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Impact of smoking on obstructive sleep apnea: a retrospective study

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Abstract

Obstructive sleep apnea (OSA) has several well-established risk factors. Smoking has been documented as a risk factor for several comorbidities associated with OSA. However, its specific contribution to the severity of OSA and its potential as an independent risk factor require further investigation. In this study, we have evaluated the association between smoking behavior and the severity of OSA and have examined the related sleep parameters and comorbidities. A retrospective analysis was conducted on 567 patients who underwent diagnostic polysomnography. Participants were grouped by OSA severity and smoking status (non-smokers, ex-smokers, and current smokers). Demographic and clinical characteristics, comorbidities, and sleep-related indices were compared across groups. Logistic regression analyses were used to identify predictors of severe OSA. Current and ex-smokers had significantly higher apnea-hypopnea index values (p=0.033), more oxygen desaturation, and lower sleep efficiency (p=0.021) compared to non-smokers. Psychiatric comorbidities and higher Mallampati scores were also more prevalent in the smokers' group. Smokers showed a 1.98 times higher risk of severe OSA as compared to non-smokers in univariate analysis (odds ratio=1.98; p=0.036) but were not retained as an independent risk factor in the multivariate model. Ex-smokers continued to show worse sleep parameters than non-smokers, indicating possible long-term effects of tobacco exposure. These results highlight the need for comprehensive management strategies that address both respiratory and systemic impacts in OSA patients with a smoking history.

Key words: obstructive sleep apnea, smoking, apnea-hypopnea index, obesity.

Introduction

Obstructive sleep apnea (OSA) is a prevalent condition characterized by intermittent collapse of the upper airway during sleep, leading to repeated episodes of partial or complete airway obstruction. These events result in fragmented sleep, intermittent hypoxia, and disturbances in nocturnal oxygenation and sleep architecture. OSA is commonly associated with loud snoring and can lead to daytime sleepiness, fatigue, and impaired cognitive function and an array of metabolic abnormalities. Epidemiological studies indicate that the prevalence of OSA among Americans aged 30 to 70 is 5% for women and 14% for men [1]. On a global scale, it is estimated that 936 million people are affected by OSA, with China having the largest burden, accounting for approximately 176 million cases [2]. A meta-analysis from India suggests that approximately 104 million working-age individuals suffer from OSA, with about 47 million having moderate-to-severe forms of the disorder, which presents a significant public health challenge [3].

OSA is associated with a wide range of comorbidities, which can be broadly classified into cardiovascular, metabolic, neuropsychiatric, pulmonary, and renal pathologies. These include diabetes, insulin resistance, hypertension, mental health disorders, cognitive dysfunction, sexual dysfunction, non-alcoholic fatty liver disease (NAFLD), cerebrovascular accidents (strokes), asthma, hypothyroidism, gastroesophageal reflux disease (GERD), and various orthodontic deformities [4,5]. Smoking is a well-established risk factor for respiratory diseases, and previous studies have demonstrated an association between smoking and several OSA-related comorbidities, such as asthma, metabolic syndrome, insulin resistance, hypothyroidism, depression and cardiovascular diseases [6-12].

The impact of smoking on OSA, however, remains an area of debate. Although smoking is known to impair respiratory function, studies on its direct effect on OSA severity have yielded inconsistent results [13]. Some studies suggest that smoking may worsen OSA severity. For instance, smokers with OSA tend to have higher AHI, lower nocturnal oxygen saturation, and higher Epworth Sleepiness Scale (ESS) scores, indicating more severe sleep apnea and greater daytime sleepiness compared to non-smokers [14]. Ex-smokers with OSA often experience prolonged periods of hypoxia during sleep, particularly those who were heavy smokers [15]. This might suggest that the long-term effects of smoking continue to impact OSA even after cessation. Conversely, some studies, including those by Hsu and Casasola, found no significant correlation between smoking and OSA severity, indicating that the relationship may not be as straightforward as previously thought [16,17]. Recent meta-analyses comprising data from over

5,000 participants investigated the association between OSA and alcohol, caffeine, and tobacco use. The study found a positive association between OSA and alcohol consumption, but insufficient evidence to confirm associations with caffeine or tobacco [18]. Although smoking's direct impact on OSA severity remains unclear, it is widely acknowledged that smoking is a major risk factor for numerous complications associated with OSA. The combined effects of smoking and OSA on health could potentially be synergistic, amplifying the risk of cardiovascular, respiratory, and other systemic conditions. While most studies investigating the relationship between smoking and OSA have been conducted in Western populations, primarily comprising Caucasian and Black individuals, there is a notable lack of research focusing on Indian populations. Given the potential influence of genetic, environmental, and lifestyle on the relationship between smoking and OSA, research in diverse populations is crucial for understanding the broader implications of this association [19].

In light of these inconsistencies, this study aims to investigate the relationship between smoking and OSA among patients at our tertiary care center in India. By comparing clinical characteristics, polysomnographic findings, and comorbidities between smokers and non-smokers, we seek to contribute to a more nuanced understanding of the impact of smoking on the severity and outcomes of OSA in an Indian cohort.

