

Clinical and prognostic characteristics of obstructive sleep apnea among smokers

Les caractéristiques cliniques et pronostiques du SAOS chez les tabagiques

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ABSTRACT

Introduction: Smoking is a major public challenge, with profound implications for individual and societal health, intertwining with many medical issues such as the on growing of obstructive sleep apnea (OSA) incidence with studies highlighting a potential association between these two entities, suggesting that smoking may contribute to the pathophysiology of OSA and the worsening of the severity degree of OSA symptoms.

Aim: This study seeks to determine the clinical and prognostic characteristics of OSA among smokers and secondly to look for factors predictive of OSA severity.

Methods: A cross-sectional study carried out in the Pneumology Department at the Charles Nicolle University Hospital in Tunis from January 2023 to June 2024, enrolling 172 patients with a confirmed OSA by a respiratory polygraphy realized in hospital. We divided patients into two groups according to smoking status. G1: smoking patients and G2: non-smoking patients. Statistical analyses were performed to compare the results of the two groups.

Results: A total of 172 patients were included. Forty-seven patients (27.3%) were smokers (G1) with a sex ratio at 3 and a mean age at 59 ± 13 years. Univariate study showed that smoking patients were predominantly men ($p < 0.001$). There was no significant relation between smoking status and cardiovascular and metabolic comorbidities except for COPD that was prevalent among G1 ($p = 0.001$) and insomnia ($p = 0.04$). Screening scores such NoSAS and StopBang were more performant in G1 ($p = 0.03$ and $p = 0.01$ respectively). Polygraphy finding showed that smoking patients had more severe OSA with higher AHI ($p = 0.007$), AI ($p = 0.01$), ODI ($p = 0.006$) and higher capnia ($p = 0.04$) and as a result more frequent recourse to CPAP use ($p = 0.036$). Multi variate study suggested that male sex and hypercapnia characterize the presentation of OSAS in patients who smoke with respectively ($OR = 1.85$; $p < 0.05$) and ($OR = 5.57$; $p = 0.015$). We also carried on a multivariate study to look for predictive factors of OSA severity, and we found that smoking status was not significantly associated with OSA severity ($p = 0.9$).

Conclusion: This study clearly shows that OSA appears to be more severe among smokers. It emphasizes the performance of screening scores and proposes male sex and hypercapnia as two factors that suggest the severity of this sleep disorder. Thus, targeted interventions for smokers at risk of OSA and encouraging smoking cessation are needed.

Keywords: Obstructive sleep apnea, Smoking, Prognosis, Severity

RÉSUMÉ

Introduction : Le tabagisme représente un défi de santé publique majeur, impactant la physiopathologie du syndrome d'apnées obstructives du sommeil (SAOS) et aggravant potentiellement ses symptômes. **Objectif :** Cette étude visait à déterminer les caractéristiques cliniques et pronostiques du SAOS chez les fumeurs et à identifier les facteurs prédictifs de sa sévérité.

Méthodes : Une étude transversale a été menée au service de Pneumologie du CHU Charles Nicolle de Tunis (janvier 2023 - juin 2024), incluant 172 patients diagnostiqués par polygraphie ventilatoire. Deux groupes ont été comparés : les fumeurs (G1) et les non-fumeurs (G2).

Résultats : 47 patients (27,3%) étaient fumeurs, avec une prédominance masculine ($p < 0,001$) et un âge moyen de 59 ans. L'étude univariée a montré une prévalence significativement plus élevée de BPCO ($p = 0,001$) et d'insomnie ($p = 0,04$) dans le G1. Les scores NoSAS et StopBang étaient plus performants chez les fumeurs. Sur le plan polygraphique, les fumeurs présentaient un SAOS plus sévère avec un index d'apnées-hypopnées ($p = 0,007$), un index d'apnées ($p = 0,01$), et un index de désaturation en oxygène (IDO) plus élevés ($p = 0,006$), ainsi qu'une hypercapnie plus marquée ($p = 0,04$), menant à un recours plus fréquent à la ventilation au long cours ($p = 0,036$). L'analyse multivariée a identifié le sexe masculin ($OR = 1,85$) et l'hypercapnie ($OR = 5,57$) comme caractéristiques majeures du SAOS chez le fumeur, bien que le statut tabagique seul ne soit pas apparu comme un facteur prédictif indépendant de sévérité ($p = 0,9$).

Conclusion : Le SAOS semble plus sévère chez les fumeurs. Cette étude souligne l'importance des scores de dépistage et de l'aide au sevrage tabagique dans la prise en charge de ces patients.

