of course, ESS is a widely used tool that subjectively measures EDS. It is a simple, self-administered questionnaire with eight-item, four-point scales that investigate the possibility of a person falling asleep during various daily activities.[15] ESS scores are on a numerical scale, which ranges from 0 to 24. However, most studies have used a cutoff of 10 points on the ESS score, dichotomizing patients into those with and without EDS. In this context, investigators had to accept the assumption that patients with ESS score of 11 had same degree of EDS with those who had ESS score of 24. In addition, studies might classify patients with ESS score of either 10 or 11 to belong into non-EDS and EDS groups, despite the difference in subjective score being 1 point out of the 24-point scale between these two groups of patients.

In this present study, we evaluated snoring individuals and examined several demographic and polysomnographic profiles, in an attempt to identify predicting factors for EDS in snoring patients. We analyzed the ESS score as numerical variables using a multiple linear regression analysis.

MATERIALS AND METHODS

This study was conducted up on the approval of the institutional review board at the Haeundae Paik Hospital (Busan, Korea). This case control observational study was performed retrospectively at the single tertiary hospital. From the hospital database, we identified and reviewed the medical records of 151 patients who had undergone overnight polysomnography, whereby each patient was admitted and monitored during an entire night's sleep. The inclusion criteria were made for patients 1) who visited our hospital with the chief complaint of snoring, and 2) who completed the validated Korean version of ESS. The exclusion criteria were for patients 1) younger than 14 years, 2) with medications causing excessive daytime sleepiness such as hypnotics, antidepressants, or antihistamines, or 3) with presence of other sleep disorders such as narcolepsy, central sleep apnea, restless leg syndrome, or periodic limb movement disorders.

The overnight polysomnography included electroencephalography, electrooculography, electrocardiography, chin and tibial electromyography, oral-nasal airflow by thermocouples and nasal pressure, oxyhemoglobin saturation by finger pulse oximeter, chest and abdominal movement by respiratory inductive plethysmography, body position, and snoring noise by a microphone. Digital video recording was performed throughout the night. The polysomnography recordings were analyzed by a certified polysomnography technologist. Total sleep time was defined as the period from sleep onset to final awakening. Each patient was staged according to the The AASM manual for scoring of sleep and associated events.16 Apnea was defined as a pause of airflow more than 10 seconds, and hypopnea was defined as a ≥ 50% decrease in airflow that persisted for more than 10 seconds, and accompanied by oxygen desaturation of 3% or greater or by arousal.16 AHI was calculated as the total number of respiratory events (apnea plus hypopnea) per hour of sleep. The number of episodes of obstructive apnea and hypopnea, and respiratory effort-related arousals per hour of sleep is referred to as the respiratory disturbance index (RDI). Oxygen desaturation index (ODI) was defined as the sum of the number of oxyhemoglobin desaturation of > 4% events per hour of total sleep time. Minimum pulse oxygen saturation (SpO_2) was defined as the lowest night-time saturation point, and duration of SpO_2 < 90% was measured in minutes.

Height, weight, and neck circumference were determined on the night of the sleep study. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Neck circumference, in centimeters, was measured at the level of cricothyroid membrane with a tape measure.

ESS was assessed for each patient using the validated Korean version of the ESS questionnaire.[15] This survey was conducted in the evening before the polysomnographic study by a trained technologist. We used Epworth sleepiness scale to estimate excessive daytime sleepiness.

We collected the demographic profiles such as sex, age, BMI, and neck circumference and polysomnographic parameters such as total sleep time, sleep stage, AHI, RDI, minimum SpO_2, and time length of SpO_2 < 90%. We quantified correlations among ESS and age, BMI, neck circumference, total sleep time, AHI, and ODI using the Pearson correlation coefficient test. We also analyzed the demographic and polysomnographic variables such as age, total sleep time, and ODI affecting excessive daytime sleepiness of the snoring patients using multiple linear regression analysis. All statistical tests were performed using MedCalc®. For all calculations, a p-value less than 0.05 was considered statistically significant. Categorical variables were presented as frequency and percentage. Numerical variables with normal distribution were presented as mean±standard deviation (SD), and those without normal distribution were presented as median with 95% confidence interval and range.

