Obstructive Sleep Apnea (OSA) and its Association with Depression

Md Adil Faizan[1], K Sri Divya Reddy[2], Geethanjali[3], M Raghuram[4], R Sateesh Babu[5]

How to cite this article:

Md Adil Faizan, K Sri Divya Reddy, *et al.*, Obstructive Sleep Apnea (OSA) and its Association with Depression. RFP Ind Jr of Med Psy. 2024;7(2):43–47.

Abstract

Objective: This study investigates the correlation between Obstructive Sleep Apnea (OSA) and depression, examining how OSA impacts psychiatric well-being and quality of life.

Introduction: Obstructive Sleep Apnea (OSA) is characterized by episodes of partial or complete airway obstruction during sleep, leading to fragmented and non-restorative sleep. OSA has significant implications for mental health, including a strong association with depression and other psychiatric conditions. Despite considerable evidence linking OSA with psychiatric symptoms, the precise nature of this relationship remains ambiguous.

Methods: A cross-sectional study was conducted with 182 participants at a tertiary care center in Telangana from July 2022 to June 2023. Tools employed included the Epworth Sleepiness Scale (ESS), Polysomnography (PSG), and the Hamilton Depression Rating Scale (HAM-D). Participants included those referred for PSG who consented to the study. Exclusions were made for patients with nocturnal oxygen supplementation, recent upper airway surgery, or unstable cardiopulmonary, neurological, or psychiatric conditions.

Results: Out of 90 patients who underwent PSG, 23 were diagnosed with depression. These patients were significantly older (mean age 57.30 years) compared to non-depressed individuals (mean age 52.40 years, $P < 0.001$). Depression was associated with higher HAM-D scores (mean 17.25) and poorer sleep efficiency (68.55%) compared to non-depressed patients (HAM-D mean 8.64, sleep efficiency 72.50%). Additionally, depressed patients exhibited longer sleep latency and REM latency. The Apnoea-Hypopnoea Index (AHI) was significantly higher in depressed patients (mean 26.04) compared to non-depressed patients (mean 20.30, $P = 0.005$). The majority of depressed patients had abnormal PSG results, with 91.3% showing aberrant findings.

Discussion: The study confirms a significant association between OSA and depression. Depression exacerbates OSA symptoms and impairs sleep quality, with substantial implications for patient management and treatment outcomes. The link between OSA and psychiatric conditions such as depression highlights the need for integrated treatment approaches that address both sleep and mental health issues.

Conclusion: OSA is prevalent among individuals with depression, and its impact on psychiatric well-being is considerable. Clinicians should be vigilant in screening for OSA

Author's Affiliation: [1]Resident, [2,3]Assistant Professor, [4,5]Professor, Department of Psychiatry, Mamata Medical College, Khammam, Telangana, India.

Corresponding Author: Md Adil Faizan, Resident, Department of Psychiatry, Mamata Medical College, Khammam, Telangana, India.

E-mail: adilmrpr07@gmail.com

Received on: 20-08-2024 **Accepted on:** 05-10-2024

in depressed patients to improve overall treatment outcomes. Further research is needed to explore the mechanisms underlying the OSA-depression connection and to develop comprehensive management strategies.

Keywords: Obstructive Sleep Apnea, Depression, Polysomnography, Psychiatric Co-morbidity, Sleep Disorders.

INTRODUCTION

Episodes of total or partial airway collapse, accompanied by a drop in oxygen saturation or an awakening from sleep, are the hallmarks of obstructive sleep apnea (OSA). A fragmented, non-restorative sleep is the outcome of this disturbance. The effects of OSA on mental illness, driving safety, quality of life and cardiovascular health aresubstantial1.Up to 20% of the society suffers from sleep disordered breathing, according to epidemiological studies. A portion of these patients—4-5% of the middle-aged population—have obstructive sleep apnoea/hypo-apnoea syndrome, which is characterized by concomitant symptoms of excessive daytime sleepiness caused by their nocturnal breathing disorders[2]. OSA may have a harmful impact on a number of organs, leading to the emergence of cardiovascular disorders and cognitive issues[3].

