Prévalence de la dépression et de l'anxiété dans l'apnée obstructive du sommeil

Prevalence of depression and anxiety in obstructive sleep apnea

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Abstract

Introduction: Organic comorbidities of obstructive sleep apnea (OSA) have been widely studied. However, psychiatric disorders, especially depression and anxiety, have not attracted so much attention.

Aim: The primary aim was to determine the prevalence and the predictive factors of depression and anxiety in OSA patients. The secondary aim was to investigate the association between OSA severity and these psychiatric disorders.

Methods: A cross-sectional study including untreated OSA patients without mental illness history was conducted. Patients were administered the Hospital Anxiety (HADS-A) and Depression Scale (HADS-D). Depression and anxiety were diagnosed for HAD-D and HAD-A scores ≥ 8.

Results: Eighty patients were included (mean age: 54.83 ± 13.12 yr; female: 52 (65%); mean Body mass index (BMI) :34.7±6.14 kg/m2). The prevalence of depression and anxiety was 35 % and 43.8% of patients respectively. Both depressive and anxious OSA patients had more libido disorder (p=0.011, p=0.0007 ;respectively), anhedonia (p= 10-4, p= 10-4respectively) and suicidal ideas(p= 0.002 ,p=0.019 respectively). Moreover, depressed OSA patients had lower socio-economic condition (p= 0.019), more coronary artery diseases (CAD) (p=0.019) and less cognitive disorder (p= 0.005). The HADS-D (r=0,095; p=0,404) and the HADS-A (r=0,212; p=0,059) were not correlated with the Apnea/Hyponea Index. The determinants of depressive and anxious mood were female-sex (p= 0.035, p=0.004 respectively) and libido disorder (p=0.040, p=0.02 respectively). Anhedonia (p=10-4) and CAD (p=0.010) were also identified as a predictive factors of depression.

Conclusions: In our study, the high prevalence of depression and axiety in apneic patients demonstrates the importance of the psychiatric component in the management of this disease. A collaboration between pneumologists and psychiatrists is necessary in order to improve the quality of life of these patients.

Keywords : Obstructive sleep apnea, Depression, Anxiety, Sleep monitoring, HAD Scale

Résumé

Introduction : L'échoguidage en temps réel de la veine jugulaire interne est recommandé par les sociétés savantes. Cependant, peu d'études ont évalué l'apport de l'échoguidage pour le cathétérisme de la veine sous-clavière (VSC).

Objectif : Comparer le cathétérisme de la VSC par échoguidage en temps réel par apport au repérage anatomique externe.

Méthodes : Il s'agit d'une étude prospective randomisée. Les patients âgés ≥ 18 ans proposés pour cathétérisme veineux central en dehors d'un contexte d'urgence ont été inclus. Les critères de non-inclusion étaient la thrombose de la VSC ou une coagulopathie sévère. Toutes les procédures ont été effectuées par deux résidents. Les patients ont été randomisés en deux groupes : groupe échoguidage (GE) et un groupe cathétérisme par voie classique (GC). Le critère de jugement principal est le taux de succès global. Les critères de jugement secondaires étaient le taux de succès dès la première ponction et le taux de complications.

Résultats : Soixante-dix patients ont été inclus (35 dans chaque groupe). Le taux de succès global était plus élevé dans le GE par apport au GC mais statistiquement non significatif (100% vs 85,7% respectivement ; p=0,054). L'échoguidage en temps réel a permis d'augmenter significativement le taux de succès dès la première ponction (GE : 82,9% vs GC : 40% ; p<10-3) et de diminuer significativement l'incidence globale des complications mécaniques (GE : 5,7% vs GC : 37,1% ; p=0,001).

Conclusion : Selon notre étude, l'échoguidage en temps réel pour le cathétérisme de la VSC semble être une alternative intéressante par apport au repérage anatomique externe.

Les mots clés : cathétérisme veineux central, échographie, veine sous-clavière, unité de soins intensifs.

