

Frequency and Factors Associated with Obstructive Sleep Apnea in Adults. Cali-Colombia 2014-2016

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Abstract

Introduction: Obstructive sleep apnea (OSA) is a common entity present in 9% to 38%, but with an important underreporting of 85%¹. Classically, it is observed in middle-aged, obese and sleepy men², closely related to multiple comorbidities of cardiovascular³⁻⁶, respiratory⁷ and metabolic^{8,9} and is associated with increased mortality. Recent studies indicate that majority of people with type 2 diabetes also has OSA. The aim of this study was to assess the prevalence and severity of OSA and risk factors contributing to it among people with chronic and severe type 2 diabetes. Methods A total of 203 people with type 2 diabetes (mean age: 54 ± 8 years, 145 males, 58 females, HbA1c ≥ 7% [53mmol/mol] types, generating high costs of health systems.¹⁰ The purpose of this study was to determine the frequency and identify the clinical factors associated with obstructive sleep apnea within the adult population of a specialized center in Cali, Colombia.

Materials and Methods: Observational, analytical case-control study nested within a cohort. First-time polysomnography records were analyzed in adults with clinical suspicion of OSA in a specialized center. The characteristics of the study population were summarized through descriptive statistics. Associations were determined through OR, 95% CI and values of $p \leq 0.05$ taken as significant for the statistical tests. Through multivariate logistic regression, a 6-variable model was identified, where the 6 variables independently explain the event.

Results: We analyzed 566 polysomnographies, where the prevalence of OSA was 85.3% (483 of 566, 95% CI 82.4-88.35%), the mean age was 51.80 ± 13.73 years and 50% were men. The final model included male gender (OR 4.46 95% CI 2.04-8.04, $p < 0.000$), hypertension (OR 3.78 95% CI 2.48-8.04, $p < 0.000$), Mallampati grade IV (OR 4.14, 95% CI 2.41-7.10, $p < 0.000$) and excessive sleepiness (OR 5.70 95% CI 1.66-19.53, $p < 0.006$) and normal weight (OR 0.48 95% CI 0.24-0.97, $p < 0.043$).

Conclusion: The predictive probability showed that being male, hypertensive with Mallampati grade IV and excessive sleepiness are associated independently with OSA, while normal weight decreased the risk.

Key words: Obstructive sleep apnea; Polysomnography; Associated factors

Introduction

Obstructive sleep apnea (OSA) is an entity that belongs to the group of breathing disturbances during sleep¹¹, and is the product of recurrent upper airway collapse induced by sleep, which reduces (hypopnea) or stops (apnea) the airflow to the respiratory system; this causes hypoxemia and hypercapnia that excite the cortical area of the nervous system resulting in an activation of the pharyngeal muscles, thus

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recovering the permeability of the airway¹². According to the American Academy of Sleep Medicine, the following conditions are necessary for an OSA diagnosis: signs and symptoms (associated somnolence, fatigue, insomnia, snoring or observed apnea) or associated medical or psychiatric disorders (that is to say, hypertension, coronary heart disease, atrial fibrillation, congestive heart failure, stroke, diabetes, cognitive dysfunction or mood disorder) together with five or more respiratory events mostly obstructive per hour of sleep during the polysomnography. A 15/h frequency of obstructive respiratory events meets the criteria, even in the absence of symptoms or associated disorders¹³. The classification of severity was made basing on the frequency of said events into: mild (AHI: 5-15 events/hour), moderate (AHI: 16-30 events/hour) and severe (AHI > 30 events/hour)¹⁴.

The prevalence of OSA in the general population oscillates between 9% and 38%, increasing with age, reporting a prevalence of up to 84% within the geriatric population¹. However, the disease is not widely suspected, thus a > 85% underreporting is estimated^{15, 16}.

