

Gender differences in obstructive sleep apnoea in an elderly French population

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Abstract

Obstructive sleep apnoea (OSA) affects differently women and men, and increases in prevalence with age. We characterize the clinical, anthropometric and polygraphic gender differences in a large elderly OSA population.

641 subjects aged 68 yr were examined. Measurement of fat mass using dual-energy X-ray absorbsiometry (DEXA) and polygraphy were obtained in all subjects. An apnoea+hypopnoea index (AHI) >15 identified the presence of OSA.

OSA was diagnosed in 57% of the sample, 34% having a mild form and 23% having an AHI>30. OSA women had a lower AHI, less severe hypoxemia, greater peripheral fat mass and reported frequently anxiety and depression. Comparison of women with and without OSA did not show significant differences in clinical, anthropometric and DEXA data. After adjustment for BMI, hypertension, diabetes, smoking, anxiety and depression, logistic regression analysis revealed that presence of hypertension (Odd Ratio=1.52, $p=0.04$) was significantly associated with OSA risk in women.

In a general community healthy population, the prevalence of undiagnosed OSA in women increases with age, with a risk similar to men. In women, the clinical spectrum, the anthropometric data and the fat distribution appear to be more gender-related than OSA-dependent. The occurrence of OSA contributes to hypertensive risk in elderly women.

Key words: elderly, gender, obesity, hypertension, sleep apnoea, symptoms

Introduction

According to epidemiological studies on obstructive sleep apnoea (OSA) prevalence, aging and gender substantially affect the incidence of the disorder, with rates rising from less than 15% in middle aged adults ¹ up to 24-38% in older adults.^{2,3} Gender seems to affect the incidence of the disease, OSA being more prevalent in men than in women ^{4,5} with a male:female ratio of 8-10:1 or greater in clinical studies, and around 2:1, 3:1 in epidemiological studies. The reason for the reduced susceptibility to OSA in women is not entirely clear and many factors have been evoked, such as body fat distribution ⁶, upper airways shape⁷, craniofacial morphology ⁸, and hormonal influences.^{9,10,11} Women with OSA are less likely to have the classic symptoms of OSA such as snoring and apnoea, they report less frequently sleepiness and more frequently fatigue, depression and anxiety ¹², and they have lower apnoea-hypopnea index, frequently confined to rapid-eye movement sleep (REM sleep).¹³ Therefore OSA may be underdiagnosed in women due to a different clinical profile. Concerning the cardiovascular consequences, results are controversial, some authors reporting a greater incidence in men ¹⁴ while others reporting similar data between genders. ^{15, 16}

Since OSA in older adults may not have the same functional consequences seen in middle-aged adults^{17, 18, 19, 20} it will be interesting to determine whether even in elderly subjects some factors differentiate men and women with OSA.

The objectives of this study were to examine the gender differences in sleep related symptoms, sleepiness, mood disorders, fat distribution and cardiovascular risk in a large cohort of community-dwelling elderly persons aged less than 70 years and having OSA. The age-matched population, the high proportion of women, the extensive examination as well as the presence of a non-OSA sample, allowed us to assess whether, in older women, clinical and anthropometric differences are related more to gender characteristic rather than OSA.

Methods

Subjects

Subjects for the study were selected from the participants of the Prognostic indicator of cardiovascular and cerebrovascular events study (PROOF study),²¹ a prospective cohort study of 1011 community-dwellers aged 65 (± 2.0) yr, randomly recruited from the electoral lists of the city of Saint-Etienne, France. An ancillary study addressing the association between OSA, assessed by at-home polygraphic study, and cardiovascular and cerebrovascular morbidity during a 7-yr follow-up was proposed to participants (SYNAPSE study). Of the original sample of 1011, 851 subjects participated in the SYNAPSE study. When compared to those who refused polygraphy, the final sample did not differ in any variables, including educational level, gender, daytime sleepiness, and incidence of prior disease. From this sample 641 subjects, aged $68.5 \pm$

0.06 yr, having a complete evaluation including clinical interview, anthropometric measurements, 24-hour blood pressure measurement, polygraphy and body composition analysis using the dual-energy X-ray absorptiometry (DEXA), were included in the current study. None of the examined women was taking hormone replacement therapy.