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INTRODUCTION

Obstructive sleep apnea (OSA) is the most common form of sleep-disordered breathing characterized by repetitive episodes of airflow cessation or airflow reduction that occur during sleep as a consequence of collapse of the upper airway [1]. Its prevalence in the world has been estimated at 18% from studies of large populations such as the Sleep Heart Health Study [2]. This frequency is in perpetual growth, yet this disease remains largely underdiagnosed [3]. Organic comorbidities associated with this disease have been widely studied [4,5]. However, psychiatric disorders, especially depression and anxiety, have not attracted so much attention until the past few years with an increase in the number of published studies on this topic. Some studies have demonstrated significantly higher rates of depression among OSA patients [6]. We were interested to the association between OSA and depression/anxiety given the increased morbidity and mortality of these pathologies. Indeed, these pathologies share various biological mechanisms and risk factors suggesting a potential bidirectional association between them [7]. They are responsible of consequent decreased of the quality of life and considerable social and professional impact [7]. These repercussions are more severe when these diseases are associated [8].

However, because of the overlap between symptoms of these psychological disturbances and OSA (fatigue, daytime sleepiness, poor concentration...) [7,9], depression and anxiety may stay undiagnosed in OSA patients. Given the evidence that adequate treatment of OSA with continuous positive airway pressure (CPAP) may be influenced by psychological conditions, the early detection of depressive or anxious symptoms in OSA may be a challenge for the clinicians [9]. In Tunisia, this association is still poorly known by the general public and competent medical centers.

The primary aim was to determine the prevalence and the predictive factors of depression and anxiety in OSA patients. The secondary aim was to investigate the association between the severity of OSA and these psychiatric disorders.

METHODS

A cross-sectional study was performed in A. Mami Hospital (Ariana, Tunisia) between January 2014 to June 2016. Participants were adult patients who visited our sleep laboratory for evaluation of suspected OSA. Their chief complaints were OSA-related symptoms such as snoring, stopping breathing during sleep, choking, gasping during sleep, or excessive daytime sleepiness (EDS).

Inclusion criteria: The study inclusion criteria were an age of >18 years and a confirmed incident case of OSA based on the Apnea-Hypopnea Index (AHI).

Exclusion criteria: Patients were excluded if they had psychiatric or significant comorbidity (malignancy , severe heart failure, stroke), if they were previously diagnosed with OSA, if they were previously treated for OSA, or if they refusal to filled out or fill out psychological questionnaires incompletely.

Sample size calculation

The sample size was calculated according to this formula:

$N = Z^2 P(1-P)/d^2 = 92.16$. Where N is the sample size, Z is the statistic corresponding to the level of confidence 95% = 1.96, P is estimated OSA prevalence in the general population = 4%, d is precision = 0.04.

One hundred incident cases of OSA were included, after applying the exclusion criteria, 80 cases were retained (Figure 1)

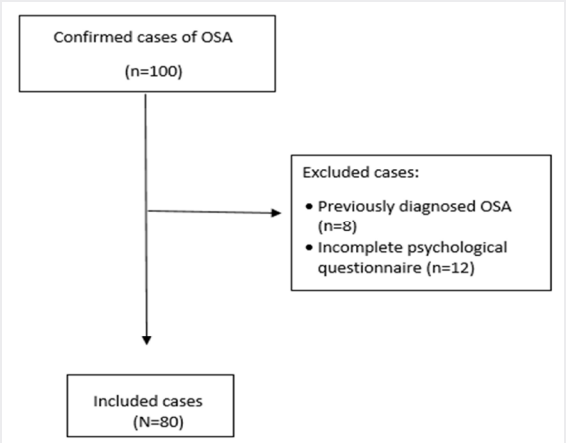

Figure1. Study flow chart

OSA diagnosis

OSA was diagnosed with a polygraphy (Embletta, Cidelec) including recording of oxygen saturation by finger probe pulse oximetry, heart rate, thoracic and abdominal movements, nasal airflow and pressure, snoring, and body position. The AHI was calculated as the number of apneas and hypopneas per hour of total sleep time.

