

The Impact of Gender on Evaluation of Polysomnographic Data

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Abstract

Background: There seems to be a gender bias in diagnosing OSAS and the disease incidence in women is generally underestimated. We investigated the gender disparities on sleep architecture, as well as incidence and severity of OSAS.

Methods: Subjects admitted for polysomnography between January 2011-December 2015 were retrospectively analysed.

Results: The study population consisted of 892 (64.3%) male, 495 (35.7%) female subjects; 1263 (91%) were diagnosed as OSAS; OSAS was diagnosed in 93% of male cases (n = 831) and 87% of female cases (n = 432). Men has higher sleep efficiency and lower sleep and REM latency. AHI, AI and ODI were significantly higher in men. Considering female population, 24.6% were in premenopausal period and 75.4% were postmenopausal; 68% of former and 93.6% of latter group had OSAS. BMI of postmenopausal women was significantly higher than men.

Conclusion: In this study, evaluating the PSG data due to gender in a large group of patients, we found out that male population had higher incidence of OSAS, more severe disease and were at younger ages. Most women with OSAS were postmenopausal. Women had lower sleep efficiency, higher latency to sleep onset and latency to REM. As a conclusion, gender is one of the major factors influencing PSG data and should be taken into consideration in evaluating OSAS cases.

Keywords: Obstructive Sleep Apnea Syndrome; Gender; Sleep Architecture; Polysomnography

Introduction

Obstructive sleep apnea syndrome (OSAS) is characterized with repetitive upper airway obstruction. The prevalence and risk factors of the disease seem to change in different communities and ethnic groups. Gender is also a major factor effecting the incidence and severity of sleep disordered breathing. It has been considered as a disease of men for decades and has remained unrecognized in women. By epidemiologic investigations the public health burden attributable to the disease was recognised and the awareness of the fact that, the female population is effected by the disease, increased [1]. Female/male ratio is about 1/2 and 10% of male population is effected by the disease. Lower incidence in female subjects seem to be attributed to hormones, body fat distribution, upper airway anatomy and functions, neurological respiratory control mechanisms [2]. We investigated the effects of gender on sleep architecture, as well as incidence of sleep disordered breathing. We aimed to determine the gender related predisposition for OSAS and characterize how gender influenced disease severity.

Methods

This study was conducted in Istanbul, Turkey. A total of 1387 subjects admitted for polysomnography between January 2011- December 2015 were retrospectively analysed. All the subjects had complaints of snoring, witnessed apnea, daytime hypersomnolence and

unsatisfactory sleep. Subjects who could not complete full night polysomnography either due to non-compliance and who underwent split-night test were excluded. Written consent was signed by all patients at time of admission.

Standard overnight polysomnography included four electroencephalogram channels (C3-M2, C4-M1, O1-M2, O2-M1) according to the 10 - 20 international electrode placement system, right and left electrooculogram channels (E1-M2, E2-M1), and chin electromyogram channel and electrocardiography channel. Air flow was measured by a nasal pressure transducer and oronasal thermistor. Respiratory effort was measured by respiratory inductance plethysmography. Measurement of arterial oxyhemoglobin saturation (SpO_2) was performed by finger pulse oxymeter. All signals were collected and digitalized by computerized polysomnography (PSG) systems (Comet Grass, Astro-Med, Inc., West Warwick, RI, USA and Viasys Cephalo Pro, SomnoStar, VIASYS Healthcare, Hoechberg, Germany) conducted by experienced technicians. Sleep stages were scored in 30-s epochs using American Academy of Sleep Medicine (AASM) 2012 [3] scoring systems. Each epoch was analyzed for the number of apnea and hypopneas. Apnea was defined as a cessation of airflow for more than 10s, and hypopnea as a reduction of airflow $> 50\%$ for >10 s plus an oxygen desaturation of $> 3\%$ or an arousal [3,4]. Apnea-hypopnea index (AHI) was calculated as total number of apneas and hypopneas divided by total sleep hours [3,4]. Scoring was made by a certificated technician experienced in sleep medicine. The classification of disease severity was made according to AASM 2005 Guide [5]. Patients were graded according to AASM 1999 [4] criteria as follows: AHI < 5 as normal, ≥ 5 and < 15 as mild, ≥ 15 and < 30 as moderate, and ≥ 30 as severe. Body mass index (BMI), age, the parameters denoting sleep architecture and detailed polysomnographic data were compared between men and women. Female subjects were further subdivided into pre- and postmenopausal groups and prevalence and severity of disease were compared.

