SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Khan *et al*., 2026
doi: 10.21276/SSR-IIJLS.2026.12.1.40

Original Article

open access

Relationship of Obstructive Sleep Apnea (OSA) with Anxiety and Depression: A Comprehensive Analysis

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Received: 14 Sep 2025/ Revised: 25 Oct 2025/ Accepted: 24 Dec 2025

ABSTRACT

Background: Obstructive sleep apnea (OSA) is common in adults and is associated with significant cardiometabolic and neurocognitive complications. In addition, depression and anxiety are frequently observed comorbidities that adversely affect quality of life, daytime functioning, and healthcare utilization. Recognizing these psychiatric symptoms is important for timely diagnosis, improved treatment adherence, and comprehensive patient care. The primary aim of this study was to determine the prevalence and predictors of depression and anxiety in patients with OSA, while the secondary aim was to assess their association with OSA severity.

Methods: A cross-sectional study of untreated OSA patients' illness 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: 100 patients were included (mean age: 42.65±9.76 yr; females: 61 (61%); mean Body mass index (BMI):37.7±2.14 kg/m^2). The prevalence of depression and anxiety was 29.8 % and 33.2% of patients, respectively. Depressed OSA patients had lower socio-economic condition (p= 0.01), more coronary artery diseases (CAD) (p=0.01) and less cognitive disorder (p= 0.005).

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

Key-words: Obstructive sleep apnea, Depression, Anxiety, Sleep monitoring, HAD Scale

INTRODUCTION

Obstructive sleep apnoea (OSA) is the most common form of sleep-disordered breathing, characterized by repetitive episodes of airflow cessation or reduction during sleep due to upper airway collapse [1]. Its prevalence worldwide has been estimated at 18% in large-population studies, 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]. An association between OSA and depression/anxiety was evaluated in the present study due to the increased morbidity and mortality of these pathologies. Indeed, these pathologies share common

How to cite this article
Khan MZ, Usmani KM, Qayum S, Gautam N, et al, Shivani Jandial. Relationship of Obstructive Sleep Apnea (OSA) with Anxiety and Depression: A Comprehensive Analysis. SSR Inst Int J Life Sci., 2026; 12(1): 9251-9256.

Access this article online
https://iijls.com/

doi: 10.21276/SSR-IIJLS.2026.12.1.40

biological mechanisms and risk factors, suggesting a potential bidirectional association [7].

They are responsible for the consequent decrease in the quality of life and for a considerable social and professional impact. 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 psychological conditions may influence adequate treatment of OSA with continuous positive airway pressure (CPAP), the early detection of depressive or anxious symptoms in OSA may be a challenge for clinicians [9].

In India, this association is still poorly known by the general public and competent medical centres. 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.

MATERIALS AND METHODS

Place of study- A cross-sectional study was conducted between January 2024 and June 2025. 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 age>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 refused to fill 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.

Diagnosis of OSA- After applying the exclusion criteria, 118 incident cases of OSA were included; 100 cases were retained. Study flow chart: OSA diagnosis. OSA was diagnosed with a polysomnography. 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. Hypopnea was defined as a >30% reduction in airflow from baseline lasting ≥10 s and associated with at least 3% oxygen desaturation. We defined OSA categories according to commonly used clinical cut-offs: no OSA (AHI < 5), mild OSA (AHI ≥ 5 but < 30), and severe OSA (AHI ≥ 30) [10].

Daytime sleepiness- Daytime sleepiness was assessed by the Epworth Sleepiness Scale (ESS). This is a commonly used self-administered scale with 8 items assessing how easily the respondent falls asleep in different situations. The ESS score ranges from 0 to 24, and a score ≥ 10 indicates EDS.

Clinical assessment- Demographic and clinical data were assessed. Symptoms including fatigue, daytime sleepiness and cognitive disorders were also taken into consideration. A detailed history of hypertension, diabetes, dyslipidemia, cardiac and cerebrovascular diseases, respiratory, neurological and psychiatric disorders was recorded. Anthropometric measurements, including height, weight and Body Mass Index (BMI), were measured for all subjects at baseline. BMI was calculated as body weight divided by the square of height (kg/m^2). Obesity was defined as a BMI ≥30 kg/m^2 [11].

Depression and anxiety diagnosis- Anxiety and depression symptoms were assessed using the Hospital Anxiety and Depression Scale (HADS). The HADS was developed in 1983 to identify the caseness (possible and probable) of depression and anxiety disorders among patients in nonpsychiatric hospital clinics. It is a self-rating 14-item scale consisting of seven subscales of depression (HADS-D) and seven subscales 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-

doi: 10.21276/SSR-IIJLS.2026.12.1.40

case'; 8–10 'possible case'; and 11–21 for 'definite case' of depression or anxiety. For the main analyses, we defined depression and anxiety 'non-caseness' as a HADS score of 0–7, and 'caseness' as a score of 8–21, as a cut-off score of 8 was found to be optimal for sensitivity and specificity [12]. A score of 8 or higher is indicative of suffering from either anxiety or depression [13].