Mots clés: Apnée obstructive du sommeil, Tabagisme, Pronostic, Gravité

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INTRODUCTION

Smoking remains a significant global health crisis, impacting millions of lives and economies worldwide. Its prevalence is on a continuous surge posing a great challenge as it was up to 32.6% and 6.5% among men and women, respectively in 2020 with 1.18 billion people regularly smoke tobacco, causing to more than 7 million deaths, making it the leading cause of preventable mortality (1).

Obstructive Sleep Apnea (OSA) is a prevalent sleep disorder and according to a systematic review by Benjafield et al., nearly 1 billion people are affected by OSA worldwide, with rates exceeding 50% in certain countries (2) and ~16% among young adults according to a systematic review published in 2024 (3). On the national level and according to study conducted in the Central Eastern of Tunisia and published in 2024, the crude incidence rate of OSA cases was 12.3/100000 inhabitants/year (4).

Obstructive Sleep Apnea (OSA) is characterized by repeated interruptions in breathing during sleep which can be intermittent (apnea) or significant reduction (hypopnea) contributing to fragmented sleep and excessive daytime sleepiness.

Clinical manifestations include a wide range of symptoms such as: excessive daytime sleepiness (5)loud snoring, choking sensations during sleep and as it progresses, it may also lead to cognitive impairments (6).

OSA may cause physiological disturbances and various clinical impacts leading to significant health consequences, including alteration in the quality of life, metabolic and cardiovascular diseases(7,8).

The onset of OSA involves a complex interaction of multiple factors like loss of muscle tone during sleep (9),obesity, male gender and advanced age(10).

Recent studies have also highlighted a potential association between smoking and OSA, suggesting that smoking may contribute to its pathophysiology and worsen the degree of severity of OSA symptoms. Yet, the relationship between these two entities remains complex and somewhat controversial as some findings suggest that the risk of developing OSA escalates with the duration and intensity of smoking habits while some other studies have failed to establish a definitive link between smoking and OSA after adjusting for confounding factors such as age, sex, and body mass index (BMI).

Given the rising prevalence of both smoking and OSA and the need to understand more this relationship, we conducted this study with a focus to investigate the clinical and prognostic characteristics of OSA in smoking patients by comparing them to non-smoking OSA patients, and in a second position to determine whether smoking is linked to the severity of this sleep disorder.

METHODS

Design and study population

We conducted a cross-sectional study carried out in the

Pneumology Department at the Charles Nicolle University Hospital in Tunis from January 2023 to June 2024.

The study enrolled 172 patients with a confirmed OSA by a respiratory polygraphy realized in hospital, referred by primary care physicians or pre-anesthesia consultation to our sleep unit.

We included:

- Patients over 18 years of age
- Patients with confirmed sleep apnoea syndrome, i.e. an $AHI \geq 5/h$ on ventilatory polygraphy
- Patients with obstructive sleep apnoea hypopnoea syndrome.

We excluded:

- Non-consenting patients
- Patients with incomplete medical records

We divided patients into two groups according to smoking status. G1: smoking patients (active smokers and ex-smoking patients) and G2: non-smoking patients. Data relating to each patient were collected on a detailed pre-established information sheet after systematic consultation. Socio-demographic data, personal history, family history of obesity and OSAS, anthropometric parameters, general signs of OSAS were collected. Questionnaires such stopBANG, NoSAS, Epworth sleepiness scale (ESS) and Pichot fatigue scale were used. We compared the clinical, paraclinical and polygraphy data of smokers and non-smokers patients.

Statistical analysis

Results were expressed as mean \pm standard deviation (SD) for continuous variables and as frequency (percentage) for categorical variables. Student's T test and ANOVA test were used for comparison of means, according to variables distribution. For comparison of percentages, we used chi-square test, or two-tailed Fisher test when necessary. In all statistical tests, the significance level was set at 0.05. A binary logistic regression analysis was used to investigate the association between smoking and OSA by including variables with a p-value less than 0.2.

RESULTS

Baseline Characteristics

From January 2023 to June 2024, we included 172 patients where the diagnosis of OSA was confirmed. We divided the patients in two groups, G1: 47 patients (27.3%) and G2: 125 patients (72.7%). The mean age was 59 ± 13 years, ranging from 19 to 85 years and most of the patients were females (n=137; 79.7%) versus 35 males (20.3%) with a sex ratio of 0.3.

Smoking habit

Forty-seven of the patients (27.3%) were smokers defining those in group G1.

The prevalent smoking type was conventional smoking in 66% with an average smoking of 7 year-pack, within whom only 14 patients (8.2 %) have quit smoking.