Statistical analyses were performed using SPSS 17.0 software. Mean values (with standard deviations where applicable) were calculated for age, BMI and polysomnographic measurements. The comparison between groups was done with T-test for continuous variables and chi-square test for categorical variables. $P < 0.05$ was considered as statistically significant. Multivariate logistic regression analysis was used in evaluation of independent determinants.

Results

Overall, 1387 patients were included in the study. The study population consisted of 892 (64.3%) male, 495 (35.7%) female subjects; 1263 (91%) were diagnosed as OSAS; 363 (29%) had mild disease, 342 (27%) had moderate disease and 558 (44%) had severe disease. OSAS was diagnosed in 93% of males ($n = 831$) and 87% of females ($n = 432$). Male and female subjects were stratified by severity of OSAS; both prevalence and severity of disease were higher in men. Majority of the male population (48.3%), had severe OSAS, whereas female OSAS patients were more uniformly distributed in mild, moderate and severe groups (Figure 1). Table 1 and 2 present demographic and polysomnographic characteristics of male and female subjects in the whole study population and OSAS group. Considering the whole group, women were significantly older. Body mass index (BMI) was similar in men and women. Men had higher sleep efficiency and lower sleep and rapid eye movement (REM) latency ($p < 0.0001$). There were no differences with respect to stage 1, stage 2 and REM duration; stage 3 duration was significantly longer in women in the whole group; in OSAS cases, stage 2 was significantly shorter and stage 3 was significantly longer in women. AHI, apnea index (AI) and oxygen desaturation index (ODI) were significantly higher in men ($p < 0.0001$). The duration of oxygen saturation (SpO_2) remaining below 90% and lowest values of oxygen saturation were similar in both groups.

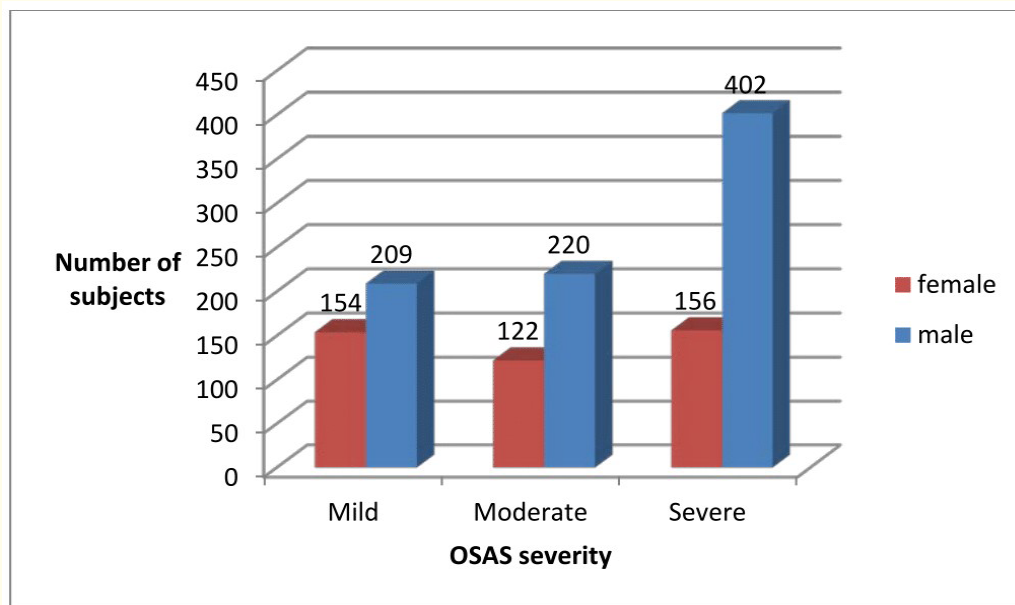


Figure 1: Distribution of male and female cases due to OSAS severity.

