Section: Miscellaneous

Original Research Article

STUDY OF OBSTRUCTIVE SLEEP APNEA IN OBESE PATIENTS WITH TYPE 2 DM

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is increasingly recognized as a common comorbidity in patients with type 2 diabetes mellitus (T2DM), particularly in those with obesity. OSA is associated with intermittent hypoxia, sleep fragmentation, and metabolic dysregulation, which may adversely affect glycaemic control and cardiovascular risk. However, data on the burden and clinical correlates of OSA among obese patients with T2DM in the Indian population remain limited.
Materials and Methods: This hospital-based cross-sectional study included 163 obese patients (BMI ≥30 kg/m²) with type 2 diabetes mellitus. All participants underwent detailed clinical and anthropometric assessment, laboratory evaluation including HbA1c, and overnight attended polysomnography. OSA was diagnosed using the apnea–hypopnea index (AHI) and classified as mild, moderate, or severe. Associations between OSA, its severity, and clinical and metabolic parameters were analyzed using appropriate statistical tests.
Results: OSA was diagnosed in 116 patients (71.2%). Mild, moderate, and severe OSA were observed in 28.2%, 23.9%, and 19.0% of participants, respectively. Patients with OSA were older, predominantly male, and had significantly higher BMI, neck circumference, duration of diabetes, HbA1c levels, and prevalence of hypertension compared to those without OSA ($p < 0.05$). Mean HbA1c increased progressively with OSA severity, from 7.6 ± 1.2% in patients without OSA to 9.1 ± 1.1% in those with severe OSA ($p < 0.001$). Apnea–hypopnea index showed significant positive correlations with HbA1c, obesity indices, duration of diabetes, and daytime sleepiness.
Conclusion: Obstructive sleep apnea is highly prevalent among obese patients with type 2 diabetes mellitus and is strongly associated with poor glycaemic control and adverse clinical profiles. Increasing severity of OSA is linked to progressively worsening metabolic parameters. Routine screening for OSA should be considered in obese patients with T2DM to enable early identification and comprehensive management.
Keywords: Obstructive sleep apnea; Type 2 diabetes mellitus; Obesity; Apnea–hypopnea index; Glycaemic control; Polysomnography.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, leading to intermittent hypoxemia, sleep fragmentation, and marked fluctuations in intrathoracic pressure.[1] OSA is increasingly recognized as a major public health problem due to its strong association with cardiometabolic diseases, impaired quality of life, and increased morbidity and mortality.[1] The global prevalence of OSA in adults is estimated to range from 9% to 38%, with substantially higher rates reported among individuals with obesity and metabolic disorders.[2]

Type 2 diabetes mellitus (T2DM) and obesity frequently coexist and share several pathophysiological pathways with OSA, including insulin resistance, chronic low-grade inflammation, oxidative stress, and autonomic dysfunction.[3] Obesity is a well-established risk factor for OSA, with excess adipose tissue in the neck and upper airway contributing to airway collapsibility, while central obesity alters respiratory mechanics and reduces lung volumes.[4] It has been reported that nearly 60–90% of patients with T2DM are overweight or obese, placing this population at particularly high risk for undiagnosed OSA.[5]

The relationship between OSA and T2DM appears to be bidirectional. Recurrent intermittent hypoxia and sleep fragmentation in OSA promote insulin resistance, impair glucose metabolism, and exacerbate β-cell dysfunction through sympathetic overactivity, hypothalamic–pituitary–adrenal axis activation, and systemic inflammation.[6] Conversely, hyperglycemia and diabetic neuropathy may worsen upper airway neuromuscular control, thereby increasing susceptibility to OSA.[7] Epidemiological studies suggest that the prevalence of OSA among patients with T2DM ranges from 40% to 70%, with even higher rates reported in obese diabetic populations.[8]