Apnea and hypopnea were scored according to the American Academy of Sleep Medicine guidelines [10]. Apnea was defined as a cessation in airflow of at least 10 seconds [10]. Hypopnea was defined as a >30% or greater reduction in airflow from the baseline value lasting ≥10 s and associated with at least 3% oxygen desaturation [10].

We defined OSA categories according to commonly used clinical cutoffs, i.e., no OSA (AHI <5), mild OSA (AHI ≥5 but <15), moderate OSA (AHI ≥15 but <30), and severe OSA (AHI ≥30) [10].

Daytime sleepiness

Daytime sleepiness was assessed by the Epworth Sleepiness Scale (ESS) [11] which was translated in Arabic. This is a commonly used self-administered scale with eight items about how easily the respondent would fall asleep in different situations. The ESS score ranges from 0 to 24, and a score ≥ 10 indicates EDS [12].

Clinical assessment

Demographic and clinical data were assessed. Symptoms including fatigue, daytime sleepiness, cognitive disorders (deficits in attention, executive function, episodic memory, visuospatial and constructional abilities, and psychomotor speed), anhedonia, libido disorders, and suicidal ideas were taken. A detailed history of hypertension, diabetes, dyslipidemia, cardiac and cerebrovascular diseases, respiratory, neurological and psychiatric disorders was recorded. Current smoking pattern, alcohol use and eventual medication use were analyzed, with the latter focusing on anxiolytic, antidepressant and hypnotic medications. Anthropometric measurements including height, weight, Body Mass Index (BMI) was measured for all subjects at baseline. BMI was calculated as body weight divided by the square of height (kg/m2). Obesity was defined as a BMI ≥30 kg/m^2 [13].

Depression and anxiety diagnosis

Anxiety and depression symptoms were assessed using the Hospital Anxiety and Depression Scale (HADS). The HADS was developed in 1983 [14,15] to identify caseness (possible and probable) of depression and anxiety disorders among patients in nonpsychiatric hospital clinics. It is a self-rating 14 items scale consisting seven subscale of depression (HADS-D) and seven subscale of anxiety (HADS-A). All participants gave written informed consent before HADS.

Possible total scores on both subscales range from 0 to 21 HADS subscale scores range from 0 to 21 and can be used to categorise mood as: 0–7 'non-case'; 8–10 'possible case'; and 11–21 for 'definite case' of depression or anxiety [16]. For the main analyses, we defined depression and anxiety 'non-caseness' as having a HADS score of 0–7; and 'caseness' as having a HADS score of 8–21, as a cut-off score of 8 was found to be optimal for best sensitivity and specificity [16]. A score of 8 or higher is indicative of suffering from either anxiety or depression [16]. Patients completed the validated Arabic version of the HADS [17]. In addition, they answered questions about whether they were currently in treatment for mental diseases and whether they had been diagnosed previously with depression or anxiety.

Data analysis

For each patient, the following categorical variables were collected:

- Sex, marital status, occupation and socio-economic status.
- Comorbidity: diabetes, hypertension, coronary artery disease, dyslipidemia, dysthyroidism, asthma and chronic obstructive pulmonary disease (COPD).
- Lifestyle habits: smoking and alcohol consumption.

Similarly, the following quantitative variables were obtained for each patient:

- Age, weight and height.
- Biological data: total cholesterol level; LDL (Low Density Lipoprotein) level; HDL (High Density Lipoprotein) level; Triglycerides level and thyroid hormone levels.
- Heart rate and respiratory rate, oximetry and spirometry data
- Score data: AHI, ESS and HADS.

Data analyses were conducted using IBM SPSS Statistics Version 16 software. Means with standard deviations were used to describe quantitative variables. Absolute frequencies and percentages were used to describe qualitative variables. A chi-square test or a Fisher's exact test were used to compare percentages of qualitative data between groups of patients with or without depression and anxiety. Student's t-test was applied to compare mean values of quantitative variables between groups of patients with or without depression and anxiety. Analysis of variance (ANOVA) was used to compare mean values of quantitative variables by OSA severity groups. Pearson's coefficient was used to test the correlation between apnea severity, anxiety and depression. The standard p threshold of 0.05 was considered significant for all analyses.