RESULTS

Of the 151 patients who underwent an overnight polysomnography at our hospital, 119 patients met the

Table 1. A comparison of thedemographic profiles between male and female patients.

Parameter	Male (n = 97)	Female (n = 22)	p-value
Age, years (±SD)	45.2±14.5	48.3±13.5	0.3726
BMI, kg/m2 (range)	25.6 (19.5-36.4)	23.0 (19.7-34.7)	0.0757
Neck circumference, cm (±SD)	38.3 (±2.6)	33.3 (±2.6)	0.0001

BMI, body mass index

Table 2. Pearson correlation coefficient between clinical and polysomnographic profiles and Epworth sleepiness scale score.

Parameter	Pearson correlation coefficient	p-value
Age	-0.25	0.0061
BMI	-0.03	0.7316
Neck circumference	0.02	0.8653
Total sleep time	0.22	0.0181
AHI	0.01	0.8928
ODI	0.05	0.6160

BMI, body mass index; AHI, apnea-hypopnea index; ODI, oxygen desaturation index

Table 3. Results of multiple linear regression analysis of demographic and polysomnographic profiles affecting Epworth sleepiness scales in snoring patients.

Independent variables	Beta	p-value
Age	-0.078	0.0218
Total sleep time	0.009	0.1822
ODI	0.019	0.3858

Beta, standard regression coefficients; ODI, oxygen desaturation index

inclusion criteria for this study. Ninety seven patients were men and 22 patients were women. The mean age was 45.8±14.3 years. The median BMI was 25.4 kg/m2 (95% CI 24.7-26.4 kg/m2, range 19.5-36.4 kg/m2). The mean neck circumference was 37.8±3.3 cm. Table 1 summarizes demographic profiles for patients according to sex. On polysomnograms, the mean total sleep time was 321.1±63.7 minutes; the mean total sleep stage N was 90.4±6.2%; the median sleep stage N1 was 43.9% (95% CI 39.0-51.0%, range 7.5-88.9%); the mean sleep stage N2 was 37.4±13.3; the median sleep stage N3 was 4.5% (95% CI 3.3-6.8%, range 0-32.3%); the mean sleep stage R was 9.6±6.1; the median AHI was 22.0 (95% CI 15.0-30.0, range 0-84.3); the median RDI was 31.2 (95% CI 22.4-36.9, range 0-86.9); the median ODI was 13.9 (95% CI 9.4-20.3, range 0-72.3); the median minimum SpO2 was 83% (95% CI 81-85%, range 53-95%); and, the median time length of SpO2 < 90% was 11.9 minutes (95% CI 7.4-22.0 minutes, range 0-287.4 minutes). The mean ESS was 8.8±4.8.

Correlations among ESS and age, BMI, neck circumference, total sleep time, AHI, or ODI are shown in Table 2. Age and total sleep time were found significantly correlated with ESS (r = -0.25, p = 0.0061; r = 0.22, p = 0.0181, respectively). After adjusting for demographic and polysomnographic variables such as age, total sleep time, and ODI, multiple linear regression analysis revealed that younger age was the sole independent variable for predicting EDS (Table 3).

DISCUSSION

The main finding in this study was that none of the polysomnographic parameters were significant predictors for EDS, and only age was associated with subjective sleepiness. This result was consistent with previously published findings.[6-7) 9) 13) 17-18)] Young et al. demonstrated that many patients with OSA had no complaints of daytime sleepiness, with only 22.6% of women and 15.5% of men with AHI > 5 reporting EDS.[19)] Another group of investigators has suggested that continuous positive airway pressure did not improve EDS, especially in milder cases of sleep apnea.[20)] Potential explanation for the weak relationship between EDS and polysomnographic parameters may lie in multifactorial pathophysiology of sleepiness. Sleepiness is not only associated with sleep fragmentation, but also various co-morbid conditions such as respiratory diseases, sleep restriction, insomnia, and nocturnal leg complains.[21)] Depression, diabetes, obstructive airway disease, heart disease, stroke, alcohol use, and hypothyroidism may also contribute to increased subjective sleepiness.[22] Thus, sleepiness cannot be explained by a single factor. Nevertheless, some studies have found relationships between EDS and nocturnal hypoxemia or sleep fragmentation. One plausible explanation for this controversial finding is the problem of assessing EDS. Because many confounding factors including depression, personality, or culture influence ESS scores, ESS is probably not the best instrument to detect identify components of overnight sleep that influence daytime sleepiness. In addition, the word "sleepiness" can have a variety of meanings and interpretations. Patients with OSA often use terms such as fatigue, tiredness, or lack of energy instead of sleepiness.[23] Thus, using more objective tools to measure EDS like multiple sleep latency test (MSLT) may be useful. Fong et al. demonstrated that MSLT was better than ESS in the assessment of EDS in patients with OSA.[24]