	Male	Female	p value
BMI (kg/m ²)	30.96 ± 5.26	33.76 ± 7.08	< 0.0001
Total sleep time (minutes)	295.64	297.54	0.73
Sleep efficiency (%)	82.62	76.46	< 0.0001
Sleep latency (minutes)	13.82	23.30	< 0.0001
REM latency (minutes)	120.34	142.65	< 0.0001
Stage 1 (%)	8.64	9.04	0.4
Stage 2 (%)	63.29	61.31	0.003
Stage 3 (%)	13.81	16.08	< 0.0001
Stage REM (%)	14.08	14.10	0.97
AHI	37.46	29.81	< 0.0001
AI	24.56	14.48	< 0.0001
Time elapsed with SpO ₂ < 90% (%)	19.16	18.03	0.57
ODI	31.70	24.65	< 0.0001
Minimum spO ₂ (%)	80.22	79.82	0.52
Mean spO ₂ (%)	91.88	92.31	0.18

Table 1: Polysomnographic data in men and women in OSAS group.

	Male	Female	p value
BMI (kg/m ²)	30.71 ± 5.44	32.91 ± 7.28	0.31
Total sleep time (minutes)	299	300	0.84
Sleep efficiency (%)	83.0	76.5	< 0.0001
Sleep latency (minutes)	13.96	23.54	< 0.0001
REM latency (minutes)	120	143	< 0.0001
Stage 1 (%)	8.5	8.8	0.62
Stage 2 (%)	62.7	60.8	0.002
Stage 3 (%)	14.3	16.6	< 0.0001
Stage REM (%)	14.2	14.4	0.64
AHI	35	26	< 0.0001
AI	23	13	< 0.0001
Time elapsed with SpO ₂ < 90% (%)	17.5	15.8	0.33
ODI	29.6	21.6	< 0.0001
Minimum spO ₂ (%)	81.2	80.8	0.53
Mean spO ₂ (%)	92.0	92.6	0.05

Table 2: Polysomnographic data in men and women in the whole study group.

Considering female population, 122 (24.6%) were in premenopausal and 373 (75.4%) were postmenopausal; 68% of former and 93.6% of latter group had OSAS. None of the women in the postmenopausal ages reported use of hormone replacement treatment. In premenopausal group majority of the subjects had mild OSAS (57.8%), whereas majority of OSAS patients in postmenopausal group had severe disease (37.8%) (Figure 2).

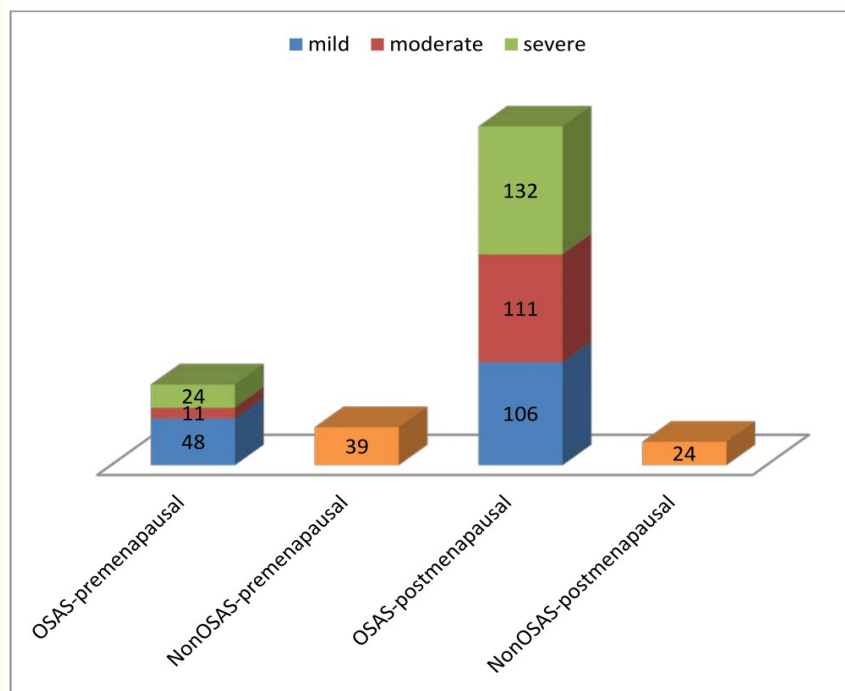


Figure 2: Distribution of female subjects with respect to age groups and OSAS severity.