Materials and Methods

Study design and data collection

This retrospective, single-centre study was conducted at a tertiary care hospital in India, covering the period from January 2016 to December 2024. We reviewed patient records from the hospital's pulmonary department OSA database and extracted detailed clinical information, including demographics (age, sex), anthropometric data, smoking history, alcohol consumption, medication use (specifically sleeping pills), comorbidities, and OSA-related symptoms and signs. Patients under the age of 20 and those with incomplete clinical records were excluded. All adult patients (≥18 years) who underwent overnight home-based level 2 polysomnography and had complete demographic, clinical, and smoking-related data were included in the analysis. Patients were excluded if they had incomplete or poor-quality sleep recordings and predominant central sleep apnea. A total of 567 patients who met the inclusion criteria were included in the study. Institutional ethics committee approval was obtained, and only de-identified data were used for the retrospective analysis (EC outward number- 02/2025).

Polysomnography and obstructive sleep apnea classification

The diagnosis of OSA in this study was established based on the polysomnographic assessment performed during the study period; pre-existing OSA diagnoses were not used for classification. Overnight home-based polysomnographic testing (level 2) was performed on all patients, and the results were used to calculate the AHI. As per the ICSD-3 framework, an apnea event was identified when airflow dropped by about 90% from baseline and the reduction persisted for a minimum of 10 seconds. Depending on whether respiratory effort was present, events were categorized as obstructive, central, or mixed. A hypopnea was noted when airflow decreased by roughly 30% or more for at least 10 seconds, accompanied by either a fall in oxygen saturation of 3% or greater, or a cortical arousal. OSA severity was classified based on AHI as follows: no sleep apnea (NSA) (AHI < 4.9), mild OSA (AHI 5-14.9), moderate OSA (AHI 15-29.9), and severe OSA (AHI ≥ 30). In accordance with the international classification of sleep disorders (3^{rd} edition), OSA is defined as the AHI≥5 with related symptoms viz., fatigue, excessive daytime sleepiness, snoring, gasping or choking while sleeping, pause in breathing during sleep or AHI≥15 with or without the related symptoms. The diagnostic framework followed the ICSD-3, and relevant aspects of the updated AASM 2021 guideline were considered for aligning diagnostic and management principles.

Smoking history classification

For the purpose of analyzing smoking history, patients were divided into three groups according to their reported smoking habits. The Smokers group included individuals who were actively smoking, either on a regular or occasional basis. The ex-smokers group consisted of individuals who had previously smoked but had quit at least six months prior to their first consultation at our hospital [20]. Non-Smokers group comprised individuals who had never engaged in smoking.

Modified Mallampati score

The modified Mallampati score (MS) was used as a non-invasive physical examination tool to assess the degree of oropharyngeal crowding. The MS was graded as follows:

- Class 0: Any part of the epiglottis is visible
- Class I: soft palate, uvula, and pillars are visible
- Class II: soft palate and uvula are visible
- Class III: only the soft palate and base of the uvula are visible

- Class IV: only the hard palate is visible

Statistical methods

The continuous variables (characteristics) were summarized in terms of mean and standard deviation, while the categorical variables were expressed as frequencies and percentages. The difference in the means of continuous variables across OSA severity groups was determined using one-way analysis of variance, while the association of categorical variables with OSA groups was obtained using Pearson's chi-square test. The comparison of continuous variables between two groups was performed using t-test for independent samples. The risk of severe OSA associated with different characteristics was assessed in terms of odds ratio, while the adjusted odds ratios for the selected characteristics were obtained using multiple logistic regression. OSA severity was treated as dependent variable and the characteristics as independent predictors in the model. All the analyses were performed using SPSS version 26.0 (IBM corp., ARMONK USA) and the statistical significance was tested at 5% level of significance.

Results

Table 1 presents the distribution of patient characteristics according to OSA severity, categorized by AHI**.** Patients with moderate and severe OSA had a higher proportion of males (p=0.003), obese patients - particularly in BMI Grades II and III (p=0.002), and a significantly higher prevalence of smoking compared to those with mild OSA and NSA (p=0.042). In contrast, the mild OSA and NSA groups exhibited higher use of sleeping pills (p=0.031) and recorded greater proportion of patients with gastroesophageal reflux disease (GERD) in mild OSA group (p<0.001) and obstructive airway disease (OAD) in NSA and mild OSA group with p=0.015. Refer to Table 1 for details.

In the gender-based comparison (refer to Table 2), females were older (p=0.002)and showed a higher prevalence of Grade III obesity (p<0.001), type 2 diabetes mellitus (p=0.02), OAD (p=0.007), and hypothyroidism (p<0.001), as well as more frequent use of sleeping pills (p=0.002) as compared to males. No female patients reported smoking or alcohol consumption. Males had significantly higher AHI scores (p<0.001), whereas females demonstrated better sleep efficiency (p=0.004) but lower average oxygen saturation levels (p=0.021).

