Effect of Smoking on Parameters of Polysomnography

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Abstract

Background and Objective: Obstructive sleep apnea syndrome (OSAS) is a kind of sleep-related disorder with complex features and may lead to daytime sleepiness. This study aimed to find the differences between smokers and non-smokers regarding the parameters of polysomnography.

Materials and Methods: This was a cross-sectional study conducted on consecutive patients referred to sleep lab to perform polysomnography for sleep-related breathing disorders in Shahid Sadoughi University of Medical Sciences in 2014. For all participants overnight polysomnography study was performed for at least six hours. Then, parameters of polysomnography were compared between smokers and non-smokers. Data were analyzed by SPSS using chi-square test, Student's t test, Mann-Whitney U test, and logistic regression analysis.

Results: Totally 228 subjects were enrolled in the study (155 non-smokers). Obstructive apnea number and index, total apnea number, apnea hypopnea index (AHI), baseline and average saturation by pulse oximeter (SPO2), snore, and snore arousal were significantly different between smokers and non-smokers ($P<0.05$). Severe OSA was most commonly observed among smokers, but AHI was not significantly correlated with pack-years of smoking.

Conclusion: The results of this study showed that smokers suffer from severe OSA more frequently than non-smokers, obstructive apnea is more common among smokers with higher AHI.

Keywords: Obstructive sleep apnea syndrome, Smoking, Apnea hypopnea index, Snoring

Introduction

Obstructive sleep apnea syndrome (OSAS) is a kind of sleep-related chronic disorder with such complex features as repetitive sleep apnea, snoring, and oxygen desaturation which may lead to daytime sleepiness (1,2). Obstructive apnea is a complete or near-complete interruption of airflow for at least 10 seconds during sleep (2). OSAS may lead to some complications such as cardiovascular disorders and hypertension, depression, metabolic syndrome, and sudden death (2).

OSAS is more common in males with an incidence of 2.1% to 8.8% in different populations (1,3-5). Its prevalence has been estimated to be between 8% and 33% and is affected by such factors as age and obesity (2,6,7,8).

Other risk factors of this disorder include: Craniofacial anatomy and anomalies, neck circumference, abdominal circumference, genetics (2,9), alcohol ingestion, and ethnicity (10,11). Smoking which is a known predisposing factor for pulmonary and cardiovascular diseases is highly prevalent in OSAS patients (12). Smoking has also been implicated as a risk factor of OSAS (13).

Smoking affects the mucosa of the upper airway and uvula (1). Studies have confirmed that smoking increases the morbidity of sleep- related respiratory disorders (13,14), but the association between smoking and OSAS is yet controversial; the correlation between the amount of cigarette smoking and the severity of OSAS and other

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parameters measured in polysomnography such as arousals are not well-documented as well.

Some studies, based on questionnaires, have shown a higher frequency of sleep problems such as difficulty falling asleep, maintaining sleep, and snoring among smokers (8, 15-17). Moreno et Al. in a questionnaire-based study found smoking as an independent risk factor for OSA (18).

Boussoffara et al. found that severe OSAS is more common among smokers (19); Kashyap et el., and Wetter et al. also found a higher frequency of moderate and severe OSAS among smokers than non-smokers (13,20). Hoffstein et al. found a greater number of current smokers with AHI ≥50 than among those with AHI <10, and they concluded that smoking is not an independent risk factor for sleep apnea when considering such confounding variables as age and body mass index (BMI) (21).

Casasola et al. found that smoking doesn't affect OSA (22). Conway et al. observed that both current and former smokers had a higher arousal index compared to never smokers (23). Zhang et al. found that cigarette smoking could increase the latency to sleep onset and make a shift toward lighter stages of sleep (24). Neruntarat et al. found a higher prevalence of OSA in smokers than non-smokers (25).

Although an association has been found between smoking and some parameters of sleep, there is still controversy about the exact effect of smoking and parameters of polysomnography. This study was conducted to find the differences between smokers and non-smokers regarding the parameters of polysomnography.

Materials and Methods

This was a cross-sectional study conducted in the sleep lab of university occupational medicine department, in Yazd, Iran. The study was approved by the ethics committee of Shahid Sadoughi University of Medical.

Subjects

Subjects were selected from consecutive patients who were referred to sleep lab to perform polysomnography for sleep-related breathing disorders during December 2012-March 2014 that were diagnosed as having OSAS. The subjects were assessed in two groups; those with known chronic respiratory diseases, opium and hookah use, and smoking less than one pack-year were excluded from the study. An informed consent was obtained from each participant.