A close relationship has been shown between the OSA and multiple comorbidities of the cardiovascular type, such as myocardial infarction, need for coronary revascularization, development of heart failure and cardiovascular death in 2.5 times³. The risk of suffering a stroke increases 6% for every increase in AHI⁴, and the atrial fibrillation increases three times in comparison with the general population⁵. In a hypertensive population, it may affect up to 40%, even 90% in cases of resistant hypertension⁶. This indicates that for every increase in the AHI the risk of developing resistant hypertension increases 1%, whereas for every 10% decrease in overnight oxygen saturation, the risk of developing OSA increases 13%¹⁷.

With regard to the respiratory disease, the OSA may increase mortality in patients with chronic obstructive pulmonary disease up to 1.79 times⁷ and show even double the postoperative cardiopulmonary complications¹⁸ with a stronger probability to desaturate and being transferred to the intensive care unit after the surgery¹⁹.

The OSA is also associated with metabolic disorders. A prospective evaluation performed in a diabetes treatment center in India found that 23.65% of diabetic patients had OSA (AHI \geq 15), with a higher percentage of diabetic complications such as cardiovascular disease, retinopathy and neuropathy⁸. The Sleep Heart Health Study showed that this entity affects mainly men, in whom a 10 kg weight gain increases five times the risk of elevating the AHI > 15⁹; it is also reported that 45% of patients who underwent bariatric surgery have OSA, obtaining after the procedure a three times reduction of their AHI, thus improving minimum oxygen saturation, sleep efficiency and the latency of rapid eye movement sleep (REM sleep)².

A study carried out in Italy estimates that medical costs generated by the treatment of comorbidities of patients with OSA add up to €2.9 trillion, representing 55% of total medical costs in Italy, 6% of which belong to the diagnosis and treatment of OSA, and 49% due to lack of diagnosis and prevention of related comorbidities. So, timely detection of OSA would save the governments €1.5 trillion¹⁰. Other costs derived from OSA are related to low productivity, days lost from work and work accidents²⁰ and car crashes caused by microsleep²¹.

Given the high prevalence, the high underreporting, the close relationship with frequent comorbidities and limited information available in Colombia about this topic, this research has the purpose of determining the frequency and identifying the clinical factors associated with the obstructive sleep apnea-hypopnea syndrome within the adult population in a specialized center in Cali, Colombia.

Materials and Methods

Design

We performed an observational, case-control study nested in a cohort of patients treated at a center that specializes in pulmonology, immunology, cardiology and cardiac and lung rehabilitation who showed clinical suspicion of OSA and had been referred by specialized physicians for diagnosis confirmation. Data collection was carried out in a retrospective manner.

Study Population

We analyzed the records of adult patients of 18 years or older who went for the first time to AIREC specialized medical group, in the city of Cali, Colombia, due to clinical suspicion of OSA to be confirmed by diagnostic polysomnography type I performed with the Philips Respironics Alice-6 polysomnograph. Records showed at least 4 hours of sleep (TST > 240 min). We excluded the records of previous polysomnographies of pregnant women, patients with craniofacial deformities, tumors in the upper airway, restrictive lung disease, history of tuberculosis, incomplete clinical information limiting the performance of the corresponding analyses or evidence of previous OSA treatment with continuous positive airway pressure (CPAP).

Every record with AHI ≥ 5 event/hour determined through polysomnography, positive for OSA was considered as case, and every record with AHI values of less than 5 measured during the same period from January 2014 until September 2016 was defined as control.

Statistical Analysis

In order to sum up the characteristics of the study population we used descriptive statistics. Quantitative variables were presented through their measures of central tendency and of dispersion. In order to compare data distribution, we used the Kolmogórov-Smirnov Test and considered the values of $p \leq 0.05$ as significant. Qualitative variables were summed up as proportions and were presented in frequency tables. Prevalence was estimated as a proportion, taking the number of OSA positive cases as numerator and all patients with suspicion of OSA as denominator. Also, the stratified general prevalence of the event was estimated according to severity.

In order to explore possible associations between exposure variables and result variables (having or not having OSA), we used the odds ratio (OR) as a measure of association, with its respective confidence intervals of 95% (95% CI). The statistical significance of associations between numerical variables was calculated with the Mann Whitney U Test, and for dichotomous variables we conducted the Chi2 or Fischer Test as applicable. We considered the values of $p \leq 0.05$ as significant to reject null hypotheses.