Table 3 provides a comparison of patient characteristics based on smoking habits among 567

participants, comprising 455 non-smokers, 58 ex-smokers, and 54 current smokers. The mean age of ex-smokers was significantly higher (55.22 years; SD: 12.09) than that of non-smokers and current smokers ($p = 0.004$). Alcohol consumption, both occasional and regular, was significantly more prevalent among ex-smokers and current smokers compared to non-smokers ($p < 0.001$). OAD was more prevalent in non-smokers (23.08%) compared to the other two groups ($p = 0.034$). Psychiatric disorders were significantly more common among ex-smokers and current smokers ($p = 0.041$), and the proportion of patients with a higher MS was significantly more in the smoker and ex-smoker groups compared to the non-smoker group ($p=0.048$). Importantly, the mean AHI was significantly higher among ex-smokers (AHI = 32.37±27.56) and current smokers (AHI = 40.56±29.85) with $p = 0.033$, while mean sleep efficiency was significantly lower in these two (ex- smokers = 54.55±22.86 % and current smokers = 52.94±20.62 %) groups ($p = 0.021$).

When ex-smokers and current smokers were combined into a single group and compared with non-smokers, similar patterns were evident. The proportion of alcohol users remained significantly higher in the smoker group ($p < 0.001$). Non-smokers continued to show a higher prevalence of OAD ($p = 0.04$). The distribution of MS remained significantly different ($p = 0.048$), with higher score in the smoker group. Mean AHI was significantly higher in the smoker group ($p = 0.023$), while mean sleep efficiency was significantly greater in non-smokers ($p = 0.006$).

The risk factors for severe OSA associated with different patient characteristics is given in Table 4. Each characteristic was independently assessed for its effect on severe OSA through univariate logistic regression analysis. Characteristics showing significant effect were considered together in the multiple logistic regression model to determine the adjusted risk. In the univariate analysis, sex, BMI, smoking, consumption of sleeping pills, GERD, OAD and MS showed statistically significant effect on the severity of OSA. Males showed 1.94 (95% CI: 1.36 to 2.78; $p < 0.001$) times higher risk of severe OSA as compared to females. Further BMI grade II and III showed 3.10 (95% CI: 1.34 to 7.38; $p=0.009$) and 2.82 (95% CI: 1.20 to 6.83; $p=0.018$) times higher risk of severe OSA as compared to patients with normal BMI. Smokers showed 1.98 (95% CI: 1.07 to 3.86; $p=0.036$) times higher risk of severe OSA as compared to non-smokers. Consumption of sleeping pills, presence of GERD and OAD showed significantly reduced risk of severe OSA in patients. Each increase in the grade of MS was associated with 1.13 times increased risk of severe OSA.

The multivariate model revealed that the males have 1.79 (95% CI: 1.18 to 2.71; $p=0.006$)

times increased risk of severe OSA as compared to females. Patients with BMI grade II and III had 2.68 (95% CI: 1.11 to 6.52; p=0.029) and 2.71 (95% CI: 1.09 to 6.72; p=0.032) times increased risk of severe OSA respectively as compared to normal BMI patients. Patients with GERD and OAD showed significantly reduced risk of severe OSA with adjusted OR of 0.39 (95% CI: 0.19 to 0.79; p=0.009) and 0.62 (95% CI: 0.39 to 0.95; p=0.030) respectively.

Discussion

The present study demonstrated a statistically significant association between smoking behavior and OSA, particularly in relation to the severity of the condition, as patient characteristics varied notably across different levels of OSA severity. When stratifying patients by smoking history, current and ex-smokers demonstrated a tendency toward more severe OSA compared to non-smokers. Smokers exhibited significantly higher AHI values, greater oxygen desaturation, and longer durations with oxygen saturation below 90% during sleep. These findings are consistent with results from a 2020 Clinical Respiratory Journal study, which similarly reported that smokers exhibit higher AHI and more pronounced nocturnal desaturation than non-smokers [21]. These observations suggest that smoking may exacerbate OSA severity, consistent with findings from Yosunkaya et al and Oţelea et al [22,23]. Although smoking did not emerge as an independent risk factor in multivariate analysis, its role in exacerbating OSA is strongly suggested by univariate associations and significant association with clinical parameters similar to what was observed by Zeng X et al who described smoking as a key aggravating factor in OSA pathophysiology [24]. Biologically, smoking contributes to upper airway inflammation, mucosal thickening, and reduced neuromuscular tone, all of which increase airway collapsibility during sleep. Chronic smoking may also lead to nasal congestion, impaired mucociliary clearance, and disruption of reflexes that maintain airway patency [25]. Wisconsin Sleep Cohort Study reported that active—but not former—smoking was associated with a higher risk of developing moderate or severe OSA, even after adjusting for confounding variables, particularly among heavy smokers [26]. This distinction supports the hypothesis that the ongoing inflammatory and neuromuscular effects of active smoking contribute more directly to OSA pathogenesis than residual effects in ex-smokers.