Nevertheless, in this present study, we made an effort to keep objectiveness. All ESS survey was conducted by one experienced technologist, and we also analyzed the relation between ESS and demographic/polysomnographic profiles using multiple linear regression analysis. By these efforts, we found that one of the strongest predicting factors for EDS was age, and our data showed significantly negative correlation between age and ESS score. This finding was in agreement with that of a previous study,[25] and is consistent with known age-related changes in sleep architecture. In general, sleep becomes lighter and more fragmented, and total sleep time decreases with age.[26] Thus, older patients could already be adapted to sleep fragmentation. In addition, chronically sleep-deprived individuals often do not recognize the subjective feeling of sleepiness.[27] Thus, older patients may feel EDS to a lesser degree compared to younger patients. Moreover, younger patients usually have increased unmet sleep needs from a more active lifestyle.[25] On the other hand, a previous study demonstrated that EDS was more prevalent in the very old (> 75 years) despite of decreasing EDS in a linear fashion with increasing age between the age limits of 30 and 75 years.[25] However, because we enrolled only one patient who was more than > 75 years old, we were not able to evaluate it.

Slater et al reported that obesity was one of the most important determinants of EDS,[4] but our study did not find such evidence. Our findings was consistent with other studies.[6] [7] [10] [11] These conflicting results might be due to differences in the degree of obesity. The median BMI was 25.4 kg/m2 in our patients, whereas the mean BMI of the patients was 30.9 kg/m2 in the Slater and colleague study.[4] A previous study demonstrated that the BMI-specific prevalence of EDS remained constant until the overweight threshold (28 kg/m2 in BMI), and beyond this BMI, the prevalence of ECD increased in an exponential manner.[25] Considering lower BMI in Asians compared to Caucasians, we would like to suppose that BMI is not related with EDS in Asians.

There were several strengths of this study. First, in contrast to previous studies using logistic regression analyses, we used a multiple linear regression analysis because ESS scores were normally distributed in our study. The multiple logistic regression analysis is usually used when the dependent variable is binary, and the magnitudes of dependent variable are not meaningful.[28] However, because ESS scores are on a numerical scale, multiple linear regression analysis may be more appropriate. Second, all of the subjects of this study were Asians. Few studies were done for Asian populations, especially that of Far East Asian. As far as sleep disorders are concerned, there is a significant difference across various sleep study populations. Anatomically, Asians has a shorter and steeper anterior cranial base and a smaller and more posteriorly positioned mandible than Caucasians, and because of these differences, Asian individuals are believed to be more prone to OSA.[29] In addition, Asians have different cultures and personalities from those in Caucasians. Thus, the study of the Asians like this study may be needed.

There are several limitations to this study. First, this was a retrospective study with a relatively small sample size. Second, we did not investigate the sleep pattern of our patients using sleep diary which are often more accurate than relying on recall of their sleep patterns in daily life. Increased unmet sleep needs from a more active lifestyle can be associated with EDS. Third, the accuracy of ESS is dependent on participant interpretation and estimation, which renders it highly subjective.[13] [18] More objective instruments, such as MSLT, may be needed to evaluate predicting parameters in polysomnography or demography for EDS. Fourth, previous studies demonstrated that lack of regular exercise, depression, or diabetes is associated with EDS, but these factors were not evaluated in our study.[25] [30]

CONCLUSIONS

We demonstrated that only age was associated with daytime sleepiness in Asians, and there were no significantly predicting factors among polysomnographic parameters. Clinicians have to be cautious, when they use Epworth sleepiness scale for the diagnosis of obstructive sleep apnea and determining the response to treatment.

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