In order to adjust possible confounding factors and determine the weight of each variable in the presence of the obstructive sleep apnea-hypopnea syndrome, a binary logistic regression was used. When building the models, we included the variables that showed a statistical significance of ≤ 0.20 in the bivariate analysis. Modelling was made through the Backward strategy and the most parsimonious model was presented through the Hosmer-Lemeshow Test. For the degree of success of the model we used the ROC (receiver operating characteristic) curve. All the analyses were carried out with software Stata® (Stata Corp, 2011, Stata 12 Base Reference Manual, College Station, TX, USA).

Ethical Considerations

This study has been approved as minimal risk research by the research bioethics committee of the Universidad Libre and the institutional committee, having expressed there isn't any kind of conflict of interest.

Results

Between January 2014 and September 2016, 1089 subjects with suspicion of OSA were evaluated in the institution through polysomnography. The subjects had been referred by the following medical specialties: internal medicine, family medicine, otorhinolaryngology, neurology, psychiatry, pulmonology, cardiology and rheumatology. After applying the selection criteria, 566 records were analyzed in this study. Exclusion criteria are shown in **Figure 1**.

Mean age was 51.80 ± 13.73 years, and 50% were male. According to the polysomnography report, the general prevalence of OSA was 85.3% (483 of 566, 95% CI 82.4-88.35%), where the most frequent was mild OSA in 42.2% (AHI 8.4 ± 3.0), with similar behavior between moderate OSA (27.3% with AHI 21 ± 4.1) and severe OSA (30.5% with AHI 49.2 ± 18.7), with higher prevalence in men (92.6%,

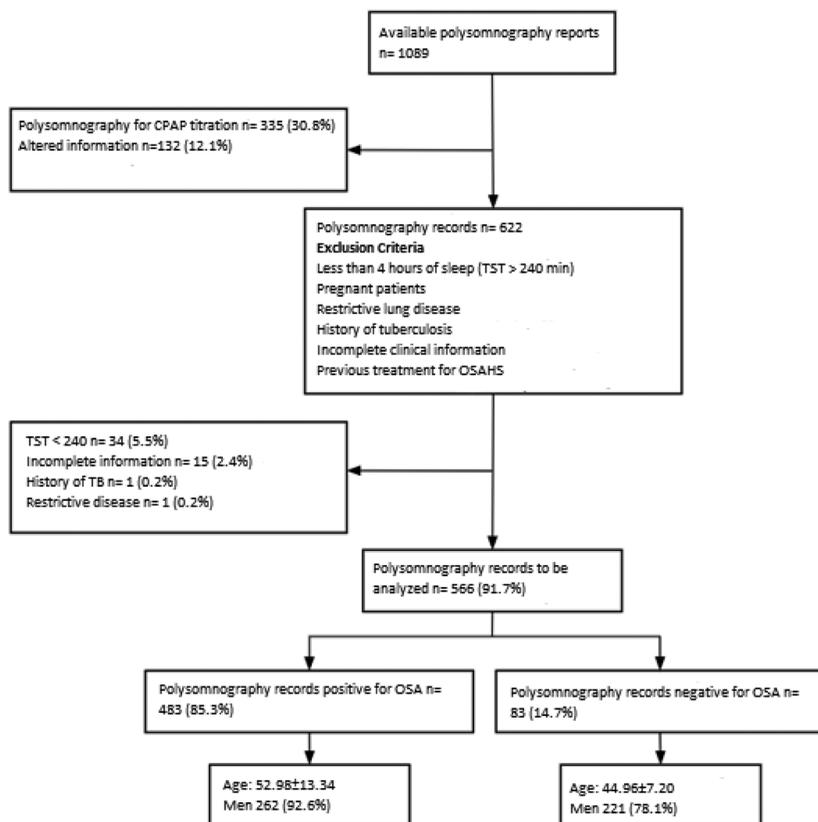


Figure 1. Polysomnography diagnostic records made at the institution between January 2014 and September 2016 n=1089

95% CI 89.5-95.6%) than in women (78.1%, 95 % CI 73.3-89.5%, $p < 0.001$), showing a mean AHI of 26.05 ± 21.89 event/hour that increases when the patient is in supine position (AHI of 32.76 ± 25.82 event/hour).