In our study however, even ex-smokers, despite cessation, continued to exhibit higher OSA severity than non-smokers possibly due to the long-term structural and inflammatory changes caused by prior tobacco exposure. Varol Y et al. reported a significantly higher prevalence of moderate to severe OSA among current and ex-smokers compared to non-smokers [27]. On

the other hand, Hsu et al. found no significant association between cigarette smoking and OSA after adjusting for age, sex, and BMI. While the unadjusted analysis showed a positive association (OR = 1.51), this was not sustained in the adjusted model (OR = 1.02), suggesting that smoking may not independently predict OSA when major confounders are accounted for [16]. A systematic review and meta-analysis of 13 studies indicated that while smoking behavior was significantly associated with OSA, the relationship was complex because the association between smoking and OSA severity was not straightforward, and was influenced by smoking duration, intensity (e.g., pack-years), BMI, age, sex, and the presence of comorbidities [24]. These findings suggest that while smoking is often associated with more severe OSA symptoms and indices, its role as an independent risk factor remains inconclusive as evidenced by the findings in our study.

In addition to its direct impact on OSA severity, smoking was associated with several comorbidities, especially psychiatric disorders such as depression and anxiety, which were more common among current and former smokers. Prior studies by Leonard et al., Hahad et al., and Fluharty et al. have reported positive associations between smoking and mental health issues—including poor sleep quality—and suggest a bidirectional relationship between smoking and depression. Similarly, Cederlöf et al. found that substance use, including smoking, was linked to increased sleep problems in patients with psychotic disorders, which in turn were associated with worsened psychiatric outcomes [28-31]. These overlapping risk factors highlight the need for integrated care strategies addressing both sleep and mental health in smokers.

Smokers were found to have higher MS, indicating increased upper airway obstruction and greater OSA severity. For example, Nuckton et al. reported that each 1-point increase in MS was associated with more than a twofold increase in the odds of having OSA, as well as an increase of over five events per hour in the AHI [32]. Similarly, a study by Liistro et al. reported that a high MS represents a predisposing factor for OSA, especially when associated with nasal obstruction [33]. These findings further support the mechanistic link between smoking and upper airway collapsibility.

While alcohol use was significantly more common among smokers in our study, its relationship with OSA was not independently analyzed and is only briefly noted as a potential confounder. Our gender-based analysis revealed patterns consistent with previous research. Males had significantly higher AHI values and were more prevalent in moderate to severe OSA categories, while females tended to be older and exhibited higher rates of Grade III obesity,

diabetes mellitus, hypothyroidism, and use of sleep medications. Gender differences in OSA may be influenced by hormonal changes, body fat distribution, and healthcare-seeking behavior, with studies showing that declining estrogen and progesterone levels after menopause reduce upper airway stability and contribute to increased OSA risk in women [34-38]. Although women showed lower AHI values, they experienced a higher burden of comorbidities indicating that OSA severity alone may underestimate clinical impact in females. Additionally, higher use of sleep medications among women in our cohort mirrors existing findings that females are more likely to use hypnotics across age groups and socioeconomic strata, potentially due to increased insomnia or differing perceptions of sleep health [39,40].
Our analysis of univariate and multivariate results aligns with established literature identifying male sex and obesity as the strongest independent risk factors for OSA with elevated MS [41-43]. More recent studies support this, with a UK cohort showing that males had over threefold higher odds of OSA (OR 3.27), and individuals with BMI ≥35 kg/m² had nearly fourfold greater risk than those with BMI <30 kg/m² [44]. A meta-analysis involving over 12,000 adults reported that overweight individuals (BMI 25–30) had OR 2.18, and obese individuals (BMI ≥30) had OR 4.84 for OSA [45]. The Health Improvement Network UK study reported a 27-fold increased risk in individuals with BMI ≥40 kg/m², and the Wisconsin Sleep Cohort found a 10% weight gain increased OSA risk nearly six-fold [25,46]. Although smoking couldn't be proven as a risk factor, our findings cement the already established facts that smokers are noted to have significantly higher AHI, greater oxygen desaturation and poorer sleep efficiency. Understanding these relationships is crucial for identifying individuals at higher risk for severe OSA and tailoring appropriate interventions.

Limitations

The retrospective design introduces the potential for missing or incomplete data. Additionally, smoking history was categorized broadly and lacked granularity in terms of pack-years, limiting our ability to assess dose-response relationships. Future prospective studies should aim to collect detailed smoking exposure data and evaluate the impact of smoking cessation on OSA progression and outcomes.

Conclusions

In conclusion, this study highlights the male gender and higher BMI was associated with severe OSA after adjusting for confounders. Smokers demonstrated higher AHI, greater O2

desaturation and poorer sleep efficiency along with higher prevalence of psychiatric disorder. These findings highlight the need for comprehensive management strategies for OSA patients, especially those with a smoking history, to address both the respiratory and systemic consequences of the disease. Further research is needed to explore the underlying mechanisms by which smoking exacerbates OSA severity and to evaluate the long-term benefits of smoking cessation in this population. These findings highlight the need for comprehensive management strategies for OSA patients, especially those with a smoking history, to address both the respiratory and systemic consequences of the disease. Further research is needed to explore the underlying mechanisms by which smoking exacerbates OSA severity and to evaluate the long-term benefits of smoking cessation in this population.