Taking the nonapneics into consideration, mean BMI was 27.19 in women and 27.49 in men ($p = 0.8$). However, in OSAS patients, mean BMI was 33.46 in women and 30.96 in men ($p < 0.0001$). The mean BMI values in mild, moderate and severe OSAS groups in men and women are compared. Mean BMI in women and men were 32.58 and 29.13, respectively in mild OSAS; 33.13 and 29.91, respectively in moderate OSAS; 35.45 and 32.42, respectively in severe OSAS. In all groups the difference between males and females is statistically significant ($p < 0.0001$).

In female OSAS subjects, mean BMI was 29.59 in premenapausal period and 33.98 in postmenapausal period. The difference was statistically significant ($p < 0.0001$).

The differences between men and women due to sleep architecture are summarised in table 3 and 4. Total sleep time was similar in men and women in non-OSAS, mild and severe OSAS groups, however in moderate OSAS group males had significantly longer total sleep time than female counterparts ($p = 0.005$). Sleep efficiency was similar between men and women if they had $AHI < 5$, however when $AHI > 5$, women had significantly lower sleep efficiency regardless of the severity of the disease ($p = 0.01$ in mild cases and $p = 0.001$ in moderate and severe cases). Sleep latency was significantly longer in all women, with or without OSAS. Still, the difference between male and female subjects with respect to sleep latency tended to increase as the disease got more severe ($p = 0.02$ in nonapneics, $p = 0.002$ in mild cases, $p = 0.001$ in moderate cases and $p < 0.0001$ in severe cases). Similarly REM latency was significantly lower in female subjects with OSAS in all groups, and the difference with their male counterparts increased with the severity of OSAS ($p = 0.04$ in mild group, $p = 0.004$ in moderate group and $p < 0.0001$ in severe group). REM latency was not significantly different in two sexes in nonapneics.

	Total sleep time		Sleep efficiency		Sleep latency		REM latency	
	Minutes	P value	%	P value	Minutes	P value	Minutes	P value
Non-OSAS n								
Female n	316.47	0.08	76.88	0.07	25.25	0.02	145.00	0.09
Male n	342.71		89.00		15.84		120.06	
Mild OSAS								
Female	331.14	0.15	80.27	0.01	19.36	0.002	131.28	0.04
Male	341.89		84.12		13.08		115.60	
Moderate OSAS								
Female	304.99	0.005	76.69	0.001	25.50	0.001	136.71	0.004
Male	330.42		82.06		16.17		110.73	
Severe OSAS								
Female	258.81	0.51	72.51	0.001	25.58	<	161.37	< 0.0001
Male	252.34		82.15		12.91	0.0001	129.78	

Table 3: The differences between men and women due to sleep architecture.

	Stage 1		Stage 2		Stage 3		Stage REM	
	%	P value	%	P value	%	P value	%	P value
Non-OSAS								
Female	6.83	0.60	57.59	0.13	20.65	0.54	16.94	0.7
Male	7.26		55.06		21.57		15.93	
Mild OSAS								
Female	6.64	0.78	58.15	0.69	19.68	0.18	15.82	0.48
Male	6.50		58.50		18.66		16.35	
Moderate OSAS								
Female	7.58	0.62	60.07	0.16	17.08	0.92	15.88	0.34
Male	7.31		58.70		17.15		16.67	
Severe OSAS								
Female	12.55	0.04	65.40	0.015	11.76	0.004	10.98	0.54
Male	10.47		68.29		9.45		11.48	

Table 4: The differences between men and women due to sleep stages.

Percentage stage 1 and percentage stage REM were similar in men and women in both OSAS and non-OSAS subjects, as well as in mild, moderate and severe OSAS groups. Percentage stage 2 and 3 did not differ between sexes in nonapneics and mild and moderate OSAS cases, however men with severe OSAS had significantly longer stage 2 sleep ($p = 0.015$) and significantly shorter stage 3 sleep ($p = 0.004$) compared to women.