As shown in **Figure 2**, the general prevalence of OSA showed positive linear growth regarding age ($p < 0.001$), a phenomenon that was also observed in women; men, on the other hand, showed the opposite behavior (negative linear growth, $p < 0.001$); only in the age group of 60 to 69 years the OSA distribution was the same for both men and women.

Tables 1 and 2 show the distribution of the study population and prevalence of OSA according to sociodemographic variables, comorbidities, and clinical and polysomnographic characteristics.

The analysis showed that the probability of having OSA is 3.5 times higher in men compared to women (95% CI 2.02-6.23, $p < 0.000$), 3.35 times higher in hypertensive patients (95% CI 1.91-6.10, $p < 0.000$), 4.89 times higher in patients with Mallampati grade IV (95% CI 2.90-8.28, $p < 0.000$) and 4.75 times higher in patients with excessive sleepiness (95% CI 1.50-24.09, $p < 0.001$). There were other important clinical variables such as diabetes mellitus (OR 2.25 95% CI 0.94-5.35, $p < 0.061$), arrhythmias (OR 4.86 95% CI 0.78-201.14, $p < 0.089$) and obesity (mild OR 1.36 95% CI 0.76-2.53, moderate OR 1.73 95% CI 0.82-4.09 and morbid OR 2.97 95% CI 0.73-26) with a higher probability of presenting the event, but these results are not statistically significant. One of the factors reducing the probability of the event is normal weight (OR 0.29 95% CI 0.16-0.55, $P < 0.000$) and an Epworth scale < 11 points, which means normality (OR 0.47 95% CI 0.26-0.82, $p < 0.004$), (**Table 1**).

The average duration of sleep latency time was longer in the group without OSA (29.85 ± 23.47 min vs. 23.26 ± 21.63 min, $p < 0.005$), not showing onset insomnia (> 30 minutes). Regarding the total