Statistical Analysis- Data were analyzed using IBM SPSS Statistics Version 16. Quantitative variables were expressed as mean±standard deviation, and qualitative variables as frequencies and percentages. The chi-square or Fisher's exact test was used to compare categorical variables, while Student's t-test was applied for continuous variables between patients with and without depression or anxiety. ANOVA was used to compare mean values across OSA severity groups. Pearson's correlation assessed the relationship between OSA severity, depression, and anxiety. A p-value <0.05 was considered statistically significant. Binary logistic regression analysis was performed to identify independent predictors of depression and anxiety.

RESULTS

Total 100 newly diagnosed OSA patients were included in this study. There were 39 men (39%) and 61 women (61%), with a mean age of 42.65±9.76 yrs. The most common comorbidities were hypertension (70%), dyslipidemia (48.9%), Type 2 diabetes (48.5%), coronary artery disease (CAD) (22%) and dysthyroidism (17%). Obesity was present in 72.2% of all participants and the mean BMI was 37.7±2.14 kg/m2. The ESS score was ≥10 in 44%. The mean AHI score was 38.62±35 events/hour. Twenty-eight (28%) patients had mild OSA, 44% had moderate, and 28% had severe OSA (Fig. 1). obesity, fatigue, cognitive disorder and excessive daytime sleepiness were more reported in severe OSA; p-values were respectively p=0.033, p≤10, p=0.021, p≤10, p=0.04.

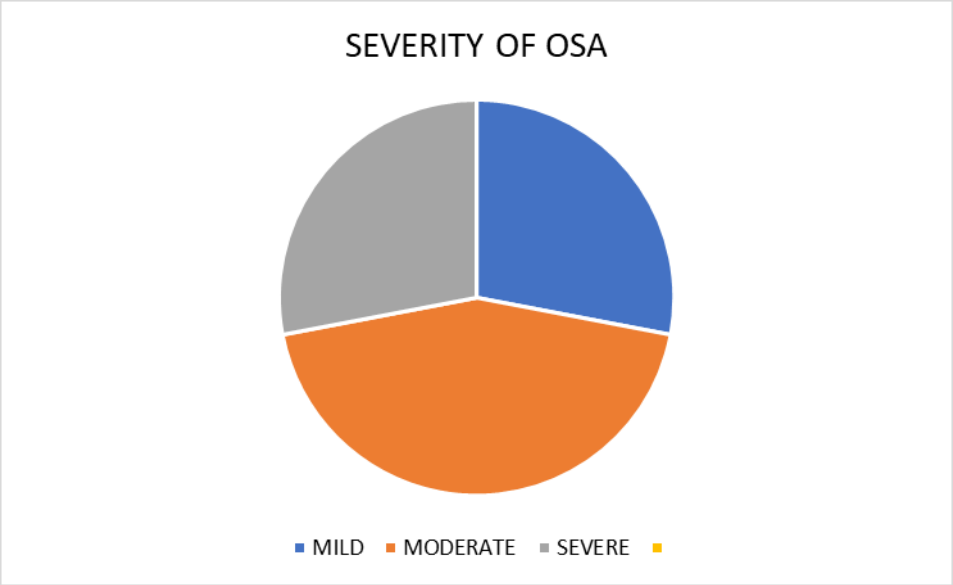

Fig 1: Severity of OSA

Depression symptoms were present in 19% of patients. 42% patients were positive for anxiety symptoms. The mean HAD scores of depression and anxiety were 9±4.8 and 9.5±4.23, respectively (Table 1). There were fewer smokers (p=0.003, p=0.02, respectively) compared to patients without depression or anxiety. Moreover, depressed OSA patients had lower socioeconomic condition (p=0.01), more coronary artery diseases (p=0.019) and less cognitive disorder (p=0.005). A comparison between the sexes revealed that women had higher HADS-D and HADS-A scores than men.

Correlation analysis of the HADS-D and AHI showed no significant correlations between the 2 parameters (r=0.095; p=0.40). The HADS-A was also not associated with AHI (r=0.212; p=0.059). Depression and anxiety symptoms were more common in women than in men. 27.86% vs. 5.1% for depressive symptoms and 60.65% vs. 8.1% for anxiety. Female sex (p=0.035) and coronary artery diseases (p=0.01) were identified by the multivariate analysis as predictive factors for depression in OSA patients.