The Binary logistic regression model was used to identify independent predictors of psychiatric disorders. Depression and anxiety were the two dependent variables. The inclusion of independent variables in the regression models was made when the significance level was less than 0.2.

Ethical consideration

All subjects gave consent to participate in the study after appropriate information was given. The study was approved by the Ethics Committee of the A. Mami Hospital.

RESULTS

Eighty newly diagnosed OSA patients were included in this study. The patients' characteristics are shown in Table 1. They were 28 men (35%) and 52 women (65%) and their mean age was 54.83 ± 13.12 yr. Patients were aged more than 40 years in 90%, married in 75% and employed in 50%. The most common comorbidities were hypertension (60%), dyslipidemia (43.8%), Type 2 diabetes (37.5%), coronary artery diseases (CAD) (10%) and dysthyroidism (15%). Obesity was present in 83.8% of all participants and the mean BMI was 34.7±6.14 kg/m2. The ESS score was ≥10 in 40%. The mean AHI score was 36.52±25 events/hour. Twenty one (26.2%) patients had mild OSA, 6 (7.5%) had moderate, and 53 (66.3%) had severe OSA. Table 2 shows that obesity, fatigue, libido disorder, cognitive disorder and excessive daytime sleepiness were more reported in severe OSA, p value were respectively p=0.033, p=<10^{-3} , p=0.021, p=<10^{-3}and p=0.04.

Table 1. Patients characteristics and Comparison of variables according to the Apnea Hypopnea Index .

	Number (%)	Means ± SD	AHI			P-value
			5-15 N (%)	15-30 N (%)	>30 N (%)	
Age (yearr)	-	54.83±13.12				
Age < 40	8 (10)		4 (50.0)	1 (12.5)	3 (37.5)	0.099
Age ≥ 40	72 (90)		17 (23.6)	5 (6.9)	50 (69.4)	
Sex		-				
Male	28 (35)		6 (21.4)	2 (7.1)	20 (71.4)	0.861
Female	52 (65)		15 (28.8)	4 (7.7)	33 (63.5)	
Marital status		-				
Married	60 (75)		16 (26.7)	3 (5.0)	41(68.3)	0.349
Not Married	20 (25)		5 (25.0)	3 (15.0)	12 (60.0)	
Occupation		-				
Employed	40 (50)		8 (20.0)	4 (10.0)	28 (70.0)	0.386
Not employed	40 (50)		13 (32.5)	2 (5.0)	25 (62.5)	
Comorbidity		-				
Hypertension	48 (60)		9 (18.8)	4 (8.3)	35 (72.9)	0.178
Type 2 diabetes	30 (37.5)		4 (13.3)	3 (10.0)	23 (76.7)	0.120
Dyslipidemia	35 (43.8)		5 (14.3)	3 (8.6)	27 (77.1)	0.099
Coronary artery diseases	8 (10)		0	1 (12.5)	7 (87.5)	0.225
Dysthyroidism	12 (15)		3 (25.0)	0	9 (75.0)	0.114
BMI (kg/m2)	-	34.7±6.14				
BMI< 30	13 (16.2)		7 (53.8)	0	6 (46.2)	**0.033**
BMI ≥ 30 (obesity)	67 (83.8)		14 (20.9)	6 (9.0)	47 (70.1)	
Symptoms		-				
Fatigue	47 (58.7)		3(6.4)	1(2.1)	43(91.5)	**$<10^{-3}$**
Suicidal Ideas	8 (10)		1(12.5)	1(12.5)	6 (75.0)	0.466
Libido disorder	28 (35)		3(10.7)	1(3.6)	24(85.7)	**0.021**
cognitive disorder	49 (61.3)		3(6.1)	2(4.1)	24(89.8)	**$<10^{-3}$**
Anhedonia	26 (32.5)		5(19.2)	1(3.8)	20(76.9)	0.364
ESS	-	8.35±5.01				
0-10	48 (60)		18(37.5)	5(10.4)	25 (52.1)	**0.04**
10-24	32 (40)		3 (9.4)	1 (3.1)	28 (87.5)	
AHI		36.52±25				
[5 - 15[	21 (26.2)		-	-	-	-
[15 – 30[	6 (7.5)		-	-	-	-
≥ 30	53 (66.3)		-	-	-	-