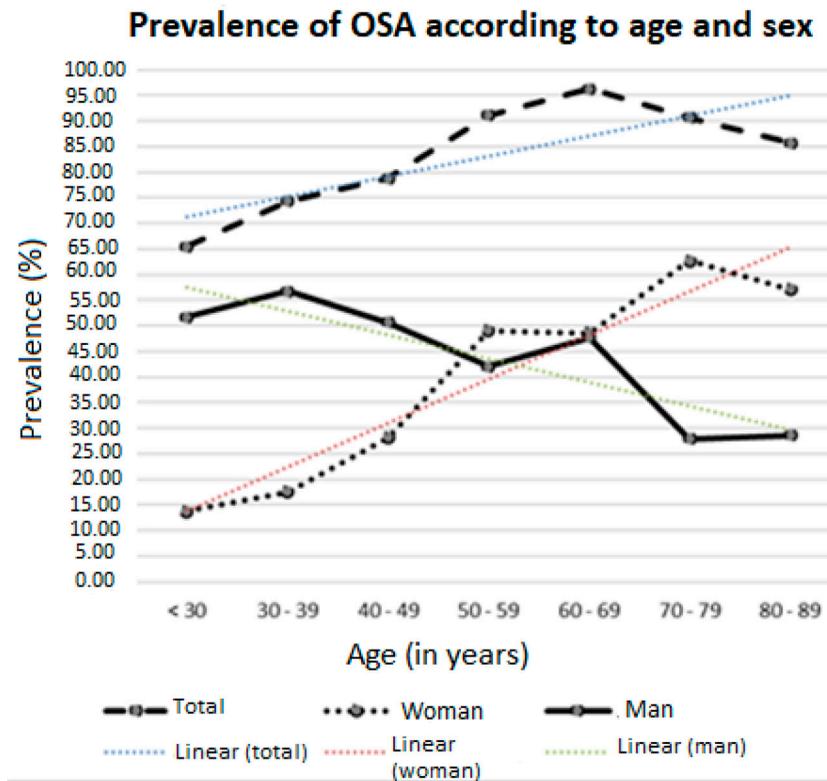


Figure 2. Prevalence of OSA according to age and sex

sleep time (TST), the average duration of the OSA group was shorter than the group without OSA (346.60 ± 41.50 min vs 386.09 ± 339.03 min, $p < 0.005$); however, in terms of sleep efficiency, there weren't statistically significant differences between the groups and the average was adequate, $84.70 \pm 38.16\%$. Patients with OSA showed higher desaturation in the different stages of sleep and during respiratory events, in comparison with patients without OSA (Table 1).

The multiple logistic regression analysis showed a model in which the predictor variables included gender, AHT, arrhythmias, Mallampati, BMI and Epworth scale. It was identified that men (adjusted OR 4.46 95% CI 2.04-8.04, $p < 0.000$), hypertensive patients (adjusted OR 3.78 95% CI 2.48-8.04, $p < 0.000$), patients with Mallampati grade IV (adjusted OR 4.14 95% CI 2.41-7.10, $p < 0.000$) and patients with excessive sleepiness (adjusted OR 5.70 95% CI 1.66-19.53, $p < 0.006$) were significantly and independently associated with OSA. The lower probability of having OSA with normal weight was 52% (adjusted OR 0.48 95% CI 0.24-0.97, $p < 0.043$), setting this parameter as a protective factor. As summarized in Table 3, there wasn't a significant association with other factors.

As shown in Figure 3, we evaluated the model's degree of success of the event 3. compared to the real cases that were presented through a ROC curve with 86.35% of correct classification.

We conducted the Hosmer-Lemeshow test obtaining a non-significant p value of 0.4130, thus concluding that the model may be considered acceptable to explain the study data.

Discussion

This is the first research made in Cali about the frequency and factors associated with the presence of OSA in patients with high clinical suspicion. Our study showed 85.3% prevalence of OSA, in accordance with the information published by Morales et al in Bogotá indicating 75.7%²² prevalence in this

TABLE 1. Demographic and clinical characteristics of the study population and determinant factors for OSA

Characteristic		n	OSA (n = 483)	No OSA (n = 83)	OR (95% CI)	p values
Gender	Men	283 (50%)	262	21	3.50 (2.02-6.23)	0.000
	Women	283 (50%)	221	62		
Smoking		7 (1.2%)	6	1	0.98 (0.12-48.01)	0.977
AHT		260 (45.9%)	241	19	3.35 (1.91-6.10)	0.000
DMT2		78 (13.7%)	72	6	2.25 (0.94-6.55)	0.061
COPD		30 (5.3%)	23	7	0.54 (0.22-1.55)	0.168
Asthma		51 (9.0%)	41	10	0.68 (0.32-1.59)	0.295
Rhinitis		58 (10.2%)	48	10	0.81 (0.38-1.87)	0.558
GERD		54 (9.5%)	46	8	0.99 (0.44-2.52)	0.974
Nasal deviation		65 (11.5%)	57	8	1.25 (0.56-3.17)	0.568
CHF		16 (2.8%)	14	2	1.21 (0.27-11.15)	0.804
Coronary artery disease		26 (4.6%)	24	2	2.12 (0.51-18.81)	0.304
Stroke		13 (2.3%)	13	0	0.59	0.131
Arrhythmia		28 (4.9%)	27	1	4.86 (0.78-201.14)	0.089
Epworth Scale	Normal	351 (62.0%)	288	63	0.47 (0.26-0.82)	0.001
	Very high certainty	139 (24.6%)	122	17	1.31 (0.73-2.48)	
	Excessive sleepiness	76 (13.4%)	73	3	4.75 (1.50-24.09)	
Mallampati	1	1 (0.18%)	1	0		0.000
	2	15 (2.72%)	9	6	0.24 (0.07-0.85)	
	3	132 (23.95%)	92	40	0.24 (0.14-0.41)	
	4	403 (73.14%)	369	34	4.89 (2.90-8.28)	
Tonsils	0	28 (5.1%)	22	6		0.107
	1	426 (78.0%)	361	65	0.79 (0.40-1.48)	
	2	64 (11.7%)	55	9	1.06 (0.49-2.54)	
	3	27 (4.9%)	27	0	1.27	
	4	1 (0.18%)	1	0		
BMI classification	Low weigh	2 (0.3%)	2	0		0.000
	Normal weigh	64 (11.3%)	43	21	0.29 (0.16-0.55)	
	Overweight	222 (39.2%)	189	33	0.97 (0.59-1.62)	
	Mild	278 (49.1%)	132	18	1.36 (0.76-2.53)	
	Moderate	142 (24.7%)	84	9	1.73 (0.82-4.09)	
Morbid	93 (16.4%)	33	2	2.97 (0.73-26.00)		
Sleep efficiency Yes		279	238	41	1.00 (610-1.63)	0.984
SO ₂ classification min %	Mild	231/559 (41.3%)	164	67		0.000
	Moderate	259/559 (46.3%)	248	11	6.61 (3.35-14.17)	
	Severe	69/559(12.4%)	68	1	12.87 (2.16-522.29)	