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Table 1. Descriptive statistics for patient characteristics according to OSA severity categories.

Characteristic	Overall (n=567)	NSA (n=102)	Mild (n=116)	Moderate (n=119)	Severe (n=230)	p
Age, Mean ± SD (years)	52.35 ± 12.14	50.86 ± 12.82	52.55 ± 12.55	52.96 ± 12.41	52.59 ± 11.50	0.621
Sex, n (%)						**0.003**
Female	185 (32.63)	42 (41.18)	49 (42.24)	34 (28.57)	60 (26.09)	
Male	382 (67.37)	60 (58.82)	67 (57.76)	85 (71.43)	170 (73.91)	
BMI, Mean ± SD (kg/m^2)	31.10 ± 5.53	29.34 ± 5.46	30.62 ± 5.46	30.99 ± 5.13	32.17 ± 5.59	**<0.001**
Grade, n (%)						**0.002**
Normal	26 (4.59)	9 (8.82)	6 (5.17)	3 (2.52)	8 (3.48)	
Overweight	36 (6.35)	14 (13.73)	7 (6.03)	6 (5.04)	9 (3.91)	
I	209 (36.86)	38 (37.25)	51 (43.97)	45 (37.82)	75 (32.61)	
II	167 (29.45)	25 (24.51)	26 (22.41)	43 (36.13)	73 (31.74)	
III	129 (22.75)	16 (15.69)	26 (22.41)	22 (18.49)	65 (28.26)	
Smoking, n (%)						**0.042**
Non-smoker	455 (80.25)	83 (81.37)	103 (88.79)	93 (78.15)	176 (76.52)	
Ex-smoker	58 (10.23)	12 (11.76)	6 (5.17)	17 (14.29)	23 (10.00)	
Smoker	54 (9.52)	7 (6.86)	7 (6.03)	9 (7.56)	31 (13.48)	
Alcohol, n (%)						0.104
No	443 (78.13)	85 (83.33)	94 (81.03)	94 (78.99)	170 (73.91)	
Ex-alcoholic	12 (2.12)	4 (3.92)	2 (1.72)	4 (3.36)	2 (0.87)	
Occasional	77 (13.58)	11 (10.78)	11 (9.48)	14 (11.76)	41 (17.83)	
Alcoholic	35 (6.17)	2 (1.96)	9 (7.76)	7 (5.88)	17 (7.39)	
Sleeping pills, n (%)	17 (3)	7 (6.86)	5 (4.31)	2 (1.68)	3 (1.30)	**0.031**
DM, n (%)	156 (27.51)	26 (25.49)	30 (25.86)	35 (29.41)	65 (28.26)	0.885
IHD, n (%)	40 (7.05)	8 (7.84)	11 (9.48)	8 (6.72)	13 (5.65)	0.604
Stroke, n (%)	6 (1.06)	0 (0)	0 (0)	3 (2.52)	3 (1.30)	0.18
HTN, n (%)	319 (56.26)	46 (45.10)	73 (62.93)	70 (58.82)	130 (56.52)	0.055
GERD, n (%)	42 (7.41)	9 (8.82)	18 (15.52)	6 (5.04)	9 (3.91)	**<0.001**
Allergic rhinitis, n (%)	56 (9.88)	12 (11.76)	8 (6.90)	16 (13.45)	20 (8.70)	0.304
OAD, n (%)	120 (21.16)	31 (30.39)	29 (25.00)	24 (20.17)	36 (15.65)	**0.015**
Thyroid disorder, n (%)						0.526
No	453 (79.89)	81 (79.41)	87 (75)	98 (82.35)	187 (81.30)	
Hyperthyroidism	2 (0.35)	0 (0)	1 (0.86)	1 (0.84)	0 (0)	
Hypothyroidism	112 (19.75)	21 (20.59)	28 (24.14)	20 (16.81)	43 (18.70)	
RLD, n (%)	5 (0.88)	1 (0.98)	0 (0)	1 (0.84)	3 (1.30)	0.679
Psychiatric, n (%)	5 (0.88)	2 (1.96)	0 (0)	1 (0.84)	2 (0.87)	0.495
Dyslipidemia, n (%)	19 (3.35)	5 (4.90)	2 (1.72)	1 (0.84)	11 (4.78)	0.140
Seizure, n (%)	4 (0.71)	1 (0.98)	0 (0)	0 (0)	3 (1.30)	0.398
MS Score, n (%)						0.054
0	114 (20.21)	26 (25.49)	25 (21.74)	23 (19.49)	40 (17.47)	
1	3 (0.53)	2 (1.96)	0 (0)	0 (0)	1 (0.44)	
2	26 (4.61)	8 (7.84)	5 (4.35)	8 (6.78)	5 (2.18)	
3	218 (38.65)	42 (41.18)	41 (35.65)	46 (38.98)	89 (38.86)	
4	203 (35.99)	24 (23.53)	44 (38.26)	41 (34.75)	94 (41.05)	
Sleep efficiency, Mean ± SD	58.86 ± 20.34	60.85 ± 21.31	59.30 ± 17.66	57.88 ± 19.70	58.24 ± 21.51	0.697
Min.O_2, Mean ± SD	77.29 ± 11.32	86.56 ± 5.98	81.09 ± 9.92	78.66 ± 9.05	70.60 ± 10.85	**<0.001**
Avg.O_2, Mean ± SD	92.04 ± 4.84	94.22 ± 2.76	93.48 ± 4.01	92.58 ± 4.24	90.07 ± 5.47	**<0.001**
Sleep REM, Mean ± SD	23.49 ± 24.46	26.72 ± 24.83	23.01 ± 21.73	22.54 ± 22.63	22.76 ± 26.44	0.552
Min.O_2, Mean ± SD	77.29 ± 11.32	86.56 ± 5.98	81.09 ± 9.92	78.66 ± 9.05	70.60 ± 10.85	**<0.001**