AHI: Apnea-Hypopnea Index, BMI: Body mass index, ESS: Epworth Sleepiness Scale

Table 2. Depression and Anxiety data for women and men

	Men N=28	Women N=52	p
HADS score of depression (mean)	5.39 ± 3,71	9.79 ± 4,64	**10^{-4}**
Depression (n, %)	2(7.1%)	26(92.9%)	**10^{-4}**
Moderate depression (n,%)	1(5.9%)	16(94.1%)	1
Severe depression (n, %)	1(9.1%)	10(90.9%)	
HADS score of anxiety (mean)	6.89 ± 3.37	10.67 ± 4.08	**10^{-4}**
Anxiety, (n, %)	4(11.4%)	31(88.6%)	**10^{-4}**
Moderate anxiety (n,%)	4(15.4%)	22(84.6%)	0.553
Severe anxiety (n, %)	0	9(100%)	

HADS: Hospital Anxiety and Depression Scale

Depression symptoms were present in 28 (35%) of patients. Thirty five (43.8%) patients were positive for anxiety symptoms. The mean HAD score of depression and of anxiety were 9±4.8 and 9.5±4.23 respectively.

Both depressive and anxious OSA patients had more libido disorder (p=0.011, p=0.0007; respectively), anhedonia ($p=10^{4}$, $p=10^{-4}$respectively) and suicidal ideas (p=0.002, p=0.019 respectively). They were less smokers (p=0.003, p=0.024 respectively) compared to patients without depression or anxiety. Moreover, depressed OSA patients had lower socio-economic condition (p=0.019) more coronary artery diseases (p=0.019) and less cognitive disorder (p=0.005).

Comparison analysis between sexes revealed that women demonstrated more severe levels of HADS-D score and HADS-A score than men ($p=10^{-4}$, $p=10^{-4}$ respectively). Depression and anxiety symptoms were more common in women than in men (92.9% vs. 7.1%, $p=10^{-4}$for depressive symptoms and 88.6% vs. 11.4%, $p=10^{-4}$ for anxiety) (**Table 2**).

Table 3. Univariate and multivariate analysis: Factors predicting depression in OSA patients.

	Univariate regression			Multivariate regression		
variable	**OR**	**CI 95%**	**P value**	**OR**	**CI 95%**	**P value**
Sex (female)	7.407	0.806 – 66	0.078	11.433	1.188 – 110.029	**0.035**
Age	1.075	0.991 – 1.166	0.083			
Low SES	7.432	0.701 – 78.759	0.096			
Smoking	5.223	0.391 – 69.830	0.211			
Coronary artery disease	21.770	1.855 – 255.472	**0.014**	39.587	2.444 – 641.183	**0.010**
Dyslipidemia	2.883	0.358 – 23.225	0.320			
Fatigue	1.011	0.174 – 5.879	0.990			
Cognitif disorder	1.278	0.180 – 9.055	0.806			
Libido disorder	35.674	1.556 – 618.080	**0.025**	8.704	1.108 – 68.378	**0.040**
Anhedonia	57.109	7.627 – 427.640	**10^{-4}**	75.016	8.980 – 626.688	**10^{-4}**

SES: socio-economic status, CI: Confidence Interval, OR: Odds Ratio

Correlation analysis of the HADS-D and AHI showed no significant correlations between the 2 parameters (r=0,095; p=0,404) (Figure 2). The HADS-A was also not associated with AHI (r=0,212; p=0,059) (Figure 3).