The study was approved by the local ethics committee (CPPRB Rhône-Alpes Loire) and written informed consent for participation was obtained from all individuals.

Clinical assessment

Detailed clinical assessment was focused on cardiac and cerebrovascular disease, hypertension, diabetes, and respiratory, neurological and psychiatric disorders. Current medication was analyzed with regard to antihypertensive, hypnotic, anxiolytic and/or antidepressant therapy. Subjects were defined as normotensive if they did not report history of hypertension (HTA) and antihypertensive treatment, and at the ambulatory blood pressure monitoring they did not have a mean systolic blood pressure >135 mm Hg and a mean diastolic >85 mm Hg.

Anthropometric and DEXA measurements

Body mass index (BMI) was calculated as weight (kg)/height squared (kg/m^2). Neck circumference (NC) was measured in the midway of the neck between mid-cervical spine and mid-anterior neck to 0.5 cm just below the laryngeal prominence. Waist circumference (WC) was measured midway between the lower rib margin and the iliac crest and hip circumference (HC) measured at the level of the two bony prominences in front of hips.

Regional (arms, legs, trunk and head) body fat and lean body mass were measured with a whole-body DEXA scanner (Hologic QDR-2000, Hologic Inc., Bedford, MA, USA) within 4 months after polygraphy. The standard procedures for DEXA measurement were applied.²² Peripheral fat mass (PFM) was calculated by adding the fat mass of the legs to that of the arms and central fat mass (CFM) was calculated by adding the fat mass of the trunk to that of the head.

Questionnaires

Depressive symptoms were assessed using the QD2A questionnaire²³ and subjects with a score >6 were considered as having depressive symptoms. Anxiety was assessed using the French version of the Goldberg scale, individuals with a score >4 considered as anxious.²⁴

The impact of sleepiness was evaluated using the Epworth Sleepiness Scale (ESS)²⁵ and an excessive daytime sleepiness was retained for a score ≥ 10 .

The Berlin Questionnaire has been proposed to screen for OSA in the general population.²⁶ Patients were considered to be at high risk if they had a positive response to at least two of the following three criteria: (i) persistent symptoms for at least two snoring questions, (ii) persistent somnolence during daytime and/or while driving (>3 times per week), and (iii) history of hypertension or a BMI >30 kg/m^2 .

All questionnaires were administered at the time of sleep study.

Sleep study

Nocturnal unattended home-sleep study was performed in all subjects using a polygraphic system (HypnoPTT, Tyco Healthcare, Puritan Bennett, CO,USA), which included the following parameters: sound measurement, electrocardiogram, pulse transit time, R-R timing, airflow by nasal pressure, thoracoabdominal respiratory efforts by one inductance plethysmography, and body position. Oxygen saturation (SaO₂) was measured by pulse oximetry. A software package was used for downloading the tracings and all recordings were visually validated and manually scored for respiratory events and nocturnal SaO₂ with an intrascorer reliability of 88%. A recording was considered acceptable if at least five hours of recording without missing data on respiratory signals or SaO₂ was obtained. A second night of monitoring was performed when subjective sleep latency exceeded two hours on the first night, sleep duration was shorter than five hours or when the respiratory recording was considered as not acceptable. Scoring was done following the standard criteria proposed for polygraphy.²⁷ Hypopnoea was defined as a 50% or greater reduction in airflow lasting at least 10 s and associated with at least 3% oxygen desaturation. Apnoeas were defined as the absence of airflow on the nasal cannula lasting >10 s. The apnoea + hypopnoea index (AHI) was established as the ratio of the number of apnoeas and hypopnoeas per hour of recording. As indices of nocturnal hypoxemia we considered the mean SaO₂, the % of recording time below 90%, the minimal SaO₂ value and the oxygen desaturation index (ODI) i.e., the number of oxygen desaturations $\geq 3\%$ per hour of recording time. Using PTT,²⁸ an autonomic respiratory-related and total arousal index was calculated. An AHI >15 with at least 50% of events scored as obstructive was considered diagnostic of OSA²⁹. Cases were stratified as mild (AHI>15<30) and moderate to severe (AHI>30).

Statistical analyses

Subjects' characteristics were summarized as means \pm SEM for continuous variables, and counts and percentages for categorical variables. The groups were compared using an unpaired Student's t-test for variables measured on a continuous scale, and, where appropriate, one-way ANOVA with post-hoc Scheffé comparison.