Avg.O_2, Mean ± SD	92.04 ± 4.84	94.22 ± 2.76	93.48 ± 4.01	92.58 ± 4.24	90.07 ± 5.47	**<0.001**
Sleep REM, Mean ± SD	23.49 ± 24.46	26.72 ± 24.83	23.01 ± 21.73	22.54 ± 22.63	22.76 ± 26.44	0.552

°Continuous variables were compared using one-way analysis of variance and categorical were compared using Pearson's chi-square test; Bold p-values indicate statistical significance; NSA: No OSA.

Table 2. Descriptive statistics for patient characteristics according to sex.

Characteristic	Overall (n=567)	Female (n=185)	Male (n=382)	P-value°
Age, Mean ± SD (years)	52.35 ± 12.14	54.53 ± 11.21	51.29 ± 12.45	**0.002**
BMI, Mean ± SD (kg/m^2)	31.10 ± 5.53	32.67 ± 6.38	30.33 ± 4.90	**<0.001**
Grade, n (%)				**<0.001**
Normal	26 (4.59)	9 (4.86)	17 (4.45)	
Overweight	36 (6.35)	12 (6.49)	24 (6.28)	
I	209 (36.86)	49 (26.49)	160 (41.88)	
II	167 (29.45)	50 (27.03)	117 (30.63)	
III	129 (22.75)	65 (35.14)	64 (16.75)	
Smoking, n (%)				**<0.001**
Non-smoker	455 (80.25)	185 (100)	270 (70.68)	
Ex-smoker	58 (10.23)	0 (0)	58 (15.18)	
Smoker	54 (9.52)	0 (0)	54 (14.14)	
Alcohol, n (%)				**<0.001**
No	443 (78.13)	185 (100)	258 (67.54)	
Ex-alcoholic	12 (2.12)	0 (0)	12 (3.14)	
Occasional	77 (13.58)	0 (0)	77 (20.16)	
Alcoholic	35 (6.17)	0 (0)	35 (9.16)	
Sleeping pills, n (%)	17 (3)	12 (6.49)	5 (1.31)	**0.002**
DM, n (%)	156 (27.51)	63 (34.05)	93 (24.35)	**0.020**
IHD, n (%)	40 (7.05)	14 (7.57)	26 (6.81)	0.875
Stroke, n (%)	6 (1.06)	1 (0.54)	5 (1.31)	0.689
HTN, n (%)	319 (56.26)	115 (62.16)	204 (53.40)	0.060
GERD, n (%)	42 (7.41)	19 (10.27)	23 (6.02)	0.101
Allergic rhinitis, n (%)	56 (9.88)	12 (6.49)	44 (11.52)	0.083
OAD, n (%)	120 (21.16)	52 (28.11)	68 (17.80)	**0.007**
Thyroid disorder, n (%)				**<0.001**
No	453 (79.89)	121 (65.41)	332 (86.91)	
Hyperthyroidism	2 (0.35)	2 (1.08)	0 (0)	
Hypothyroidism	112 (19.75)	62 (33.51)	50 (13.09)	
RLD, n (%)	5 (0.88)	2 (1.08)	3 (0.79)	0.999
Psychiatric, n (%)	5 (0.88)	2 (1.08)	3 (0.79)	0.999
Dyslipidemia, n (%)	19 (3.35)	3 (1.62)	16 (4.19)	0.179
Seizure, n (%)	4 (0.71)	2 (1.08)	2 (0.52)	0.835
MS Score, n (%)				0.495
0	114 (20.21)	39 (21.20)	75 (19.74)	
1	3 (0.53)	0 (0)	3 (0.79)	
2	26 (4.61)	6 (3.26)	20 (5.26)	
3	218 (38.65)	76 (41.30)	142 (37.37)	
4	203 (35.99)	63 (34.24)	140 (36.84)	
AHI, Mean ± SD	30.79 ± 27.16	23.70 ± 22.50	34.23 ± 28.55	**<0.001**
Sleep efficiency, Mean ± SD	58.86 ± 20.34	62.36 ± 19.59	57.14 ± 20.51	**0.004**
Min.O_2, Mean ± SD	77.29 ± 11.32	76.13 ± 12.55	77.85 ± 10.65	0.110
Avg.O_2, Mean ± SD	92.04 ± 4.84	91.24 ± 6.35	92.43 ± 3.86	**0.021**
Sleep REM, Mean ± SD	23.49 ± 24.46	22.78 ± 22.99	23.84 ± 25.17	0.626

°Continuous variables were compared using t-test for independent samples and categorical were compared using Pearson's chi-square test.