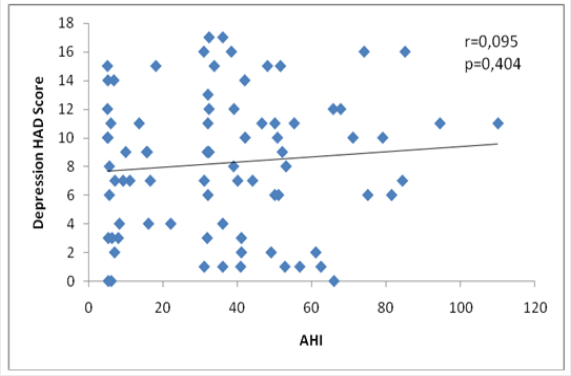

Figure 2. Correlation between depression HAD score and AHI.

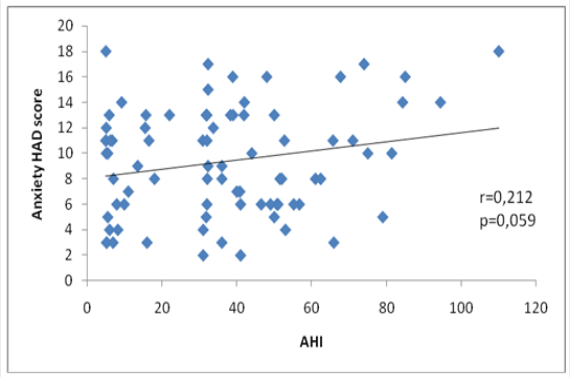

Figure 3. Correlation between anxiety HAD score and AHI.

Female-sex (p=0.035), libido disorder (p=0.040), anhedonia ($p=10^{-4}$) and coronary artery diseases (p=0.010) were identified by the multivariate analysis as predictive factors for depression in OSA patients (**Table 3**). Predictive factors for anxiety were female-sex (OR=7.102; CI95%=1.861-27.096; p=0.004) and libido disorder (OR=4.093; CI95%=1.249-13.412; p=0.02).

DISCUSSION

The first finding of our study is the prevalence of comorbid depression and anxiety of newly diagnosed and untreated OSA patients affecting 35 % and 43.8% of patients respectively.

Secondary, we found that female-sex and libido disorder were the predictive factors for anxiety and depression in OSA patients. Anhedonia and coronary artery diseases also increase risk of depression in OSA patients.

The prevalence fluctuated considerably for both depression (5–63%) [18] and anxiety (11–70%) [19] in patients with OSA. In a recent meta-analysis published in 2018 [20], the prevalence of depressive and anxious symptoms in OSA patients were 35% and 32%, respectively.

Variations in depression and anxiety prevalence are affected by the variability of the mood assessment methods [18]. These scales and questionnaires included [18], but were not limited to, the Minnesota Multiphasic Personality Inventory [21] (MMPI), Beck Depression Inventory [22] (BDI), Center for Epidemiological Studies Depression Scale [23] (CES-D), Hospital Anxiety and Depression Scale [24] (HADS), the Profile of Mood States [25] (POMS), and the Zung Depression Rating Scale [26] (ZDRS).

We opted for the use of the HADS in this study because its Arabic version was validated. In addition, it is the easiest to practice and it is dedicated to non-psychiatrists practitioners

Variation of depression and anxiety prevalence can also be explained by the overlap between mood alterations and OSA-related symptoms [6,7]. OSA and depression share a number of clinical symptoms, including fatigue, EDS, and cognitive disorders. The particularity of cognitive impairment in OSA patients is that they mainly affect attention, executive function, and psychomotor speed [27]. OSA patients with depression may also have depressive symptoms for years and yet remain undiagnosed [18].