Followed by use of water pipe in 23.4% of G1 patients and 'NAFFA' in 10.6% of patients.

Clinical Characteristics

Eighty-four-point nine percent of the patients had comorbidities: respiratory diseases such asthma was encountered in 24 patients (24%) and chronic obstructive pulmonary disease (COPD) in 4 patients (2.3%). Cardiovascular and metabolic disorders were predominant: 82% of OSA patients were obese, with a mean Body Mass Index (BMI) of $36.6 \pm 6.9 \text{ Kg/m}^2$. High blood pressure was found in 107 patients (62.2%), diabetes in 78 patients (45.3%), dysthyroidism in 17 patients (9.9%). Psychiatric disorders were encountered among only 4% of the patients.

The prevalent symptom was snoring in 169 patients (98.3%), followed by excessive daytime sleepiness in 112 patients (65.1%) with a mean Epworth scale of 8 ± 4.9 .

Pichot fatigue score used to evaluate tiredness and assess the extent of its impact on daily life was 13.8 ± 8.8 .

Insomnia was seen in 96 of the patients (55.8%) and the sensation of being paralyzed among 31 patients. Nocturia was present in 70.3% of the patients.

The mean NoSAS and STOP-Bang scores that help identify people at risk of sleep apnea syndrome were respectively 10.6 ± 2.9 and 3.7 ± 1.2 .

Polygraphy Characteristics

The apnea-hypopnea index (AHI), a measure of sleep apnea severity allowed us to classify the OSA into Mild OSA in 63 patients (36.6%), moderate OSA in 45 patients (26.2%) and severe OSA ($\text{IAH} \geq 30/\text{h}$) in 64 patients (37.2%). The mean hypopnea index (HI) was $15.5/\text{h} \pm 10.54$, the mean apnea index (AI) was $8.2/\text{h} \pm 12.83$. The mean Oxygen desaturation index (ODI) was 24.5 ± 18 . Sixty-six patients (38.4%) required recourse to continuous positive airway pressure (CPAP).

Table 1 summarizes clinical and polygraphy characteristic of the study population.

Comparative Analysis of Clinical and polygraphy data

Clinical data

Smokers diagnosed with OSA were mainly men with 32 male patients in G1(68.1%) vs. 3 men in G2(2.4%) with a significant difference ($p < 0.001$).

However, age didn't differ between groups (G1: 56 years vs G2:57 years; $p > 0.05$).

Comorbidities such as asthma, hypertension, diabetes, obesity, dysthyroidism and psychiatric disorders didn't differ between groups (p value > 0.05), except for the chronic obstructive pulmonary disease (COPD) which affect 8.5% of G1 patients ($p = 0.001$).

For the symptoms: there was a statistically significant association with insomnia (G1:68.1% vs. 51.2%, $p = 0.04$). There was no significant association between smoking status and the importance of excessive daytime sleepiness with an Epworth scale above 10 ($p = 0.4$), same for tiredness evaluated by Pichot fatigue scale with

a mean of 14 ± 8 in G1 vs. 13 ± 9 in G2 ($p = 0.6$), snoring ($p = 0.8$), nocturia($p = 0.7$), apnea ($p = 0.8$) and the sensation of paralysis($p = 0.4$).

Detailed comparison in table 2

Table 1. Clinical and polygraphy characteristic of the study population

characteristics	
Age (years)(mean \pm SD)	59 \pm 13
Smoking n(%)	47(27.3)
Male n(%)	35(20.3)
Female n(%)	137(79.7)
Observed apnea n(%)	49(28.5)
Sensation of paralysis n(%)	31(18)
Snoring n(%)	169(98.3)
Excessive daytime sleepiness n(%)	112(65.1)
Insomnia n(%)	96(55.8)
Nocturia n(%)	121(70.3)
Tiredness: Pichot score (mean \pm SD)	13.81 \pm 8.8
Epworth (mean \pm SD)	8 \pm 4.9
NoSAS(mean \pm SD)	10.6 \pm 2.9
STOP-Bang (mean \pm SD)	3.7 \pm 1.2
BMI (Kg/m^2) (mean \pm SD)	36.6 \pm 6.9
Neck circumference (cm)(mean \pm SD)	35.91 \pm 5
waist circumference(cm) (mean \pm SD)	127 \pm 11
OSA severity n(%)	63(36.6)
● Mild	45(26.2)
● Moderate	64(37.2)
● severe	