Logistic regression analysis was used to examine the effect of clinical and anthropometric data on gender with adjustment for the covariates such as hypertension, diabetes, dyslipidemia, anxiety, depression, smoking, BMI and AHI. All statistical analyses were conducted using the

SPSS statistical software package (SPSS for Windows, version 12.0, SPSS, Chicago, IL). After Bonferroni's correction, statistical significance was taken at $p<0.05$.

Results

General sample

A total of 379 women and 262 men (Table 1), were analysed. Compared to males, females had lower BMI and NC and reported more frequently being diagnosed and treated for hyperlipidemia, anxiety and depression. Men reported more frequently to be smokers and diabetic. The prevalence of HTA was 42.6%, similar in both sexes.

An AHI>15 was identified in 369 (57%) subjects, with 34% having a mild form and 24% having an AHI>30 (Table 2). Significant increase in odds ratio for AHI>30 was found in men (OR: 3.02, 95%CI 2.00-4.56, $p<0.001$), 33% of men being at greater risk to have an AHI>30 (women 16.6%).

Differences between women and men with OSA

Within the AHI>15 group (Table 2), past history revealed a high percentage of females having hyperlipidemia and intake of antidepressants and benzodiazepines. Females more frequently did not report snoring and apnoea at the first item of Berlin questionnaire ($p=0.005$), they self-perceived to be less sleepy at the ESS, and more anxious ($p<0.05$) and depressed ($p<0.001$). OSA men were more frequently smokers and diabetic. HTA was more frequent in men than in women but the difference did not reach statistical significance ($p=ns$).

Table 3 shows anthropometric, polygraphic and DEXA data in the OSA group according to gender. Overall, men had a greater BMI compared to women. In the overweight group (BMI>25), men were proportionally more frequent (67%), whereas in the pathologically obese group (BMI>30) women were proportionally higher (16%). Women had lower NC and WC compared to men. Comparison of DEXA data according to gender (Table 3) revealed that while CFM did not differ between sexes, women had a significantly higher PFM.

Polygraphic findings in males and females with OSA are reported in Table 3. Females had less severe AHI, more hypopnoeas than apnoeas, and lower values of ODI and time <90% ($p<0.05$). Only 16% of women had an AHI>30. No differences in autonomic arousal index, reported sleep time (men vs women: 6.8 ± 0.1 h vs 6.7 ± 0.1 h), and mean oxygen saturation (men: $95.0\pm0.1\%$, women: $95.3\pm0.1\%$) were found between men and women with OSA.

Overall, sleepiness was not frequently reported, the mean ESS score being 5.6. 58 subjects, 40 men and 18 women reported an ESS \geq 10.

Differences between subjects with and without OSA

Compared to the 186 women without OSA (Table 2), women with OSA were more frequently positive at the Berlin questionnaire ($p<0.01$) and more frequently reported HTA (45.6% vs. 35.5%, $p=0.04$) without differences for the other clinical data. When we considered medication, we noted that OSA women had a greater intake of antidepressants and a trend to increased use of anxiolytics. Considering anthropometric data, women with OSA (Fig.1, upper panels) had greater BMI (25.6 ± 0.3 vs. 24.2 ± 0.3 Kg/m², $p<0.01$) and a greater neck circumference (OSA women: 34.9 ± 0.2 cm vs. women without OSA: 34.0 ± 0.2 cm, $p=0.02$) without differences for waist and hip circumference. Comparison of DEXA data (Fig.1, upper panels) did not shown any significant differences among women with and without OSA, central, total and peripheral fat mass similar in both groups.

Comparison of men with and without OSA showed that OSA men reported more frequently HTA than men without OSA and scored more positively at the Berlin questionnaire. DEXA and anthropometrics data (Fig.1, bottom panels) did not shown any significant difference between men with and without OSA, central fat mass being the only parameters showing a trend to no significant rise in OSA men.

Factors associated with OSA in women

To examine the effect of clinical, anthropometric and DEXA data on OSA risk in women we employed multiple logistic regression analysis. The final model was adjusted for BMI, NC, depression and anxiety scores, medication, history of HTA, dyslipidemia and blood pressure. Gender was associated with HTA, women having increased HTA risk (OR=1.52, 95%CI: 1.00-2.30, $p=0.04$) and increased 24-h systolic blood pressure (OR= 1.03, 95%CI: 1.00-1.5, $p=0.006$) compared to men.