Discussion

This study includes a large group of caucasian subjects. Our main finding is that men had higher incidence of OSAS, more severe disease and were at younger ages compared to women. Most women with OSAS were postmenapausal. Women had lower sleep efficiency,

higher latency to sleep onset and latency to REM. In our study, female subjects diagnosed as OSAS had significantly higher BMI compared to male OSAS patients; moreover in postmenopausal period BMI tended to increase significantly; therefore obesity seems to be a major determinant in OSAS.

Sleep quality is a major determinant of daytime performance. The duration of sleep stages, timing of transition from one stage to another, latency to sleep onset and period elapsed until REM stage onset provide information about sleep architecture. Sleep fragmentation caused by frequent arousals or respiratory disturbances lead to nonrestorative sleep and impairment in quality of life [6].

When AHI is below 5, sleep efficiency seems to be similar in both sexes; however in case of OSAS several measures of sleep quality, namely sleep efficiency, sleep latency and REM latency reveal poorer sleep architecture in women. Women with AHI more than 30 have significantly longer stage 1 and stage 3 sleep and significantly shorter stage 2 sleep, compared to men. On the other hand, percentages of NREM and REM stages did not seem to differ between men and women in case of AHI below 30.

Taking the whole group into consideration, women have poorer sleep quality diagnosed by PSG. They have lower sleep efficiency and longer latency to sleep onset and longer latency to REM onset. The main difference has to be attributed to the OSAS group. The anxiety of sleeping in an unfamiliar environment, knowing to be observed by a stranger all through the night and having the electrodes and other equipment on face and body may be more pronounced in women.

Prevalence estimates of sleep disordered breathing vary in males and females. In the past, studies on sleep disorders have been biased towards men, therefore sleep disordered breathing in women has been underestimated. Male/female ratio of sleep apnea is thought to be 8/1 in clinical patient populations and 2-3/1 in the general population [7]. The difference of OSAS incidence between male and female subjects decreases with increasing age [8]. It is clear that postmenopausal women are as susceptible for OSAS, as men. One explanation for this could be based on the fact that BMI in postmenopausal group is significantly increased compared to premenopausal period. Obesity is a major risk factor for development of OSAS in women. Obesity is a well-known risk factor for OSAS. Body mass index, waist, hip and neck circumferences, waist/hip ratio and skinfold thickness are strong predictors of sleep disordered breathing [1,7].

In OSAS group, independent of stage, BMI was higher in women compared to male counterparts. This finding is consistent with several reports [9,10]. Guilleminault, *et al.* found that massive obesity was a major cause of OSAS in women [11]. In Greece, a neighbouring country, gender differences on polysomnographic findings were investigated and in Greek population, BMI in male and female subjects with OSAS were similar, female OSAS patients were older, AHI was higher in male subjects and sleep quality was worse in women [12]. A similar report from our country revealed that there was no difference due to BMI in male and female subjects with OSAS and female patients were significantly older [13]. Inconsistent with these reports, another study performed in Saudi Arabia, showed that Saudi women had higher BMI than male counterparts [9]. Japanese women with OSAS also had higher BMI than AHI-matched men [10].

Compared to previous studies on gender perspectives in OSAS, our study population is much larger; nevertheless there are some limitations of the current work. The study group may not represent the entire population because it consists of subjects who had applied for sleep study due to existing symptoms. The prevalence of OSAS in general population and male and female subgroups still remains unknown. The cephalometric measurements, which are suggested to have impact on OSAS severity, were not recorded. Another limitation is that comorbidities and smoking habits of the whole group could not be reached, therefore comparisons based on clinical characteristics could not be made.

Conclusion

As a conclusion; men have about twice higher incidence of OSAS and more severe disease. Despite the fact that OSAS is more common among male population, women carry a significantly increased risk after menopause. Obesity is a major factor that this increase can be attributed. Physicians should promote greater attention on the risk of sleep disordered breathing in overweight women. Sleep quality is

worse in female OSAS subjects compared to male counterparts. This may influence the clinical presentation and daytime symptoms. As a conclusion, gender is one of the major factors influencing PSG data and should be taken into consideration in evaluating OSAS cases.

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Conflict of Interest

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent

Informed consent was obtained from all individual participants included in the study.

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