Smoking history

Smoking history was quantified as pack-years (PY) of smoking (i.e. number of cigarettes smoked per day divided by 20 and multiplied by years of smoking). Those with at least one pack-year smoking were considered as current smokers, and those who have quitted smoking for at least 1 year were considered as former smokers. Smokers were divided into two groups: ≥ 10 PY and < 10 PY.

Sleep study

All subjects performed an overnight polysomnography study (device: Somnomedics, Germany) for at least 6 hours. The device consisted of 10 EEG (electroencephalogram) channels, two EOG (electrooculogram) channels, a pressure transducer (cannula), a thermistor for oral and nasal airflow, a pulse oximeter for oxygen saturation and pulse, two piezoelectric bands for thoracic and abdominal movements, a snore sensor, two sensors for leg movements and two sensors for chin EMG (electromyogram). Sensors were attached in the evening, and after calibration, recording was initiated.

Parameters of polysomnography including apnea (obstructive, central, and mixed), apnea index, hypopnea and hypopnea index, apnea/hypopnea count and index (AHI), O2 desaturation count and index, baseline and average SPO2, arousal number and index, and snore number and index were recorded. The patients were asked not to sleep in the afternoon before test, eat a small dinner and not to use sleeping pills.

The recordings were analyzed using Somnomedics software (Somnomedics, Germany). The analysis was performed according to American Association of Sleep Medicine (AASM) manual, 2007 (26), i.e. complete cessation of airflow ≥ 10 s was defined as apnea using the thermistor recordings; hypopnea was defined as a more than 50% reduction in airflow associated with ≥ 3% drop in O2 saturation using the pressure sensor. AHI was defined as the number of apneas+hypopneas per sleeping hour. Arousals were defined as abrupt shift in EEG waves frequency for at least 3 seconds with at least 10 seconds of stable sleep prior to the event. The oxygen desaturation index (ODI) was defined as the number of oxygen desaturations ≥ 3% per sleeping hour.

Those with an apnea-hypopnea index (AHI) ≥ 5 were considered as having OSAS, and OSA severity was classified as mild (5 ≤ AHI < 15), moderate (15 ≤ AHI < 30), or severe (AHI ≥ 30).

Other measurements

Neck and abdominal circumferences were measured by a tape meter. Height was measured using a stadiometer and weight was measured by a digital scale (Laica, Italy, accuracy: 100g). Body Mass Index (BMI) was calculated by dividing weight (in Kg) by square of height (in m).

Statistical analysis

Data were analyzed by SPSS (ver. 20) using chi square test, Student's t test, Mann-Whitney U test, and logistic regression analysis. Level of significance was set at $P<0.05$.

Results

Totally 228 subjects entered the study (155 non-smokers and 73 smokers). Table 1 compares demographic and anthropometric data between two groups.

Table 1. Demographic and anthropometric data of two groups

Variable		Mean (SD*)	Median	*P* value
Age (yr)	S*	48.66 (12.74)	43.5	0.92
	NS*	48.75 (12.99)	47	
BMI (Kg/m^2)	S	32.58 (4.79)	31.9	0.25
	NS	32.27 (5.26)	30	
Neck circumference (cm)	S	43.86 (4.82)	44	0.004**
	NS	41.83 (3.83)	42	
Abdominal circumference (cm)	S	111.89 (12.33)	110	0.43
	NS	110.45 (13.63)	108	

* SD: Standard deviation; S: Smoker; NS: non-smoker, ** significant difference

The parameters of polysomnography that were significantly different between smokers and non-smokers included obstructive apnea number and index, total apnea number, AHI, baseline and average SPO2, snore, and snore arousal. Table 2 compares the parameters of polysomnography between two groups.

Table 2. Comparison of polysomnographic parameters between smokers and non-smokers

Variable	Mean (SD*)		Median		*P* value***
	S**	NS**	S	NS	
Obstructive apnea (n)	99.14 (125.26)	58.17 (89.73)	42	25	0.007
Obstructive apnea index	17.56 (21.62)	10.18 (14.06)	6.9	5.1	0.011
Central apnea (n)	17.63 (55.51)	6.33 (14.27)	0	1	0.89
Mixed apnea (n)	54.55 (94.07)	53.65 (97.27)	8	7	0.45
Total apnea (n)	167.75 (170.69)	118.32 (144.25)	102	54	0.013
Apnea index	30.90 (42.90)	23.36 (30.25)	14.6	10.8	0.08
Hypopnea (n)	72.18 (61.13)	69.63 (55.79)	57	58	0.83
Hypopnea index	13.47 (12.21)	12.79 (9.56)	9	10	0.87
AH number	234.95 (167.35)	185.36 (149.37)	180.5	135	0.02
AHI	40.65 (25.48)	33.98 (25.04)	37.9	26.4	0.034
O_2 desaturation (n)	214.45 (136.68)	186.81 (140.74)	167	140	0.11
O_2 desaturation index	37.51 (22.95)	37.63 (31.95)	32	29.8	0.47
Baseline SPO_2	89.38 (5.07)	91.08 (4.11)	91	93	0.005
Average SPO_2	88.49 (5.63)	89.41 (8.12)	91	92	0.044
Arousal (n)	125.46 (60.35)	115.17 (53.77)	123	109	0.19
Arousal index	21.16 (10.75)	20.15 (7.83)	20.6	19.8	0.89
Snore arousal (n)	17.60 (14.72)	13.60 (14.26)			0.036
Snore arousal index	2.85 (2.29)	2.31 (2.26)			0.056
Snore (n)	2595.42 (1323.01)	2167.41 (1270.98)	2493.5	2054	0.046
Snore index	422.29 (194.58)	376.96 (206.30)	430	373.1	0.14