Discussion

To our knowledge, this is the first epidemiological study examining a large cohort of healthy elderly subjects and investigating the gender differences in clinical, anthropometric and risk factors in subjects having OSA. Firstly, as expected, the gender differences in OSA decrease with age, the ratio of OSA risk being 1.31 in our study population. Secondly, when we consider clinical symptoms of OSA, as in middle-aged studies, women reported less frequently the cardinal symptoms of OSA and had a higher risk to have had, or to be treated for mood and anxiety disorders. Although depression and anxiety scores were higher in OSA women, comparison of women with and without OSA showed that there were no significant differences between groups for anxiety and depression scores as well as for previous psychiatric treatment. Therefore, gender differences in personality characteristics^{12, 30} more than the presence of OSA, contribute to clinical spectrum in OSA females. Thirdly, OSA women have increased risk of

pathological obesity and higher values of peripheral obesity compared to men. However, comparison of women with and without OSA shows the lack of significant differences in anthropometrics and DEXA values, suggesting that obesity and fat distribution do not contribute to OSA risk in elderly women. Finally, analysis of factors associated with OSA shows that hypertension and increased systolic blood pressure are the factors associated with the increased OSA risk in older women but not in men. This relationship should be replicated in a large clinical population in order to define if the cardiovascular risk of OSA in the elderly is a specific gender effect.

The ratio of men to women with OSA in clinical studies appears to be considerably higher than in community studies.^{4,10} The reason for this discrepancy is not clear, and misdiagnosis related to lack of specific clinical complaints has been postulated, depressive symptoms, benzodiazepine and antidepressant treatment and tiredness more commonly reported in women.^{5,12} The major limitation of all studies conducted so far was that the sample size was relatively small and the effects of confounding factors such as age were not carefully accounted for. To overcome some bias of previous studies, we chose to analyse data collected from nearly 650 subjects drawn from a community-based cohort matched for age, one of the factors mostly contributing to OSA risk in females. In our population, the approximately 1.3 male-female risk prevalence of OSA reflects ratios previously reported for community studies underlying similarities between our sample and other cohorts. Therefore, a greater OSA risk is present in the elderly equally in men and women, explaining the high cardiovascular morbidity, use of psychoactive medications and health care utilization in older adults.³¹

Consistent with previous studies,^{2,4,10} we found gender differences in the cardiovascular and metabolic risks, HTA and diabetes more commonly reported in men (49.7% and 9.7% respectively) than in women (HTA 45.6% and diabetes 3.6%). No significant difference for HTA was found between sexes. With regard to clinical symptoms, women reported less frequently snoring and apnoea, had greater depression and anxiety scores and more frequently reported treatment for mood and anxiety disorders. The comparison of women with and without OSA, however, showed that none of these atypical symptoms significantly differ between groups, suggesting that they are more related to a gender phenotype in the psychological profile rather than presence of OSA.¹²

Among factors contributing to OSA in older women, aging, body fat distribution^{6,32} and hormonal influences⁹ have been proposed. Since in our sample, all female cases were postmenopausal and did not report substitutive treatment, we were able to examine the specific role of obesity and fat distribution in OSA occurrence. As in middle-aged women,^{11,15} our females were more likely to be obese (BMI>30) and to have a significantly higher percentage of