AHT = arterial hypertension. DMT2 = Diabetes mellitus T2. COPD = chronic obstructive pulmonary disease. GERD = gastroesophageal reflux disease. CHF = congestive heart failure. BMI = body mass index. SO₂ = oxygen saturation.

TABLE 2. Polysomnographic characteristics

Sleep latency - min	24.37 ± 22.01
Sleep efficiency - average in %, standard deviation	84.70 ± 38.16
TST - average in minutes	352.39 ± 135.45
Microarousal index - average of index per hour, standard deviation	27.58 ± 20.35
AHI - average of index per hour, standard deviation	22.45 ± 22.00
sO ₂ average during vigil - average in %, standard deviation	94.48 ± 2.61
sO ₂ average during respiratory events - average in %, standard deviation	89.66 ± 6.23
T90 - average in %, standard deviation	7.42 ± 16.71
Sleeping time with snoring - average in minutes, standard deviation	11.47 ± 12.53
Minimum sO ₂ during sleep - average in %, standard deviation	81.11 ± 10.61
AHI in supine position - average in index per hour, standard deviation	28.43 ± 26.17
AHI on right side - average in index per hour, standard deviation	11.93±24.80
AHI on left side - average in index per hour, standard deviation	1.90±2.47

TST = total sleep time. T90 = oxygen saturation below 90 (%). sO₂ = oxygen saturation

TABLE 3. Multivariate analysis of factors determinant of OSA

Characteristic	n	OSA (n = 483)	No OSA (n = 83)	OR (95% IC)	p value	Adjusted OR (95%CI)	p value
Gender: men	283	221	62	3.50 (2.02-6.23)	0.000	4.46 (2.04-8.04)	0.000
AHT	260	241	19	3.35 (1.91-6.10)	0.000	3.78 (2.48-8.04)	0.000
Arrhythmia	Si	27	1	4.86 (0.78-201.14)	0.089	4.53 (0.57-35.90)	0.153
Mallampati grade 4	403	369	34	4.89 (2.90-8.28)	0.000	4.14 (2.41-7.10)	0.000
Normal BMI	64	43	21	0.29 (0.16-0.55)	0.000	0.48 (0.24-0.97)	0.043
Epworth scale: excessive sleepiness	76	73	3	4.75 (1.50-24.09)	0.0045	5.70 (1.66-19.53)	0.006