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Table 1. Depression and Anxiety data for women and men

	Men (N=39)	**Women (N=61)**
HADS score of depression (mean)	8.5±3.0	10.5±4.64
Depression (n)	2	17
HADS score of anxiety (mean)	8.89±3.07	9.67±4.08
Mild anxiety (n)	1	12
Moderate anxiety (n)	2	21
Severe anxiety (n)	2	4

DISCUSSION

Our study demonstrated a considerable prevalence of comorbid depression (19%) and anxiety (42%) among newly diagnosed and untreated OSA patients. These findings are consistent with prior reports showing wide variability in prevalence, with depression ranging from 5–63% [14] and anxiety from 11–70% [15] among OSA populations. A meta-analysis published in 2018 reported pooled prevalence rates of 35% for depressive symptoms and 32% for anxiety symptoms in OSA patients [16]. Such heterogeneity may be attributed to differences in study design, population characteristics, and especially the variability of mood assessment tools [17]. Various instruments have been used across studies [18], including the Minnesota Multiphasic Personality Inventory (MMPI), Beck Depression Inventory (BDI), Center for Epidemiological Studies Depression Scale (CES-D), Hospital Anxiety and Depression Scale (HADS), Profile of Mood States (POMS), and Zung Depression Rating Scale (ZDRS). In the present study, we selected HADS due to its ease of administration and suitability for non-psychiatric clinical settings.

The variation in reported prevalence may also be explained by symptom overlap between OSA and mood disorders [6,7]. OSA and depression share common manifestations such as fatigue, excessive daytime sleepiness (EDS), and cognitive dysfunction. Cognitive impairment in OSA predominantly affects attention, executive function, and psychomotor speed [19], which may mimic or exacerbate depressive symptomatology. Moreover, depressive symptoms in OSA patients may remain undiagnosed for years [14]. Differences in sample size, demographic composition, age distribution, and sex ratio may further explain inconsistent findings across studies [20].

In our cohort, patients with depression or anxiety did not differ significantly from non-depressed and non-anxious patients in terms of age, BMI, or AHI. However, sex showed a significant association, with females exhibiting higher rates of both depression and anxiety. Female sex was identified as a predictive factor for these psychiatric disorders. This aligns with established evidence that depressive and anxiety disorders are more prevalent in women [21,22]. Furthermore, women with OSA are more likely to report mood-related complaints [23]. A large cross-sectional study involving 9,714 patients in the United States demonstrated increased odds of depression in men with OSA (OR=2.4), with even higher odds among women (OR=5.2) compared to individuals without OSA [24]. Several studies have similarly observed higher anxiety scores in female OSA patients [25]. Differences in symptom presentation may partly explain these findings, as women with OSA often report fatigue, poor sleep quality, reduced mood, and impaired quality of life—symptoms overlapping with depression [26].

Socioeconomic status also influenced psychiatric outcomes in our study, with depressed patients more frequently belonging to lower socioeconomic strata. Social isolation and limited family support are known contributors to depression in OSA populations. Smoking was less common among depressed and anxious patients in our cohort, likely reflecting the higher proportion of women, among whom tobacco use is less prevalent in our region [27].

Coronary artery disease (CAD) emerged as a significant predictor of depression in OSA patients. The bidirectional relationship between depression and CAD is well documented [9]. Depression increases the risk of developing CAD, and conversely, the prevalence of depression in CAD patients ranges between 17% and 27%. OSA is also strongly associated with cardiovascular

doi: 10.21276/SSR-IIJLS.2026.12.1.40

morbidity. Balcan *et al.* reported that OSA was linked to depressive mood among adults with CAD. Shared biological pathways—including systemic inflammation, oxidative stress, endothelial dysfunction, and neurohormonal dysregulation—may underlie the association between OSA, CAD, and depression.

Although one might expect psychiatric symptoms to be more frequent in severe OSA, the association between OSA severity and mood disorders remains controversial. In our study, OSA severity was not significantly associated with depression, consistent with several prior reports [15]. Similarly, anxiety scores measured by HADS-A were not significantly correlated with AHI severity, as noted in multiple studies [15]. However, some recent investigations have reported either positive or even negative associations between OSA severity and psychiatric symptoms after adjustment for confounding factors. These discrepancies may reflect differences in statistical power, adjustment for confounders such as sex, age, smoking, alcohol use, and obesity, and the complex interaction between physiological severity and subjective symptom perception.

LIMITATIONS

This study has certain limitations, including its hospital-based design, possible underestimation of AHI due to polygraphy, and limited generalizability. Despite these limitations, the findings highlight the importance of systematic psychiatric screening in OSA patients.

CONCLUSIONS

Our study demonstrated that depressive and anxious symptoms are prevalent in OSA patients. However, OSA severity did not contribute to depression or anxiety. Our findings may have important clinical implications. Due to the high prevalence of depression and anxiety in patients with OSA, depression and anxiety screening are important in this population. We recommend standardizing the use of the HADS for OSA patients, especially in sleep centers such as our center, 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 working with a psychologist or psychiatrist to manage OSA patients.

CONTRIBUTION OF AUTHORS

Research concept- Dr Mohammad Zaeem Khan
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Materials- Dr Shazia Qayum
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Data analysis and interpretation- Dr Shazia Qayum
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Critical review- Dr Shazia Qayum
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Final approval- Dr Khalid Masood Usmani

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