OSA has significant implications for glycemic control and the development of diabetes-related complications [9]. Several studies have demonstrated an association between OSA severity and poor glycemic control, as reflected by higher HbA1c levels, independent of body mass index (BMI).[9] Furthermore, OSA in patients with T2DM has been linked to an increased risk of hypertension, cardiovascular disease, diabetic nephropathy, and neuropathy, thereby compounding the overall disease burden.[10] Importantly, OSA often remains underdiagnosed in diabetic patients, as symptoms such as excessive daytime sleepiness may be attributed to diabetes itself or its complications.[10]

Despite the high coexistence of obesity, T2DM, and OSA, routine screening for OSA is not consistently implemented in diabetic care settings, particularly in resource-limited environments.[11] Data on the prevalence and clinical profile of OSA among obese patients with T2DM in the Indian population remain limited, even though India bears a disproportionately high burden of both diabetes and obesity.[12] Ethnic differences in body fat distribution, craniofacial structure, and metabolic risk further underscore the need for region-specific studies.[12]

In this context, studying the burden of OSA in obese patients with T2DM is clinically relevant for early identification of high-risk individuals and timely initiation of appropriate interventions. Improved recognition and management of OSA in this population may contribute to better glycemic control, reduction in cardiometabolic complications, and overall improvement in patient outcomes. So, this study aimed to evaluate the presence and severity of obstructive sleep apnea (OSA) in obese patients with type 2 diabetes mellitus (T2DM).

MATERIALS AND METHODS

Study Design and Setting: This was a hospital-based, observational cross-sectional study conducted in the Department of Medicine in collaboration with the Department of Pulmonary/Sleep Medicine at a tertiary care teaching hospital. The study was carried out over a defined study period of 24 months, from July 2023 to June 2025. The objective was to evaluate the presence and severity of obstructive sleep apnea (OSA) in obese patients with type 2 diabetes mellitus (T2DM) using standardized clinical and polysomnographic assessments.

Study Population: Adult patients diagnosed with type 2 diabetes mellitus and attending the outpatient or inpatient services of the hospital during the study period were screened for eligibility. Patients aged ≥18 years with a body mass index (BMI) ≥30 kg/m² were considered eligible for inclusion. The diagnosis of T2DM was based on documented medical records or standard diagnostic criteria, including fasting plasma glucose, postprandial plasma glucose, or glycated hemoglobin (HbA1c) values consistent with established guidelines. Patients with previously diagnosed OSA on treatment, type 1 diabetes mellitus, pregnancy, chronic respiratory failure, congestive heart failure, cerebrovascular disease, craniofacial abnormalities affecting the upper airway, neuromuscular disorders, or those unwilling to undergo sleep study were excluded from the study.

Sample Size and Sampling Technique: The sample size was calculated based on the study by Amin et al., the prevalence of obstructive sleep apnea among obese patients with type 2 diabetes mellitus of 60%, using a confidence level of 95% and an allowable error of 7%.[12] Based on this calculation and considering a margin for possible non-response and technically inadequate sleep study recordings, the final sample size was set at 163 participants. Eligible patients were recruited using a consecutive sampling technique until the required sample size was achieved.

Clinical Evaluation and Anthropometric Measurements: All enrolled participants underwent a detailed clinical evaluation using a predesigned and pretested proforma. Demographic data, duration of diabetes, treatment details, and associated comorbidities such as hypertension and dyslipidemia were recorded. Anthropometric measurements including height, weight, waist circumference, neck circumference, and BMI were measured using standard techniques. Blood pressure was recorded in a seated position after adequate rest. Symptoms suggestive of sleep-disordered breathing, such as habitual snoring, witnessed apneas, nocturnal choking, non-refreshing sleep, and excessive daytime sleepiness, were elicited through history.

Assessment of Daytime Sleepiness and OSA Risk: Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS), which quantifies the likelihood of falling asleep in eight different situations. An ESS score >10 was considered indicative of excessive daytime sleepiness. Additionally, patients were screened for risk of OSA using a validated questionnaire such as the STOP-BANG questionnaire, which incorporates clinical and anthropometric parameters to stratify patients into low-, intermediate-, or high-risk categories for OSA.