AHT = arterial hypertension. BMI = body mass index

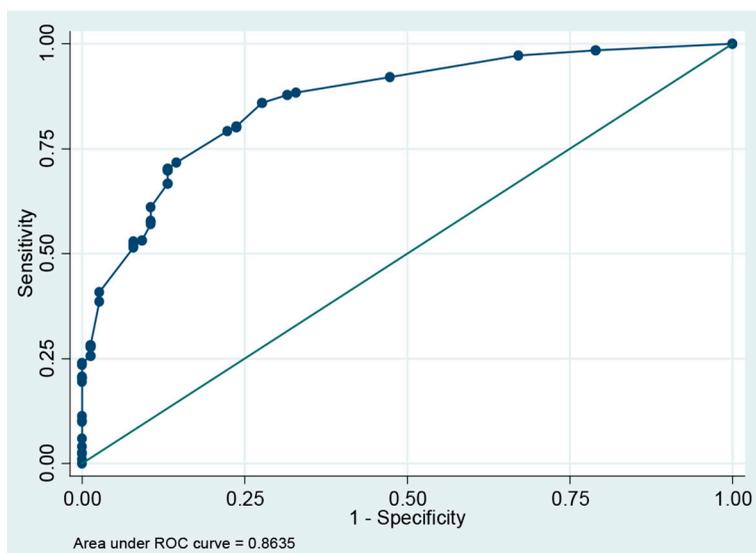


Figure 3. Correct classification of the adjusted model

kind of population. The possible cause of this phenomenon is selectivity of patients admitted to this specialized center, because they have high clinical suspicion before the test. Another relevant aspect is the identification of 45.9% of records with history of arterial hypertension, 92.7% of which have OSA, which may impact on the described prevalence.

Approximately 40% of hypertensive patients have OSA, reaching even 90% in patients with refractory hypertension⁶. In Cali, Colombia, González et al published 67.4% prevalence of OSA in the hypertensive population²³, which is lower than the values observed in this study.

It has been reported that arterial hypertension increases the probability of having OSA, because during sleep healthy people reduce diastolic and systolic numbers of arterial tension between 10 to 15%, in comparison with the vigil status. This phenomenon is known as “dipping”. However, it has been observed that in patients with OSA this phenomenon is reduced during sleep, thus causing a higher cardiovascular risk²⁴.

The Wisconsin Sleep Cohort, a study that began in 1988 with a random sample of 2,940 state employees in Wisconsin which provided essential data to estimate the prevalence of non-diagnosed breathing disturbances during sleep in adults and its importance for public health²⁵ and The Sleep Heart Health, a multicentric study that began in 1995 including 6 cohorts already in process with a sample of 6,441 participants, designed to prove if breathing disturbances during sleep are associated with a higher risk of cardiovascular mortality and morbidity²⁶ are the basis for understanding the natural history of sleep breathing disturbances, showing a positive linear relationship between the prevalence of hypertension and the severity of OSA, with a 2.7 times higher probability of showing hypertension if the disorder is mild and 4.5 times higher if it is moderate to severe.²⁷ In our study, the adjusted probability of developing OSA is three times higher in hypertensive patients.

With regard to the age variable, aging is a factor of risk at its peak as a consequence of the greater longevity of the general population. At present, 1 every 4 polysomnographies done in Spain are performed in individuals of 65 years or older, due to a higher clinical suspicion in this population²⁸. In Korea, a linear association was evidenced between age and the high risk of having OSA, which is higher in people of 60 years and older, where the prevalence is equaled between men and women²⁹. Such behavior is evidenced in this study, with the difference of the negative linear growth seen in men.

Classically, obese and sleepy men represent the OSA phenotype³⁰. As observed in this study, OSA is more prevalent in men than in women (92.6% vs. 78.1%, $p < 0.001$), with a 4.5 times higher probability of developing OSA. This finding is corroborated in the multiple studies available up to now, with prevalence values between 13% and 33%¹.

Obesity (mainly centripetal) has an important association with OSA, since it increases the pharyngeal collapsability by mechanical effects in the soft tissues of the pharynx and the restriction generated at the lung volume level, apart from affecting the neuromuscular control of the airways exercised by the central nervous system². In this study we observe that the higher the degree of obesity, the greater the opportunity for the event; however, this result is not statistically significant, unlike what happens with normal weight, considered a protective factor.