* SD: standard deviation; ** S: smoker, NS: Non-smoker, *** underlined cells show significant difference

Totally, 26.8%, 23.7%, and 49.6% of the patients suffered from mild, moderate and severe OSA, respectively.

Table 3 shows the frequency of different severities of OSA in smokers and non-smokers.

Categorization of the subjects according to the severity of OSA showed that severe OSA is most commonly observed among smokers (Odds ratio for severe OSA = 2.06, 95%CI = 1.16-3.63, $P = 0.016$). There was no significant difference between current and former smokers regarding the frequency of severe OSA (Odds ratio = 2.30, 95%CI =0.72-7.25, *P* value = 0.18). Figure

1 compares the frequency of different severities of OSA between smokers and non-smokers.

Table 3. Frequency of different severities of OSA in smokers and non-smokers

OSA severity	Non-smoker		Smoker	
	Number	Percent	Number	Percent
Mild	46	29.7	15	20.5
Moderate	41	26.5	13	17.8
Severe	68	43.9	45	61.6

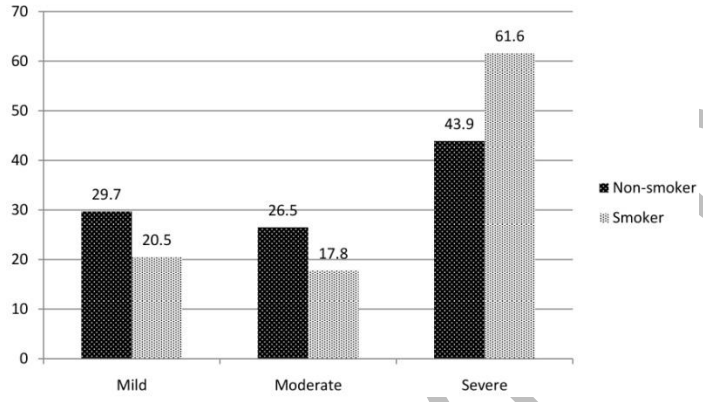

Figure 1. Comparison of different severities of OSA between smokers and non-smokers

Regression analysis showed that after adjusting for neck circumference, AHI is again significantly different between smokers and non- smokers (adjusted *P* value = 0.027).

Among smokers, 53 were current smokers and 20 were former smokers. Mean (±SD) age was 45.94 (±11.98) and 55.85 (±12.15) years in current and former smokers, respectively and the difference was significant (*P* = 0.003). Other parameters (i.e. BMI, neck circumference and abdominal circumference) were not significantly different between two groups.

Figure 2 compares the frequency of different AHI severities among non-smokers, current smokers and former smokers. About 44% of non-smokers, 54% of current smokers and 75% of former smokers suffered from severe OSAS.

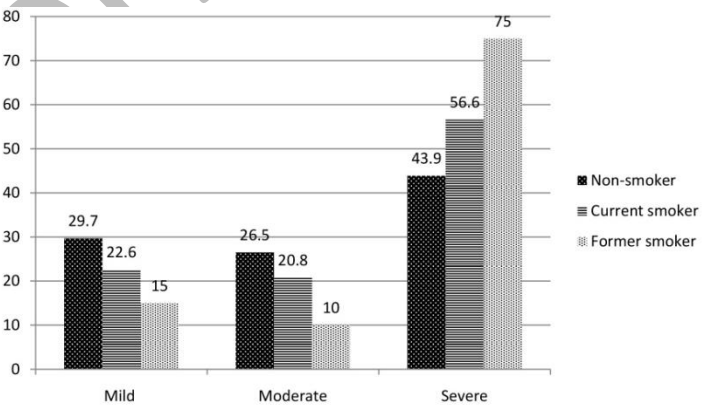

Figure 2. Comparison of different severities of OSA between current smokers, former smokers and non-smokers

There was a significant difference in AHI among subjects regarding the status of smoking (*P* = 0.023). After adjusting for age, the difference in AHI between current and former smokers was not significant (*P* >

0.05).