Laboratory Investigations: Relevant laboratory parameters were obtained from recent medical records or measured during the study period. These included fasting plasma glucose, postprandial plasma glucose, glycated hemoglobin (HbA1c), lipid profile, and other routine biochemical investigations as indicated. Glycemic control was categorized based on HbA1c levels according to standard cut-off values.

Polysomnography and Sleep Study Protocol: All eligible participants underwent overnight attended polysomnography in the sleep laboratory using a standardized protocol. The polysomnographic recording included electroencephalography, electrooculography, electromyography, electrocardiography, airflow measurement using nasal pressure transducer and oronasal thermistor, thoracoabdominal respiratory effort belts, pulse oximetry for oxygen saturation, and body position monitoring. Sleep stages and respiratory events were scored manually by a trained sleep specialist in accordance with established guidelines.

Apnea was defined as complete cessation of airflow for at least 10 seconds, while hypopnea was defined as a reduction in airflow associated with oxygen desaturation and/or arousal. The apnea–hypopnea index (AHI) was calculated as the number of apneas and hypopneas per hour of sleep. OSA was diagnosed when AHI was ≥5 events/hour. Severity of OSA was classified as mild (AHI 5–14.9), moderate (AHI 15–29.9), or severe (AHI ≥30 events/hour). Oxygen desaturation index, minimum oxygen saturation, and total sleep time spent with oxygen saturation below 90% were also recorded.

Outcome Measures: The primary outcome measure was the prevalence of OSA among obese patients with T2DM. Secondary outcomes included the severity distribution of OSA and its association with clinical, anthropometric, and metabolic parameters such as BMI, neck circumference, duration of diabetes, and glycemic control.

Statistical Analysis: Data were entered into a MS Excel spreadsheet and analyzed using SPSS 20.0 statistical software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Comparisons between groups were performed using the Student's t-test or ANOVA for continuous variables and chi-square test for categorical variables. Correlation between AHI and clinical or biochemical variables was assessed using Spearman's correlation coefficient as appropriate. A p-value <0.05 was considered statistically significant.

Ethical Considerations: The study was approved by the Institutional Ethics Committee prior to initiation. Written informed consent was obtained from all participants before enrollment. Confidentiality of patient data was maintained throughout the study, and all procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

The study included 163 obese patients with type 2 diabetes mellitus, with a mean age of 53.8 ± 9.6 years, and a predominance of males (68.7%). The mean duration of diabetes was 8.4 ± 5.1 years. The mean body mass index was 32.6 ± 2.8 kg/m², with nearly two-thirds of participants classified as class I obesity (BMI 30–34.9 kg/m²). Measures of central obesity were notable, with a mean waist circumference of 104.6 ± 9.4 cm and a mean neck circumference of 40.8 ± 3.2 cm. Hypertension and dyslipidaemia were present in 48.5% and 41.1% of participants, respectively. The mean HbA1c was 8.2 ± 1.4%, with 74.2% of patients exhibiting poor glycaemic control (HbA1c ≥ 7%). Excessive daytime sleepiness, defined by an Epworth Sleepiness Scale score >10, was observed in 57.7% of participants [Table 1].

Table 1: Baseline Demographic, Clinical, Anthropometric, and Metabolic Characteristics of the Study Population (n = 163).

Variable	Frequency (%)/mean ± SD
Age (years)	53.8 ± 9.6
Age group	
≥50 years	98 (60.1)
<50 years	65 (39.9)
Gender	
Male	112 (68.7)
Female	51 (31.3)
Duration of T2DM (years)	8.4 ± 5.1
BMI (kg/m²)	32.6 ± 2.8
BMI group	
30–34.9 kg/m²	104 (63.8)
≥35 kg/m²	59 (36.2)
Neck circumference (cm)	40.8 ± 3.2
Waist circumference (cm)	104.6 ± 9.4

Comorbidity	
Hypertension	79 (48.5)
Dyslipidaemia	67 (41.1)
Current smoker	29 (17.8)
HbA1c (%)	8.2 ± 1.4
Glycaemic control	
HbA1c <7% (good control)	42 (25.8)
HbA1c ≥7% (poor control)	121 (74.2)
Epworth Sleepiness Scale score	11.4 ± 4.2
ESS group	
>10	94 (57.7)
≤10	69 (42.3)

BMI: body mass index; T2DM: type 2 diabetes mellitus; HbA1c: glycated hemoglobin; ESS: Epworth Sleepiness Scale.