Table 3. Descriptive statistics for patient characteristics according to smoking habit.

Characteristic	Overall (n=567)	Non-smoker (n= 455)	Ex-smoker (n=58)	Smoker (n=54)	Smoker + ex-smoker (n=112)	p°	p#
Age, Mean ± SD (years)	52.35 ± 12.14	52.54 ± 12.04	55.22 ± 12.09	47.69 ± 11.97	51.59 ± 12.56	**0.004**	0.472
Sex, n (%)						**<0.001**	**<0.001**
Female	185.00 (32.63)	185.00 (40.66)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)		
Male	382.00 (67.37)	270.00 (59.34)	58.00 (100.00)	54.00 (100.00)	112.00 (100.00)		
BMI, Mean ± SD (kg/m^2)	31.10 ± 5.53	31.23 ± 5.73	30.25 ± 4.17	30.87 ± 5.12	30.55 ± 4.64	0.276	0.187
Grade, n (%)						0.524	0.336
Normal	26.00 (4.59)	24.00 (5.27)	2.00 (3.45)	0.00 (0.00)	2.00 (1.79)		
Overweight	36.00 (6.35)	27.00 (5.93)	3.00 (5.17)	6.00 (11.11)	9.00 (8.04)		
I	209.00 (36.86)	165.00 (36.26)	24.00 (41.38)	20.00 (37.04)	44.00 (39.29)		
II	167.00 (29.45)	131.00 (28.79)	19.00 (32.76)	17.00 (31.48)	36.00 (32.14)		
III	129.00 (22.75)	108.00 (23.74)	10.00 (17.24)	11.00 (20.37)	21.00 (18.75)		
Alcohol, n (%)						**<0.001**	**<0.001**
No	443.00 (78.13)	397.00 (87.25)	28.00 (48.28)	18.00 (33.33)	46.00 (41.07)		
Ex-alcoholic	12.00 (2.12)	4.00 (0.88)	6.00 (10.34)	2.00 (3.70)	8.00 (7.14)		
Occasional	77.00 (13.58)	39.00 (8.57)	15.00 (25.86)	23.00 (42.59)	38.00 (33.93)		
Alcoholic	35.00 (6.17)	15.00 (3.30)	9.00 (15.52)	11.00 (20.37)	20.00 (17.86)		
Sleeping pills, n (%)	17.00 (3.00)	16.00 (3.52)	0.00 (0.00)	1.00 (1.85)	1.00 (0.89)	0.293	0.250
DM, n (%)	156.00 (27.51)	123.00 (27.03)	15.00 (25.86)	18.00 (33.33)	33.00 (29.46)	0.592	0.691
IHD, n (%)	40.00 (7.05)	34.00 (7.47)	4.00 (6.90)	2.00 (3.70)	6.00 (5.36)	0.592	0.564
Stroke, n (%)	6.00 (1.06)	4.00 (0.88)	1.00 (1.72)	1.00 (1.85)	2.00 (1.79)	0.701	0.746
HTN, n (%)	319.00 (56.26)	249.00 (54.73)	41.00 (70.69)	29.00 (53.70)	70.00 (62.50)	0.064	0.168
GERD, n (%)	42.00 (7.41)	35.00 (7.69)	4.00 (6.90)	3.00 (5.56)	7.00 (6.25)	0.841	0.748
Allergic rhinitis, n (%)	56.00 (9.88)	48.00 (10.55)	3.00 (5.17)	5.00 (9.26)	8.00 (7.14)	0.428	0.365
OAD, n (%)	120.00 (21.16)	105.00 (23.08)	8.00 (13.79)	7.00 (12.96)	15.00 (13.39)	0.08	**0.034**
Thyroid disorder, n (%)						0.161	**0.040**
No	453.00 (79.89)	354.00 (77.80)	52.00 (89.66)	47.00 (87.04)	99.00 (88.39)		
Hyperthyroidism	2.00 (0.35)	2.00 (0.44)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)		
Hypothyroidism	112.00 (19.75)	99.00 (21.76)	6.00 (10.34)	7.00 (12.96)	13.00 (11.61)		
RLD, n (%)	5.00 (0.88)	3.00 (0.66)	2.00 (3.45)	0.00 (0.00)	2.00 (1.79)	0.078	0.563
Psychiatric, n (%)	5.00 (0.88)	2.00 (0.44)	1.00 (1.72)	2.00 (3.70)	3.00 (2.68)	**0.041**	0.088
Dyslipidemia, n (%)	19.00 (3.35)	17.00 (3.74)	1.00 (1.72)	1.00 (1.85)	2.00 (1.79)	0.589	0.463
Seizure, n (%)	4.00 (0.71)	3.00 (0.66)	1.00 (1.72)	0.00 (0.00)	1.00 (0.89)	0.533	0.999
MS Score, n (%)						**0.048**	**0.048**
0	114.00 (20.21)	98.00 (21.68)	11.00 (18.97)	5.00 (9.26)	16.00 (14.29)		
1	3.00 (0.53)	3.00 (0.66)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)		
2	26.00 (4.61)	23.00 (5.09)	3.00 (5.17)	0.00 (0.00)	3.00 (2.68)		
3	218.00 (38.65)	178.00 (39.38)	22.00 (37.93)	18.00 (33.33)	40.00 (35.71)		
4	203.00 (35.99)	150.00 (33.19)	22.00 (37.93)	31.00 (57.41)	53.00 (47.32)		
AHI, Mean ± SD	30.79 ± 27.16	29.43 ± 26.58	32.37 ± 27.56	40.56 ± 29.85	36.32 ± 28.86	**0.033**	**0.023**
Sleep efficiency, Mean ± SD	58.86 ± 20.34	60.09 ± 19.82	54.55 ± 22.86	52.94 ± 20.62	53.77 ± 21.73	**0.021**	**0.006**
Min.O_2, Mean ± SD	77.29 ± 11.32	77.12 ± 11.50	79.07 ± 10.83	76.80 ± 10.31	77.97 ± 10.59	0.416	0.456
Avg.O_2, Mean ± SD	92.04 ± 4.84	91.92 ± 5.13	92.53 ± 3.42	92.53 ± 3.37	92.53 ± 3.38	0.324	0.130
Sleep REM, Mean ± SD	23.49 ± 24.46	23.19 ± 23.93	27.82 ± 27.89	21.49 ± 25.09	24.71 ± 26.62	0.429	0.588