Table 2. Comparative Analysis of Clinical data in both groups

characteristics	G1	G2	P value
Age (years)	56 \pm 12.8	57 \pm 13	>0.05
Male n(%)	32 (68.1)	3 (2.4)	<0.001
Female n(%)	15 (31.9)	122 (97.6)	
Observed apnea n(%)	14(29.8)	35(70.2)	0.8
BMI (Kg/m^2) (mean \pm SD)	36 \pm 6	37.4 \pm 7	0.2
Neck circumference (cm)(mean \pm SD)	37.6 \pm 5	35 \pm 4.7	0.4
waist circumference(cm) (mean \pm SD)	168.2 \pm 8.9	158.5 \pm 6	0.1
Tiredness: Pichot score (mean \pm SD)	14 \pm 8	13 \pm 9	0.6
Epworth (mean \pm SD)	9 \pm 5	7 \pm 5	0.1

OSA severity

For the screening score, NoSAS score was higher than 8 in G1 patients than G2 patients (89.4% vs. 75.8%; $p = 0.03$), as well as the STOPBANG score which was higher among G1 patients (4 vs 3; $p = 0.01$).

Fifty-one-point-one percent of smokers had severe OSA vs 34.7% in G2 with a significant difference ($p=0.05$). Hypercapnia ($\text{PaCO}_2 \geq 45 \text{ mmHg}$) in arterial blood gas was more frequent in G1 patients ($p=0.04$). Smokers had a higher AHI (G1: 31/h vs. G2: 23/h; $p=0.007$) with a higher AI (G1:12/h vs. G2:6/h; $p=0.01$) and a higher ODI (G1: 31/h vs G2: 22/h; $p=0.006$). Continuous Positive Airway Pressure (CPAP) was more indicated among smokers (G1:51.1% vs. G2:33.6%; $p=0.036$). Detailed comparison in terms of polygraphy results in table 3

Table 3. Comparative Analysis of OSA features in both groups

	G1 n(%)	G2 n(%)	p
Severe OSA	24 (51.1)	43 (34.7)	0.05
NoSAS ≥ 8	42 (89.4)	94 (75.8)	0.03
StopBANG(mean)	4 \pm 1.5	3 \pm 1	0.01
AHI (mean/h)	31/h \pm 20.6	23/h \pm 17.23	0.007
AI (mean/h)	12/h \pm 17.3	6/h \pm 10.5	0.01
HI (mean/h)	17/h \pm 10.6	15/h \pm 10.5	0.3
ODI (mean/h)	31/h \pm 21.6	22/h \pm 16.5	0.006
hypercapnia	7 (14.9)	5.6 (5.6)	0.04
CPAP	24 (51.1)	42(33.6)	0.036

Prognostic Factors Associated with OSA among smokers and severe OSA:

A binary logistic regression was carried out, incorporating variables with a p-value less than 0.2 from the initial analysis, enabling us to consider the confounding factors and help us identify independent associations. The findings from the multivariate analysis suggest that male sex and hypercapnia characterize the presentation of OSA in patients who smoke with respectively ($OR=1.85$; $p<0.05$) with confidence interval (CI) [1.2-3.3] and ($OR=5.57$; $p=0.015$); CI [1.4-22].

We found also that even if severe OSA was more frequent in G1; after adjusting for confounding variables such BMI, gender and age, smoking was not significantly associated with the severity of this sleep disorder with a p value of 0.9.

DISCUSSION

The present study was conducted to elucidate the clinical and prognostic characteristics of obstructive sleep apnea (OSA) among smokers, revealing significant associations between these two entities, suggesting that smoking is a critical risk factor for the development of OSA.

Referring to the available literature, our cohort predominantly consisted of females (79.7%), which contrasts with several studies that report a higher prevalence of OSA among males(11,12).

The mean age was 59 years which is consistent with what was found by Bielicki et al., 2019 where the mean age was 57.3 ± 9.5 years(13). There was no significant association between smoking status and OSA after adjusting age

which was concordant with the results of Hsu et al.(14). Smoking was more common among men ($p<0.001$) (14,15). The male gender proved to be according to our study a good predictor of OSA among smokers with an $OR=1.85$, CI = [1.2-3.3] indicating that male smokers are twice more likely to develop OSA compared to non-smoking males which was in agreement with what was reported by Jang et al.(16).

The smoking prevalence in our study showed that 23.3% were conventional cigarette smokers, which was consistent with findings from literature where conventional smoking is still predominant even after the emergences of new types of smoking(17,18).