Polysomnography revealed that 116 of the 163 participants (71.2%) had obstructive sleep apnea (AHI ≥ 5 events/hour). Among those diagnosed with OSA, mild OSA was observed in 28.2%, moderate OSA in 23.9%, and severe OSA in 19.0% of the total study population. Only 28.8% of participants had an AHI <5 events/hour and were classified as not having OSA. This demonstrated a high burden of moderate-to-severe OSA among obese patients with type 2 diabetes mellitus [Table 2].

Table 2: Prevalence and Severity Distribution of Obstructive Sleep Apnea Based on Apnea–Hypopnea Index.

Variables	AHI (events/hour)	Frequency	%
OSA			
No	<5	47	28.8
Yes	≥5	116	71.2
OSA Category			
Mild OSA	5–14.9	46	28.2
Moderate OSA	15–29.9	39	23.9
Severe OSA	≥30	31	19

AHI: apnea–hypopnea index (events/hour); OSA: obstructive sleep apnea.

Patients with OSA were significantly older than those without OSA (55.1 ± 9.2 vs 50.6 ± 10.1 years; p = 0.008) and had a higher proportion of males (74.1% vs 55.3%; p = 0.018). Anthropometric indices, including BMI and neck circumference, were significantly higher in the OSA group (p < 0.001 for both). Patients with OSA also had a longer duration of diabetes and significantly poorer glycaemic control, reflected by higher mean HbA1c levels (8.5 ± 1.3% vs 7.6 ± 1.2%; p < 0.001). Hypertension and excessive daytime sleepiness were significantly more common among patients with OSA compared to those without OSA [Table 3].

Table 3: Comparison of Clinical, Anthropometric, and Metabolic Parameters Between Patients With and Without Obstructive Sleep Apnea.

Variable	OSA Present (n = 116)	No OSA (n = 47)	p-value
	Frequency (%) / Mean ± SD		
Age (years)	55.1 ± 9.2	50.6 ± 10.1	0.008
Gender			
Female	30 (25.9)	21 (44.7)	0.018
Male	86 (74.1)	26 (55.3)	
BMI (kg/m²)	33.1 ± 2.7	31.4 ± 2.5	<0.001
Neck circumference (cm)	41.6 ± 3.0	38.9 ± 2.8	<0.001
Duration of T2DM (years)	9.1 ± 5.3	6.6 ± 4.4	0.004
HbA1c (%)	8.5 ± 1.3	7.6 ± 1.2	<0.001
Hypertension			
No	52 (44.8)	32 (68.1)	0.006
Yes	64 (55.2)	15 (31.9)	
ESS score	12.6 ± 4.1	8.5 ± 3.2	<0.001

BMI: body mass index; HbA1c: glycated hemoglobin; ESS: Epworth Sleepiness Scale; T2DM: type 2 diabetes mellitus.

A progressive increase in HbA1c levels was observed with increasing severity of obstructive sleep apnea. Mean HbA1c rose from 7.6 ± 1.2% in patients without OSA to 9.1 ± 1.1% in those with severe OSA. The proportion of patients with poor glycaemic control (HbA1c ≥ 7%) increased significantly across OSA severity categories, from 59.6% in patients without OSA to 90.3% among those with severe OSA. This association between OSA severity and worsening glycaemic control was statistically significant (p < 0.001) [Table 4].

Table 4: Association Between Severity of Obstructive Sleep Apnea and Glycaemic Control.