Through the process of sympathetic stimulation brought on by waking from sleep, the disturbed breathing during sleep exerts its multi organ, harmful effects[4]. Recent epidemiological research has demonstrated the link between untreated OSA and a wide range of harmful health issues, including many psychiatric illnesses like depression and anxiety[5]. The majority of investigations have found a strong link between OSA and psychiatric problems[6]. The biological and/or psycho-social effects of OSA may be what causes the mental alterations[7]. It has been noted that psychiatric comorbidity in OSA patients negatively impacts their quality of life and impairs neurocognitive functioning[8]. OSA has been related to psychological issues including as depression, anxiety, impaired cognitive function, and others. Some psychological deficits can be improved with therapy, while others linger even after therapy[9]. A patient with OSA has a high prevalence of personality disorders and depression. We did this study to

Demonstrate any correlation between the two conditions due to the literature's ambiguity regarding the relationship between OSA and psychiatric symptoms[10].

METHODS

This is a cross sectional study done on 182 participants at a tertiary care centre, Telangana from July 2022 -June 2023 .The tools used in this study were the Eps worth Sleepiness Scale (ESS), PSG (Polysomnography), Hamilton Depression rating Scale(HAM-D), sociodemographic data and anthropometric measurements of each participants.

The people who gave their consent and who were referred for polysomnography (PSG) were included. The people who didn't give consent, who were on nocturnal oxygen supplementation, any upper airway surgery, unstable cardiopulmonary, neurological or psychiatric disease are excluded from study.

RESULTS

Out of 90 patients who had PSG, 23 had depression and had a mean age that was notice ably older (P<0.001) than the general population (57.30 vs. 52.40). Between these two groups, there were no statistically significant differences in neck circumference or body mass index (P> 0.005) (*Table 1*).

Basic characteristics of the study population (age, body mass index, and neck circumference)

Table 1: Comparison of different variable in population with or without depression.

	Mean+SD		P-value
	Depression (n=23)	Others (n=67)	
Age (years)	57.30+10.75	52.40+09.97	<0.001**(S)**
BMI	30.45+4.23	31.62+4.23	0.175
Neck circumference (cm)	37.85+3.54	38.41+3.13	0.359

SD: Standard deviation, BMI: Body mass index, S: Significant

When compared to non-depressive patients, patients with depression had considerably higher mean HAM-D scores (P = 0.0001) (mean standard

deviation [SD] = 17.25+5.35) than non-depressive patients (mean SD 8.64 +6.24). In comparison to non-depressives (mean 72.50%), depression patients' sleep efficiency was considerably worse (P< 0.005). those with depression experienced substantially longer mean sleep latency (23.42 min) than those without depression (21.09 min) (P<0.005).

Similar to sleep latency, mean REM sleep latency was also substantially longer inpatients with depression (76.73 min) than in the general population (72.26 min; P =0.018). Individuals with depression had mean room air, awake SpO2 values that were not statistically different from non-depressed individuals (93.49) (P= 0. 195).

The average nocturnal SpO2 of depressive patients was 83.56% against 85.46%, which was not statistically different from awake SpO2 (P = 0.104) in comparison to non-depressive patients. The mean AHI of patients with depression was 26.04 versus 20.30 (P=0.005(S), substantially higher than that of individuals without depression. Although depression patients' mean ODI was significantly higher than depression patients' mean ODI (24.81 compared 20.59; P = 0.096), this difference was not statistically significant (*Table 2*).

Table 2: Polysomnography observation in depressive patients versus those without depression.

	Mean+SD		P-value
	Depression (n=23)	Others (n=67)	
AHI	26.04+10.54	20.30+12.20	0.005**(S)**
ESS	15.70+3.29	14.66+4.11	0.117
HAM–D	17.25+5.35	8.64+6.24	0.0001**(S)**
Sleep efficiency	68.55+9.42	72.50+8.67	0.002**(S)**
Sleep latency	23.42+09.19	20.09+8.50	0.006**(S)**
REM latency	76.73+13.25	72.26+13.9	0.019**(S)**
Awake Spo2	93.49+4.02	93.36+4.00	0.195
Nocturnal Spo2	83.56+6.44	84.46+6.64	0.104
ODI	24.81+14.53	20.59+15.03	0.096

SD: Standard deviation, AHI: Apnoea Hypo-apnoea index, ESS: Epworth Sleepiness Scale, HAM-D: Hamilton Depression Rating Scale, REM: Rapid eye movement, ODI: Oxygen desaturation index, S: Significant, Spo2: Oxygen saturation.