°Comparison between non-smoker, ex-smoker and smoker categories using one-way analysis of variance. #Comparison between non-smoker and smokers + ex-smokers together using t-test for independent samples; Bold p-values indicate statistical significance.

Table 4. Risk factors of severe OSA – univariate and multivariate analysis.

Characteristic	Univariate		Multivariate	
	OR (95% CI)	p	Adj. OR (95% CI)	p
Age in years	1.01 (0.99 to 1.02)	0.362		
Sex				
Female	—			
Male	1.94 (1.36 to 2.78)	**<0.001**	1.79 (1.18 to 2.71)	**0.006**
BMI Grade				
Normal	—			
I	1.84 (0.81 to 4.29)	0.148	1.47 (0.62 to 3.47)	0.382
II	3.10 (1.34 to 7.38)	**0.009**	2.68 (1.11 to 6.52)	**0.029**
III	2.82 (1.20 to 6.83)	**0.018**	2.71 (1.09 to 6.72)	**0.032**
Overweight	0.97 (0.35 to 2.74)	0.960	0.77 (0.25 to 2.18)	0.580
Smoking°				
Ex-smoker	1.54 (0.87 to 2.82)	0.151	1.14 (0.61 to 2.15)	0.680
Smoker	1.98 (1.07 to 3.86)	**0.036**	1.48 (0.74 to 2.94)	0.266
Alcohol°				
Ex-alcoholic	0.68 (0.21 to 2.20)	0.507		
Occasional	1.70 (1.01 to 2.93)	0.051		
Alcoholic	1.48 (0.72 to 3.21)	0.299		
Sleeping pills (Yes)°	0.25 (0.08 to 0.68)	**0.006**	0.41 (0.13 to 1.29)	0.128
T2DM (Yes) °	1.16 (0.79 to 1.71)	0.441		
IHD (Yes) °	0.67 (0.35 to 1.29)	0.227		
HTN (Yes) °	1.12 (0.79 to 1.57)	0.526		
GERD (Yes) °	0.32 (0.16 to 0.60)	**<0.001**	0.39 (0.19 to 0.79)	**0.009**
Allergic rhinitis (Yes) °	1.14 (0.65 to 2.06)	0.656		
OAD (Yes) °	0.55 (0.36 to 0.82)	**0.004**	0.62 (0.39 to 0.95)	**0.030**
Thyroid disorder (Yes) °	0.76 (0.49, 1.14)	0.185		
RLD (Yes) °	2.52 (0.37 to 49.4)	0.372		
Psychiatric (Yes) °	0.94 (0.15 to 7.15)	0.943		
Dyslipidemia (Yes) °	1.07 (0.42 to 2.92)	0.883		
Seizure (Yes) °	1.88 (0.24 to 38.2)	0.567		
MS Score	1.13 (1.01 to 1.27)	**0.034**	1.06 (0.94 to 1.19)	0.372
Sleep efficiency	1.00 (0.99 to 1.00)	0.276		
Sleep REM	1.00 (0.99 to 1.00)	0.334		

°Reference: No; The variables significant in univariate analysis were included in the multivariate analysis to obtained adjusted odds of OSA associated with each variable.