their fat mass at the periphery. Even though these data are in line with the previous reports in middle-aged patients,^{15,32} the type of obesity did not completely explain the observed differences in our population. The CFM did not differ significantly between OSA men and women, and no significant differences in the anthropometric and DEXA data were observed between women with and without OSA. Therefore, we can suggest that, in older subjects, the effects of obesity and fat distribution alone do not greatly contribute to the occurrence of the disease, increased collapsibility and aging-related changes in the upper airway^{33, 34} probably playing a major role. Interestingly, we found that women with OSA had increased use of anxiolytics, medication that increasing the pharyngeal collapsibility might explain the rise in OSA incidence in our women. When we tried to explain which factors contribute to the occurrence of OSA in our older women we found that hypertension was the only one. Previous data on prevalence of hypertension in an OSA population have reported controversial results, some authors reporting an equal risk in male and female OSA groups³⁵ and others¹⁴ showing an association between OSA and hypertension almost entirely restricted to the male sex. These differences may be explained by several factors, such as composition of groups, criteria to define hypertension, and coexistence of traditional risk factors such as obesity, diabetes, smoking and hyper-lipidaemia. Our finding has clinical consequences, older women with OSA being at greater risk for hypertension and cardiovascular diseases^{35,36} compared to women without OSA. This increased susceptibility to hypertension in older OSA women may be related to gender differences in endothelial dysfunction³⁷ or genetic predisposition³⁸ and susceptibility³⁹ to hypertension, as suggested in middle-aged patients. Given that OSA is a risk factor for hypertension in older women, powered studies examining gender differences in response to treatment are needed to confirm this gender association. In the interpretation of our results some limitations need to be considered. First, participants were recruited from a cross-sectional community-based population aged 68 yr at the study entry for which previous medical, heart, neurological and cardiovascular diseases were excluded. In addition, the majority of the sample did not report the clinical symptoms of sleep-related breathing disorders, as common in elderly patients.⁴⁰ Therefore, the age at the study entry, the exclusion criteria and the lack of typical symptoms may limit our evaluation to “very healthy” and “very young” elderly, and results cannot be extrapolated to clinic-based samples. Second, we found a high prevalence of OSA in our sample, half of our subjects having an AHI>15. An explanation for this high prevalence might be the use of an ambulatory polygraphy that may include respiratory events occurring during wakefulness, inducing an overestimation of apnoeas

and hypopnoeas. Despite this limitation the polygraphic monitoring has been applied in middle-aged and elderly population.⁴¹

In conclusion, in a community elderly population, the incidence of undiagnosed OSA in women is similar to that found in men. The atypical clinical spectrum and the fat distribution in OSA women appears to be more gender-related than OSA-dependent. The presence of OSA predisposes our women to increased risk of hypertension, stressing the need for increased awareness of the disease and prompt therapy in elderly female populations.

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Legend to Figure.

Figure 1. Histograms showing the anthropometric (BMI: body mass index; HC: hip circumference; NC: neck circumference WC: waist circumference) and dual-energy X-ray absorbsiometry (DEXA) (FM: fat mass) data in the groups of women (upper panel) and men (bottom panels) with and without OSA. No significant differences in anthropometric and DEXA data were detected between groups.