Snoring 30(44.77%), day time drowsiness 27(40.29%) with a mean ESS of 15.3, Disturbed nocturnal sleep 21 (31.34%), nocturia 20 (29.85%), and witnessed apnoea 14(20.89%) were the predominant presenting symptoms of patients without depression. All of these symptoms were more frequent in depressed patients compared to non-depressed patients, and within depressed patients, they were more frequent in OSA patients compared to non-OSA patients. Only 2(8.69%) of our 23 depressed patients had normal PSG results, whereas the remaining 21 (91.3%) had aberrant results (*Table 3*).

Table 3: Prevalence of Obstructive sleep apnoea in Depression

	Depression		
	No (%)	Yes (%)	
OSA			
Yes	56(85.58%)	20(86.95%)	76(84.44%)
No	11(16.41%)	3(13.05%)	14(15.56%)
Total	67	23	90

P=0.089(NS), OSA: Obstructive sleep apnoea, NS: Not significant

Snoring was reported by 23 patients (100%), day time drowsiness by 20 patients (93.6%), disturbed nocturnal sleep by 18 patients (81.8%), nocturia by 16 patients (75%), and witnessed apnoea by 11

patients (52.3%),when compared to people without depression, patients with depression experienced all of these symptoms more frequently. Every patient with depression who also has OSA snores (*Table 4*).

Table 4: Patient presentation in depression with and without obstructive sleep apnoea

	OSA(%)	NOOSA(%)	Total(%)	P-value
Snoring				
Yes	23(100.0%)+	1(33.3%)	24(92.03%)	0.003*
No	0	2(66.7%)	2(7.69%)	
Witnessed apnoea				
Yes	11(47.82%)	0	11(42.30%)	>0.2
No	12 (52.17)	3(100.0%)	15(57.70%)	
Disturbed sleep				
Yes	18(78.26%)	1 (33.3%)	19(73.07%)	>0.1
No	05(21.74%)	2(66.7%)	07(20.92%)	
Day time sleepiness				
Yes	20(86.95%)	3(100.0%)	23(88.46%)	>0.9
No	3(13.05%)	0	3(11.54%)	
Nocturia				
Yes	16(69.56%)	1(33.3%)	17(65.38%)	>0.1
No	07(30.44%)	2(66.7%)	09(34.62%)	

+-percentage from column heading,*-significant value, OSA–Obstructive sleep apnea.

1 (4.34%) of the patients had modest AHI scores. 11 (52%) had severe OSA, while 09(40.9%) had moderate OSA. Depressive individuals significantly had higher moderate and severe OSA than non-depressive patients (P=0. 034(S) (*Table 5*).

Table 5: Severity of obstructive sleep apnoea based on the apnoea –hypo-apnoea index

	All patient (%)	Depression (%)
Mild	23(25.55%)	1 (4.34%)
Moderate	34(37.77%)	09(39.13%)
Severe	33(36.66%)	11(47.82%)
P	0.032**(S)**	

S: Significant

DISCUSSION

Recurrent upper airway obstruction in OSA, a respiratory disorder that affects sleep, frequently results in episodes of reduced (hypo-apnoea) or no (apnoea) airflow. Specific physiological abnormalities, like fragmented sleep and persistent intermittent hypoxia, are the final result.

OSA is a well-known risk factor for a wide variety of metabolic and cardiovascular conditions, which is a major morbidity in psychiatric patients and may shorten projected life duration.

There is widespread agreement that depression and OSA are closely related, and that both of these illnesses can have an impact on a patient's general well being and disease progression.

There are significant repercussions when depression is present in OSA. Depression complicates treatment, is frequently linked to chronicity, and has deleterious impact on how OSA develops.