OSA Severity	HbA1c (%) Mean ± SD	HbA1c ≥7% (n=121)	HbA1c <7% (n=42)
		Frequency (%)	
No OSA (n=47)	7.6 ± 1.2	28 (59.6)	19 (40.4)
Mild OSA (n=46)	8.0 ± 1.3	32 (69.6)	14 (30.4)
Moderate OSA (n=39)	8.6 ± 1.2	33 (84.6)	6 (15.4)
Severe OSA (n=31)	9.1 ± 1.1	28 (90.3)	3 (9.7)
p-value (ANOVA / χ^2)	—	<0.001	

OSA: obstructive sleep apnea; HbA1c: glycated hemoglobin.

Apnea–hypopnea index showed a significant positive correlation with measures of obesity, including BMI ($r = 0.42$), neck circumference ($r = 0.48$), and waist circumference ($r = 0.39$) (all $p < 0.001$). AHI was also positively correlated with HbA1c levels ($r = 0.36$; $p < 0.001$) and duration of type 2 diabetes mellitus ($r = 0.31$; $p = 0.002$). A strong correlation was observed between AHI and Epworth Sleepiness Scale scores ($r = 0.46$; $p < 0.001$), indicating increasing daytime sleepiness with greater OSA severity [Table 5].

Table 5: Correlation between Apnea–Hypopnea Index and Anthropometric, Metabolic, and Clinical Parameters.

Parameter	Correlation Coefficient (r)	p-value
BMI	0.42	<0.001
Neck circumference	0.48	<0.001
Waist circumference	0.39	<0.001
HbA1c	0.36	<0.001
Duration of T2DM	0.31	0.002
Epworth Sleepiness Scale	0.46	<0.001

Correlation assessed using Spearman's rank correlation coefficient.; BMI: body mass index; HbA1c: glycated hemoglobin; T2DM: type 2 diabetes mellitus.

DISCUSSION

The present study demonstrates a high burden of obstructive sleep apnea among obese patients with type 2 diabetes mellitus, with an overall prevalence of 71.2%. This finding reinforces the growing recognition of OSA as a common yet underdiagnosed comorbidity in patients with diabetes, particularly in those with obesity. The observed prevalence is comparable to earlier studies by Saxena et al., Singh et al., and Narayan et al., reporting OSA in 60–75% of patients with T2DM, especially when polysomnography is systematically employed rather than symptom-based screening alone.[13-15] The high prevalence noted in our cohort highlights the need for routine evaluation of sleep-disordered breathing in diabetic care settings.

In this study, moderate-to-severe OSA accounted for 42.9% of the total study population, indicating a substantial proportion of patients at increased cardiometabolic risk. Similar severity distributions have been reported in hospital-based Indian studies by Dhiman et al., and Priyadarshini et al., where obesity and long-standing diabetes were key determinants of OSA severity.[16,17] The predominance of moderate and severe OSA underscores the clinical relevance of early detection, as these categories are most strongly associated with adverse cardiovascular and metabolic outcomes.[17]

Patients with OSA were significantly older and predominantly male compared to those without OSA, consistent with established epidemiological patterns in studies by Cai et al., and Kapse et al.[18,19] Anthropometric measures reflecting both generalized and central obesity, including BMI, neck circumference, and waist circumference, were significantly higher in the OSA group. Neck circumference, which showed one of the strongest associations with OSA, is a recognized surrogate marker of upper airway fat deposition and collapsibility, particularly relevant in lietrature by Eldaabousy et al., and Isono et al., where OSA occurs at comparatively lower BMI thresholds.[20,21] These findings emphasize that regional adiposity, rather than BMI alone, plays a critical role in OSA pathogenesis.[21]