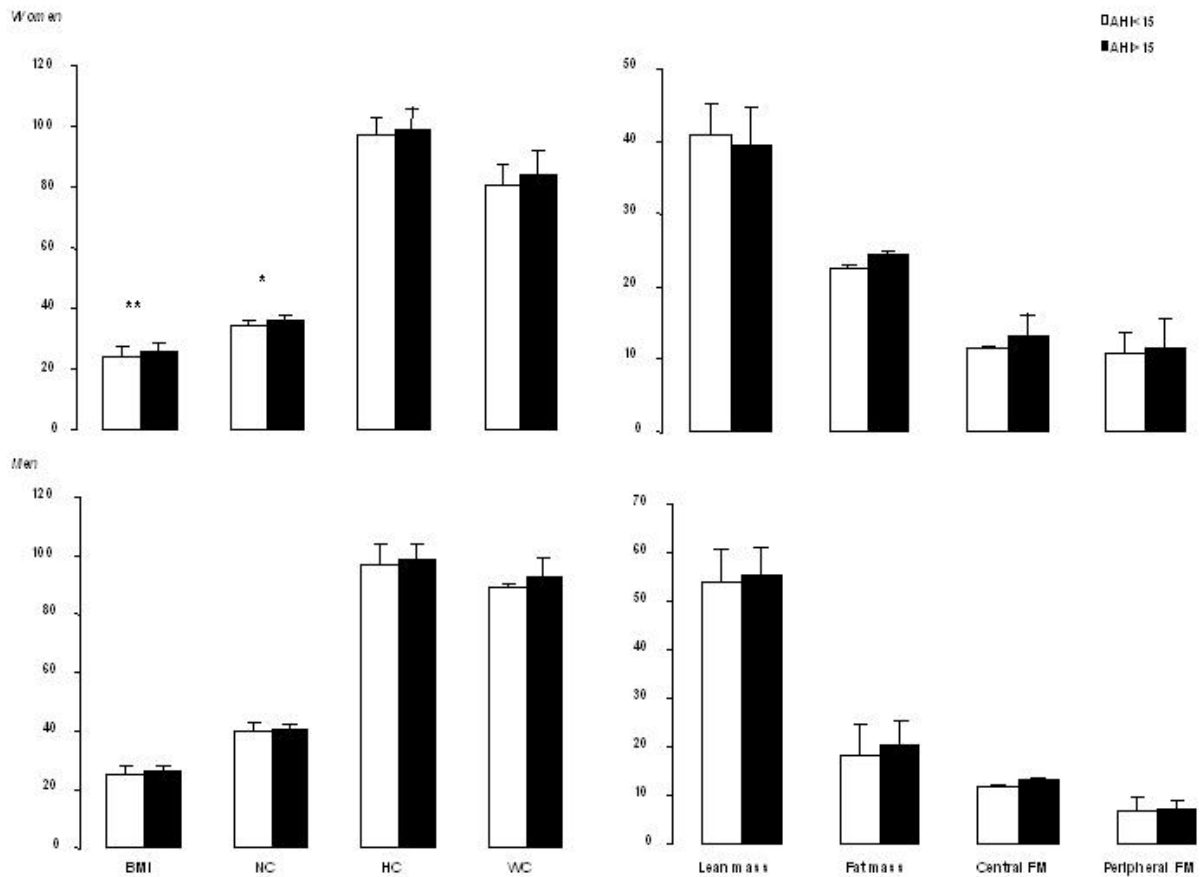


Fig. 1

Table 1. Clinical data of the studied population (n:641) according to gender (Mean \pm SEM).

	Men	Women	p*
N (%)	262 (40.9)	379 (59.1)	
Age (yr)	68.5 \pm 0.05	68.6 \pm 0.04	ns
BMI (Kg/m ²)	26.1 \pm 0.2	24.8 \pm 0.2	<0.001
Neck circumference (cm)	40.3 \pm 0.2	34.4 \pm 0.1	<0.001
Ambulatory SBP (mmHg)	121.1 \pm 0.98	116.8 \pm 0.7	ns
Ambulatory DBP (mmHg)	76.6 \pm 0.4	72.6 \pm 0.4	ns
<i>Clinical data.</i>			
Hypertension %	45.4	40.6	ns
Diabetes %	9.1	3.4	0.002
Dyslipidemia %	29.4	38.5	0.01
Smokers %	43.9	12.7	<0.001
Hypnotic treatment (%)	1.1	2.4	ns
Antidepressant treatment (%)	2.3	9.8	<0.001
Anxiolytic treatment (%)	5.7	9.5	0.05
<i>Questionnaire data</i>			
Anxiety score	2.4 \pm 0.1	3.9 \pm 0.2	<0.001
Depression score	1.8 \pm 0.1	3.0 \pm 0.2	<0.001
ESS score	6.3 \pm 0.2	5.1 \pm 0.2	ns

Legend: N: number of subjects; BMI: Body Mass Index ; SBP: systolic blood pressure; DBP: diastolic blood pressure; ESS: Epworth Sleepiness Scale

*: comparison between women and men based on independent samples *t*-test or chi-square test, as appropriate.

Table 2. Clinical and anthropometric data for the samples with and without an AHI>15 according to gender (mean \pm SEM).

	<i>AHI<15</i>		<i>AHI>15</i>	
	Men (n=86)	Women (n=186)	Men (n=176)	Women (n=193)
Age (yrs)	68.5 \pm 0.08	68.5 \pm 0.06	68.6 \pm 0.06	68.7 \pm 0.06
BMI (Kg/m ²)	25.3 \pm 0.31	24.2 \pm 0.3	26.4 \pm 0.2	25.5 \pm 0.3* ⁺
Ambulatory SBP (mmHg)	119.8 \pm 1.5	114.2 \pm 1.0	122.0 \pm 1.0	119.2 \pm 1.0+
Ambulatory DBP (mmHg)	75.7 \pm 0.7	71.9 \pm 0.5	77.0 \pm 0.5	75.8 \pm 0.6++
<i>Clinical data.</i>				
Hypertension %	37.2	35.5	49.7#	45.6++
Diabetes %	8.1	3.2	9.7	3.6*
Dyslipidemia %	27.9	37.6	30.3	39.4*
Smokers %	46.5	13.4***	42.9	22***
Hypnotic treatment (%)	0	2.1	1.7	2.6
Antidepressant treatment (%)	2.3	10.2**	2.3	19.3**+
Anxiolytic treatment (%)	10.5	8.1	9.4	10.9**+
<i>Questionnaire data</i>				
Anxiety score	2.3 \pm 0.2	3.9 \pm 0.2***	2.5 \pm 0.2	4.0 \pm 0.2***
Depression score	1.5 \pm 0.2	2.8 \pm 0.2***	1.9 \pm 0.1	3.2 \pm 0.2***
ESS score	5.7 \pm 0.4	4.9 \pm 0.2	6.7 \pm 0.3	5.3 \pm 0.3
Positif Berlin questionnaire (%)	20.9	19.9	42.1##	37.8 ⁺⁺⁺