The majority of research have demonstrated a substantial correlation between OSA and a psychiatric condition, mainly depression. Depression is a widespread medical condition that is yet poorly understood and managed. It is debatable whether depression is more common in OSA patients than in those who have other chronic illnesses.

OSA is thought to be at risk for depression, which serotonergic neurotransmission can explain. Low serotonin levels might lead to decreased upper respiratory tract muscular tone as well as sleep disruption. Psychiatric comorbidity in OSA varies and depends on a number of factors.

The results of our study imply that OSA may be a significant confounding factor for studies on depression. The reason for this is because neither research study routinely determines its presence. For a more accurate comparison to the general population, there is a critical need for more population-based research of OSA in community-dwelling people with depression.

Studies conducted in the community are necessary to gauge the severity of the comorbidity and enhance patient care. The overall results of our study show a significant and direct connection between depression and OSA.

Most doctors fail to recognize this significant depression comorbidity at first, which delays diagnosis. Screening patients for OSA and prompt psychiatric management can greatly enhance OSA sufferers' quality of life. To properly understand the connection between depression and OSA, more prevalent research must be done.

CONCLUSION

According to the results of our study, OSA is a common condition associated with depression but is generally ignored in the primary care context. There could be neuro-psychiatric side effects from OSA.

In high risk patients who present to them with cognitive and/or emotional issues, psychiatrists need to be vigilant to take OSA into account. As a result, regular depression screening should be included in OSA management and can significantly enhance these patients' quality of life.

To determine the severity of the comorbidity and enhance the therapy of these individuals, additional community-based research are necessary. The bed partner's history should be obtained to support any clinical suspicions.

REFERENCES

1. Slowik JM, Sankari A, Collen JF. Obstructive Sleep Apnea. [Updated 2022 Dec 11]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing; 2024 Jan
2. Epidemiology of sleep apnoea/hypopnea syndrome and sleep-disordered breathing. P. Jennum, R.L. Riha European Respiratory Journal Apr 2009, 33 (4) 907-914; DOI:10.1183/09031936.00180108
3. Abbasi A, Gupta SS, Sabharwal N, Meghrajani V, Sharma S, Kamholz S, Kupfer Y. A comprehensive review of obstructive sleep apnea. Sleep Science. 2021 Apr; 14(2):142.
4. Hakim F, Gozal D, Gozal LK. Sympathetic and catechol aminergic alterations in sleep apnea with particular emphasis on children. Frontiers in neurology. 2012 Jan 30; 3:20505.
5. Osman AM, Carter SG, Carberry JC, Eckert DJ. Obstructive sleep apnea: current perspectives. Nat Sci Sleep.2018Jan23;10:21-34.doi:10.2147/NSS.S124657.
6. Shoib S, Ullah I, Nagendrappa S, Taseer AR, De Berardis D, Singh M, Asghar MS. Prevalence of mental illness in patients with obstructive sleep apnea - A cross-sectional study from Kashmir, India. Ann Med Surg (Lond). 2022 Jul 6; 80:104056. doi:10.1016/j.amsu.2022.104056.
7. Olaithe M, Nanthakumar S, Eastwood PR, Bucks RS. Cognitive and mood dysfunction in adult obstructive sleep apnoea (OSA): Implications for psychological research and practice. Translational Issues in Psychological Science.2015Mar;1(1):67.
8. Turner K, Zambrelli E, Lavolpe S, Baldi C, Furia F, Canevini MP. Obstructive sleep apnea: neurocognitive and behavioral functions before and after treatment. Functional Neurology. 2019; 34(2):71-8.
9. Riemann D, Benz F, Dressle RJ, Espie CA, Johann AF, Blanken TF, Leerssen J, Wassing R, Henry AL, Kyle SD, Spiegel halder K. Insomnia disorder: State of the science and challenges for the future. Journal of sleep research. 2022 Aug; 31(4):e13604.
10. Shoib S, Malik JA, Masoodi S. Depression as a Manifestation of Obstructive SleepApnea. JNeurosciRuralPract.2017 Jul-Sep; 8(3):346-351. doi:10.4103/jnrp.jnrp_462_16.