The phenotype of excessive daytime sleepiness is most common in men and is related to an increase in the hypoxemic load (more apneas versus hypopneas) and is associated with neurocognitive, metabolic and cardiovascular impairment and a great impact on the quality of life³¹, using the Epworth scale to evaluate the frequency. It was positive in 1 every 8 evaluated records and was present in 96.1% of patients with OSA, as opposed to other studies where half the patients with sleep apnea had this condition³². Laranjeira et al established that the Epworth sleepiness scale is not connected with the disease³³. The same was observed in patients with moderate OSA, where the presence of excessive sleepiness was associated with a higher risk of hypertension, but interaction between the AHI and the Epworth score wasn't statistically significant³⁴. In this case, the logistic regression model informs that having intense sleepiness provides 4.7 times higher probability of having OSA and a scale in the normality range provides a protective factor for the event.

The anthropometric test is a secondary tool used for making the clinical diagnosis of OSA during the physical examination. Few of those tools provide a direct correlation with the presence or severity of

the disease, except for the Mallampati classification³⁵, given that with Mallampati grade III or IV the relative risk of having OSA is 1.95 times, and 2.45 times if we add nasal obstruction³⁶. In this study, Mallampati grade IV provides a 3.14 times higher probability of having OSA.

The physiopathological phenomena present during OSA could explain the independent association with other comorbidities such as T2 diabetes, where intermittent hypoxemia and sleep fragmentation lead to a modification of glucose metabolism, reaching 17% reduction of insulin sensitivity and an increase in plasma glucose levels without changes in insulin secretion³⁷. But we should take into account the confusion effects that may be generated by aging and obesity in the bidirectional association and the reverse causality between breathing disturbances during sleep and T2 diabetes³⁸.

Strausz et al showed an association with OSA in patients with T2 regardless of obesity, indicating that the risk of diabetic kidney disease increases 1.75 times in patients with OSA, as well as mortality for all the causes. In our study, despite the fact that T2 diabetes increases 2.3 times the probability of having OSA, results weren't statistically significant³⁹.

It has been shown that patients with severe OSA had higher rates of heart arrhythmia⁴⁰, 3 times higher probability of atrial fibrillation, 2 times higher probability of non-sustained ventricular tachycardia and almost double the probability of complex ventricular ectopy (bigeminy, trigeminy, quadrigeminy)⁴¹. In our study we can observe a 4 times higher probability of having OSA when heart arrhythmias are detected, but results weren't statistically significant.

The strength of the study is the degree of success and plausibility of the model that was found which explains the data obtained, possibly due to the number of records included, much higher than those reported in other studies of this type. On the other hand, given the limited information available on a national level, this type of study allows us to know more about the local difficulties, having as limiting factor the fact that the behavior of the event is only shown inside a specialized medical center; results can't be replicated in other populations.

Despite the model's degree of success, there is residual confusion not allowing us to provide a full explanation of this disease; due to the retrospective condition of the study it is impossible to make an analysis of other variables that could possibly intervene and are not available in the polysomnographic records or in the given clinical records, thus generating an information bias. So, further prospective, preferably multicentric research is needed that allows for other variables such as risk assessment questionnaires, additional anthropometric tests, quality of life, anxiety and depression and the different characteristics of possible clinical phenotypes of sleep obstructive apnea, amongst other things, with the purpose of generating a hypothesis about the event.

OSA is a complex and heterogeneous disorder, since it depends on multiple elements such as risk factors, clinical physiopathological characteristics and comorbid conditions and not simply on the polysomnographic results, because the AHI alone is not enough to make a diagnosis, not producing all the necessary tools for the approach and treatment of this entity⁴².

Conclusion

OSA is common in this population possibly because it is influenced by the prevalence of arterial hypertension, which has proven to increase the probability of having this disease. It can be explained to a large extent by clinical variables such as gender, weight, blood pressure, Mallampati classification and excessive sleepiness, all of them independently associated. Knowing these determinant factors will allow clinicians to suspect the presence of OSA.

This research provides knowledge of determinant factors within a local population with OSA. With this information, it would be possible to generate a proposal to develop diagnostic algorithms that could approximate to the results obtained with the polysomnography, the gold standard for the diagnosis.

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Conflict of interest

The authors declare that there isn't any kind of conflict of interest.

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