AHI was not significantly correlated with pack-years of smoking (r = 0.35, *P* = 0.76). After dividing the smokers into pack-year > 10 and pack-year ≤ 10, there was not a significant difference regarding the severity of OSA between two groups (*P* = 0.25).

Discussion

Smoking affects many organs in the body. It is believed that it may affect the quality of sleep as well which can be assessed by some questionnaires and more accurately by polysomnography. In this study, we compared the parameters of polysomnography between smokers and non-smokers.

The results of the current study showed that most parameters of polysomnography are different between smokers and non-smokers and this difference is statistically significant in some parameters including obstructive apnea number and index, total apnea number, baseline and average SPO2, and snore, and snore arousal. The difference in snore index and snore arousal index was not statistically significant, but there was a considerable difference which is clinically significant.

There is a controversy in the results of different studies which is probably due to different methodologies and different study populations (20,21,23,27,28). Many studies have used questionnaires for assessment of the effect of smoking on sleep parameters (18,27,29), but most recent studies have used more objective parameters such as parameters of polysomnography (1,19), especially respiratory parameters; and few studies have assessed O2 desaturation (22,23). Thus, in the current study we aimed to detect the effect of smoking and smoking severity, considering current and former smokers, on objective sleep parameters.

Smoking may affect sleep by several possible mechanisms, including upper airway inflammation, stimulation of upper airway muscles by nicotine and progressive decrease in blood nicotine concentrations, after a few hours of sleep, which may increase upper airway resistance (as a rebound effect), reduced nasal cavity dimensions, low airflow and a less-compliant nasal mucosa (20,30- 32).

The complaint of disturbed sleep is more common among smokers (24,33). Most questionnaire-based studies have found a significant difference between smokers and non-smokers regarding sleep parameters (18,27,29,29). Zhang et al. found that smoking significantly affects sleep architecture (total sleep time, and sleep pattern), but they did not assessed respiratory events of sleep (24).

Reports of the effects of smoking habits on AHI are controversial (20-23). Boussoffara et al. in a study on 151 OSAS patients found that AHI was significantly higher among current smokers than non-smokers (42.96/h versus 28.77/h) (19), a similar result was also observed in the current study, though with a smaller difference (40.65/h vs. 33.95/h). We found that smokers are 2.06 times more likely suffering from severe OSAS. This measure was 3.7 times in the study conducted by Boussoffara et al. (19). In the current study, severe OSAS was more common among smokers, but Kim et al. and Wetter et al. found that both severe and moderate OSAS were more common among smokers (1,13), although the mean age in our study population was higher. Hoffstein et al. and Casasola et el. couldn't find a relationship between smoking and severity of OSA (21,22). The latter study didn't find a difference between smokers and non-smokers regarding AHI (22).

In the current study, the arousals were not significantly different between smokers and non-smokers, but Conway et al. found that arousal index was significantly higher among current and former smokers than non-smokers (23). Due to the higher snore in the smokers, snore arousals were also more common among smokers in the current study consistent with the study conducted by Wetter et al. (13), although they didn't use polysomnography for assessment of sleep parameters.

Inconsistent with the results of the current study, Conway et al. didn't find a difference between smokers and non-smokers regarding respiratory events. Baseline and average SPO2 was significantly lower among the smokers in the present study without any difference between heavy and light smokers; Conway et al. also assessed SPO2 and O2 desaturation and found that smokers had a higher total sleep time with hypoxia and this difference was also observed between heavy and light smokers (23), although they defined heavy smoking as smoking ≥ 15 pack-years, but our definition was smoking≥10 pack-years.

We couldn't find a correlation between the pack-years of smoking and severity of OSA, but Conway et al. and Wetter et al. found this correlation (13,23). This difference is probably due to different definitions of heavy smoking.

This study had some limitations: first, this was a cross-sectional study which suffers from the intrinsic limitations of these kinds of studies, so a causal relationship cannot be concluded from this study. The data about smoking was self-reported, so it may be subject to recall bias. For detecting exclusion criteria we

used only clinical examination, and other diagnostic measures (laboratory and pathology) could not be performed due to monetary limitations. We couldn't exclude passive smokers. We didn't have information about alcohol consumption.

In conclusion, the results of this study showed that smokers suffer from severe OSA more frequently than non-smokers, and obstructive apnea is more common among smokers; but the severity of smoking didn't affect the severity of OSA. In addition current and former smokers were not significantly different regarding sleep parameters.

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