A key finding of the present study is the strong association between OSA and poor glycaemic control. Patients with OSA had significantly higher mean HbA1c levels compared to those without OSA (8.5% vs 7.6%, $p < 0.001$), and HbA1c demonstrated a progressive rise with increasing OSA severity. This dose–response relationship, observed across mild, moderate, and severe OSA categories, supports earlier studies by Bhimwal et al., and Narukulla et al., that intermittent hypoxia and sleep fragmentation contribute to worsening insulin resistance and impaired glucose metabolism independent of obesity.[22,23] Reviews by Kent et al., and Valensi et al., suggest that recurrent nocturnal hypoxemia activates sympathetic nervous system activity, increases cortisol secretion, promotes systemic inflammation, and induces oxidative stress, all of which adversely affect insulin sensitivity and pancreatic β-cell function.[24,25]

The association between OSA severity and poor glycaemic control was further substantiated by the significantly higher proportion of patients with HbA1c ≥7% among those with moderate and severe OSA. This finding aligns with longitudinal studies by López-Cepero et al., and Aronsohn et al., demonstrating that untreated OSA is associated with higher HbA1c levels and greater difficulty in achieving glycaemic targets despite optimal pharmacological therapy.[26,27] The progressive deterioration of glycaemic control with increasing AHI observed in our study suggests that OSA may represent an important, modifiable contributor to poor diabetes control.[27]

Hypertension was significantly more prevalent among patients with OSA, consistent with the well-established link between sleep apnea, sympathetic overactivity, and nocturnal blood pressure dysregulation.[28] The coexistence of OSA, T2DM, and hypertension represents a particularly high-risk phenotype for cardiovascular disease, often described as part of a “metabolic–sleep apnea cluster”.[28] This clustering effect may partly explain the excess cardiovascular morbidity observed in diabetic patients with coexisting OSA.[28]

Correlation analysis revealed significant positive associations between apnea–hypopnea index and measures of obesity, glycaemic control, diabetes duration, and daytime sleepiness. The moderate correlation between AHI and HbA1c ($r = 0.36$, $p < 0.001$) observed in our study is comparable to values reported in previous reviews by Xu et al., and Heffernan et al., and supports the biological plausibility of a link between OSA severity and metabolic dysregulation.[29,30] The strong correlation between AHI and Epworth Sleepiness Scale scores further validates the clinical impact of OSA on daytime functioning, although a subset of patients with severe OSA exhibited minimal subjective sleepiness, reinforcing the limitations of symptom-based screening alone.[31,32]

From a clinical perspective, the findings of this study have important implications. Given the high prevalence of undiagnosed OSA and its clear association with poor glycaemic control and cardiometabolic comorbidities, systematic screening for OSA should be considered in obese patients with type 2 diabetes mellitus. Early identification and appropriate management of OSA may improve glycaemic control, reduce cardiovascular risk, and enhance overall quality of life.

Limitations

This study has certain limitations that should be acknowledged. First, the cross-sectional design limits the ability to establish a causal relationship between obstructive sleep apnea and poor glycaemic control in obese patients with type 2 diabetes mellitus. Second, as the study was conducted at a single tertiary care centre, the findings may not be fully generalizable to the wider community or primary care settings. Third, although overnight polysomnography was used for the diagnosis of OSA, repeat studies to account for night-to-night variability were not performed. Additionally, potential confounding factors such as physical activity levels, dietary patterns, and adherence to antidiabetic medications were not assessed in detail. Despite these limitations, the study provides robust evidence of a high burden of OSA and its clinically significant association with metabolic parameters in a high-risk population.

CONCLUSION

Obstructive sleep apnea is highly prevalent among obese patients with type 2 diabetes mellitus, with a substantial proportion exhibiting moderate to severe disease. The presence and severity of OSA are significantly associated with poorer glycaemic control, longer duration of diabetes, adverse anthropometric measures, and higher prevalence of hypertension. The observed dose–response relationship between OSA severity and HbA1c underscores the potential role of sleep-disordered breathing as an important, yet often overlooked, contributor to suboptimal diabetes control. Routine screening for OSA in obese patients with type 2 diabetes mellitus may facilitate early diagnosis and timely intervention, potentially improving metabolic outcomes and reducing cardiometabolic risk.

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