The mixed findings among studies can be also explained by differences in sample size, study population, sex distribution and age [28]. In our study, depressive and anxious patients were comparable to non depressive and non anxious patients as regards age, BMI, and AHI However, they showed significant differences as regard sex. Femele-sex had more depression and anxiety symptoms and was a predictive factor of these two pschychiatric disorders. It is known that depressive or anxiety disorders are more common in women [29,30] . Furthermore, women with OSA are more likely to report these two diseases [31]. A large cross-sectional study (9,714 patients) conducted in the United States found a similar prevalence of depression in men (odds ratio [OR]=2.4) but even greater prevalence in women with OSA (OR=5.2) compared to patients without OSA [32]. High anxiety scores were observed in female patients in several studies involving OSA patients [6,33] , which is consistent with our results. The question is whether the high prevalence of depression or anxiety in women is related to the impact of OSA or if it is related to intrinsic characteristics of the sex. A sex differences in the clinical spectrum of OSA was described, with females reporting more frequently fatigue, perception of reduced mood and quality of life, poor and bad sleep, and symptoms that overlap depressive symptoms [6, 34].

Due to the fact that in our country the use of tobacco is less prevalent among women than among men [35] smoking attitude was less observed in patients with depressive and anxious symptoms; predominantly women in our study (92.9% and 88.6% respectively).

Depressed patients in our study had lower socio-economic condition compared to those without depression. Depression associated to OSA is more common in patients with poor family support, who live alone, and have a lack of social support [36]. OSA patients with depression had also more CAD. In fact, people who are depressed have a high risk of developing CAD [9,37,38] . In the other hand, the prevalence of depression in patients with CAD has been estimated at between 17% and 27% [39, 40]. High OSA morbidity with CAD is reported [41,42]. However, there is a lack of studies investigating mood problems in CAD patients with OSA. In a recent study, Balcan et al. concluded that OSA was associated with depressive mood in adults with CAD [43].

In addition to symptoms, OSA and depression share various biological mechanisms and risk factors suggesting a potential association between them [44].

Biological theories on the pathophysiology and temporal relationship between depression and OSA were suggested to explain the strong association between the two diseases. Some propose a unidirectional causal relationship because poor sleep quality can easily affect mood and mental health. Others suggest a bidirectional relationship explained by a common pathophysiology affecting both depression and upper airway dilator activity via increased proinflammatory cytokines causing neural injury, or abnormalities in serotonin uptake [7,45] .OSA was identified as an independent risk factor for depression [46]. In two longitudinal studies, [47, 48] the odds for developing depression were increased 2.0-fold (95% confidence interval [CI]: 1.4–2.9) in participants with mild OSA and 2.6-fold (95% CI: 1.7–3.9) in those with moderate to severe OSA.

It has been well established through various studies that there is an association between OSA and depression [9] . However, the relationship between anxiety and OSA is unclear [19]. Anxiety may result from neuronal damage that occurs during OSA [49,50].

Kumar et al. [49] observed permanent brain structural abnormalities using magnetic resonance imaging in OSA patients with anxiety. These changes were particularly pronounced in the cerebral cortex, thalamus, hippocampus and amygdale [49]. Yadav et al. [50] studied the metabolic changes that occur in the insular cortex in patients with OSA using proton spectroscopy and noted positive correlations between the choline / creatine ratio (Cho / Cr) in the right insular cortex on the one hand and the Beck Anxiety Inventory score on the other, suggesting that the metabolic abnormalities observed in these sites could contribute to levels of higher anxiety.

As OSA is associated with symptoms such as fatigue and the presence of a mood disorder, depression and anxiety would be expected more common in severe OSA compared to non severe OSA [51]. However conflicting results have been produced by many studies regarding the association between OSA severity and mental disorders [52]. In our study, OSA severity did not contribute to depression. This result was in line with several other studies which failed to find an association between OSA severity and the prevalence of depression [19,53,54,55,56,57]. This was in contrast to the conclusions of an Australian study of 426 participants [58]. In this study, the prevalence of depressive symptoms was associated positively to OSA severity.