Our results indicated an important prevalence of comorbidities among patients with OSA, particularly cardiovascular and metabolic disorders such as obesity (82%) and hypertension (62.2%) without an increased incidence among smokers ($p>0.05$) which was confirmed by Bielicki et al.(13). Whereas, smoking was associated with COPD in our study as well as other studies(19,20). The prevalent symptoms in our study included snoring (98.3%) and excessive daytime sleepiness (65.1%) measured by the Epworth Sleepiness Scale (ESS) that underscores the deep impact of OSA on daily life activities didn't exhibit any significant correlation with smoking status. This finding was concordant with what was reported in Ioannidou et al.(15) and Hsu et al(14). Meanwhile, other studies have reported significant associations between higher ESS scores and smoking status, suggesting that this relationship may vary based on population characteristics or study design.

We observed a higher NoSAS score among smokers (89.4% vs. 75.8%, $p=0.03$) and higher STOP-BANG scores (4 vs. 3, $p=0.01$). These results align with findings from other studies indicating that screening tools like STOP-Bang and NoSAS are reliable in the detection of OSA (16). Our study showed a higher prevalence and severity of OSA among smokers compared to non-smokers which was consistent with other studies. For instance, a meta-analysis by Zeng et al., 2023 (21) found that heavy smokers exhibited significantly elevated apnea-hypopnea index (AHI) levels, corroborating our results where smokers had a higher mean AHI (31/h vs. 23/h in non-smokers) with a statistically significant difference ($p=0.007$). Same result was met in the study of Bielicki et al.(13) where the mean AHI was 31/h (18.4–53.29) among smokers vs 29 (18.3–47.7) in non-smokers with a $p = 0.03$, and the study of Hsu et al.(14) where smokers had higher AHI values for total sleep time (19.37 ± 22.43 vs. 12.99 ± 18.03 ; $p<0.001$) or non-rapid eye movement sleep (18.56 ± 23.24 11.89 ± 18.53 ; $p<0.001$).

Our analysis also revealed a significant association between smoking status and nocturnal oxygen desaturation index (ODI), with smokers exhibiting higher ODI values (31/h vs. 22/h, $p=0.006$). The mean ODI was 24.5%, which is indicative of significant nocturnal hypoxemia often seen in patients with severe OSA, taking as a reference, a study conducted by Varghese et al., 2022 where it has been highlighted that an $ODI > 20$ has a sensitivity of 96.6% and specificity of 69.6% in diagnosing severe OSA (22).

This finding aligns with the study conducted by Lee et al.(23) where Smokers had a high apnea-hypopnea index (AHI) and oxygen desaturation index (ODI) ($p<0.001$) and where logistic regression analysis after adjustment of confounding factors showed that AHI was independent of smoking aggregating with what we found.

Moreover, our data indicated a tendency toward hypercapnia in smokers compared to non-smokers ($p<0.05$) and it proved to be a good predictor of OSA ($p=0.015$, OR=5.57), which is consistent with the fact that smoking can affect the respiratory system and sleep architecture due to the nicotine's effect thus an impaired ventilation(24). Smoking can lead too to intermittent hypoxic episodes during sleep(21), with the body's response including an increase in sympathetic nervous system activity(25,26) and a subsequent rise in CO₂ levels due to chronic intermittent decreased ventilation during sleep (27) .

It should be mentioned that as smoking patients had greater AHI, AI and ODI ,it wasn't found as a predicting factor of OSA nor of its severity after conducting the multivariate analysis which was consistent with what was concluded by Ioannidou et al.(15).

In terms of treatment, it is commonly known that CPAP is the gold standard of OSA therapy(28). We found that 51.1% of smokers required continuous positive airway pressure (CPAP) therapy compared to 33.6% in non-smokers ($p=0.036$), suggesting that smokers may have more severe manifestations of OSA needing more specific intervention strategies. Previous research supports this notion, for instance, a study noted that continued use of CPAP therapy was associated with the severity of AHI and to the more severe symptoms common in smokers(29). At the end of this discussion, it should also be mentioned that studies establishing a positive relationship with other lifestyle habits particularly alcohol rather than tobacco use exist and we take the example of a recent systematic review and meta-analysis of 14 identified studies that found that the risk of OSA was positively associated with alcohol consumption rather than smoking(30).

CONCLUSION

The study suggests a significant association between smoking and the severity of Obstructive Sleep Apnea, indicating that smokers are more likely to experience worse manifestations of the condition and that smoking is a critical risk factor for developing OSA and highlight the need for targeted interventions aimed at smoking cessation as part of comprehensive management strategies for individuals with OSA: by enforcing regulations on tobacco advertising and sales, by facilitating the access to smoking cessation resources or by routine screening for OSA among smokers for an early diagnosis and better management.

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