Legend: see Table 1

*: comparison between men and women with (AHI>15) and without (AHI<15) OSA using one-way ANOVA with post-hoc Scheffé test. * $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

+ : comparison between women with (AHI>15) and without (AHI<15) OSA using one-way ANOVA with post-hoc Scheffé test. $p \leq 0.05$; ++ $p \leq 0.01$; +++ $p \leq 0.001$

: comparison between men with (AHI>15) and without (AHI<15) OSA using one-way ANOVA with post-hoc Scheffé test# $p \leq 0.05$; ## $p \leq 0.01$; ### $p \leq 0.001$

Table 3. Anthropometric and body composition measurements of OSA subjects (AHI>15) according to gender (mean \pm SEM).

	Men	Women	p-value*
<i>Anthropometric measurements</i>			
BMI (kg/m ²)	26.4 \pm 0.2	25.5 \pm 0.3	<0.001
BMI > 25 (%)	67.0	47.7	<0.001
BMI>30 (%)	11.0	15.5	0.005
HC (cm)	99.1 \pm 0.5	98.9 \pm 0.7	ns
WC (cm)	92.7 \pm 0.7	84.0 \pm 0.8	0.004
Waist/Hip	0.93 \pm 0.005	0.85 \pm 0.005	ns
NC (cm)	40.4 \pm 0.2	34.9 \pm 0.2	<0.001
<i>DEXA measurements</i>			
Body mass (kg)	78.5 \pm 8.6	65.8 \pm 8.5	0.01
BMC (kg)	2.49 \pm 2.7	1.86 \pm 2.9	0.01
Total lean mass (kg)	55.4 \pm 5.6	39.5 \pm 5.2	0.004
Total FM (kg)	20.4 \pm 4.9	24.5 \pm 0.6	0.001
Central FM (kg)	13.2 \pm 3.2	13.1 \pm 3.1	ns
Central FM, % Total FM	64.8%	53.0 %	0.001
Peripheral FM (kg)	7.2 \pm 2.0	11.6 \pm 4.0	<0.001
Peripheral FM (%Total FM)	35.5 %	47.0%	<0.001
<i>Polygraphic study</i>			
Subjective total sleep time (min)	6.80 \pm 0.1	6.68 \pm 0.1	ns
Respiratory AAI (n/h)	22.0 \pm 0.9	18.2 \pm 0.5	<0.001
Snoring index (n/h)	65.6 \pm 6.9	75.8 \pm 6.8	ns
AHI (n/h)	36.1 \pm 1.4	27.1 \pm 0.8	<0.001
ODI (n/h)	15.6 \pm 0.9	10.8 \pm 0.5	<0.001
Mean SaO ₂ (%)	95.0 \pm 0.1	95.3 \pm 0.1	ns
Time SaO ₂ % <90% (min)	2.70 \pm 0.53	1.95 \pm 0.13	0.05
Minimal SaO ₂ (%)	88.6 \pm 0.3	89.9 \pm 0.3	ns

Legend: **BMC:** bone mineral content; **BMI:** body mass index; **HC:** hip circumference; **NC:** neck circumference; **DEXA:** dual-energy X-ray absorbsiometry; **FM:** fat mass; **WC:** waist circumference; **AAI:** autonomic arousal index; **AHI:** apnea+hypopnea index; **ODI:** oxygen desaturation index.

*:comparison between women and men based on independent samples *t*-test

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