Similarly to the findings of numerous studies [19,53,59], the severity of OSA was not found to be related to the HADS-A in our study. However, in a recent study, authors concluded that symptoms of anxiety and depression were associated negatively with OSA severity even after adjusting for several relevant confounders [51]. Similar to these findings, a French study including 825 elderly patients reported a close to significant lower prevalence of anxiety and reduced anti-anxiety treatment with increased OSA severity [6]. The reason why these studies showed

opposite results concerning the relationship between OSA severity and psychological symptoms is unclear. The use of different mood scales may have had an impact. In addition, a methodological limitation was noticed in many of these studies which were based on small samples. Thus, more large-scale OSA population studies are necessary to elucidate further the relationship between OSA and mental disorders. These studies may provide sufficient statistical power and enable statistical adjustment for relevant confounding factors (sex, age, smoking or alcohol use and obesity) [52] known to have influence in the prevalence of both OSA and mental disorders.

Most of the clinicians do not suspect depression or anxiety when they explore patients referred with suspicion of OSA resulting in delayed diagnosis [60]. Increased awareness of the relationship between these mental disorders and OSA and the appropriate use of assessment tools might significantly improve diagnostic for both disorders. In our study, we opted for the use of the Arabic version of the HADS a well validated questionnaire for depression and anxiety. It does not include questions about vegetative symptoms such as sleep problems or fatigue often present in somatic conditions such as OSA. It has been validated as an accurate screening tool among patients with suspicion of OSA [51, 61].

For efficient screening, it is ideal to target high-risk groups. There is further data suggesting that certain patient characteristics are predictive of developing depression in OSA. Ishman et al. in a case-control study suggested higher scores on ESS [62]. In a 1,327 Chinese patients with OSA, predisposing factors for depressive status were single marital status, AHI, hypoxemia, and reduced family and social supports [36]. In our study, attention should be paid to female-sex and libido disorder as a determinant of depressive and anxious mood in OSA patients. Moreover, anhedonia and CAD, was identified as a predictive factors of depression in this population. The high prevalence of depressive states in OSA patients encourages systematic exploration of their thymic state. CPAP treatment was shown to be effective in improving depression and neurocognitive functions in these patients, even partially. Specialized care may be needed in cases of residual anxio-depressive symptoms. Further studies evaluating the evolution of depression and anxiety in OSA Tunisian population are needed.

The present study has several strengths and limitations. Our study clearly showed the importance of the psychiatric component in OSA patients. To our knowledge, this is the first study of the prevalence and the predictive factors of depression and anxiety in OSA Tunisian population. Another strength was that we opted for the use of the validated Arabic version of the HAD scale.

However, our study has several limitations. First, this work was conducted in a hospital setting, particularly in a pulmonology department specializing in the management of sleep disorders from which we recruited our patients. Therefore, patients with the most severe stages of OSA were more likely to occur in our sample, while mild to moderate OSA were under-represented. This prevented us from generalizing our results to the rest of the population due to the non-representativeness of our sample. Second, some data which may influence our results were not consistently available such as nocturia, insomnia, neck circumference, visceral obesity and upper airway characteristics.

Finally, the use of polygraphy and not polysomnography, for diagnosing OSA can underestimated AHI [63]. It is possible that this underestimation may be more pronounced in patients with depression/anxiety, due to reduced sleep efficiency among patients suffering from these disorders usually accompanied with short sleep duration [51]. Third, all patients were studied when they were hospitalized for 1 night. Hospitalization may have influenced the results.

In conclusion, our study demonstrated that depressive and anxious symptoms are prevalent in OSA patients. However, OSA severity did not contribute to depression and to anxiety. The determinants of comorbid depressive and anxious mood were female-sex and libido disorder. Anhedonia and coronary artery diseases were also identified as predictive factors of depression in this population.

Our findings may have important clinical implications. Due to the high prevalence of depression and anxiety in patients with OSA, depression and anxiety screening is important in this population. We recommend standardizing the use of the HADS for OSA patients, especially in sleep centers such as our center, in order to detect any depressive or anxiety disorders that may accentuate the clinical symptoms, alter the quality of life of the patients and compromise the treatment. We also recommend associating a psychologist or even a psychiatrist in the management of OSA patients.

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