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Prevalence and Risk Factors of Obstructive Sleep Apnea Among Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study

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Abstract

Background: Obstructive sleep apnea (OSA) is a common yet underdiagnosed condition among patients with type 2 diabetes mellitus (T2DM), contributing to poor glycemic control and increased cardiovascular risk. Early identification of OSA in diabetic patients is essential for improving clinical outcomes. ***Aim:*** To assess the prevalence and risk factors of obstructive sleep apnea among patients with type 2 diabetes mellitus. ***Methods:*** This hospital-based cross-sectional study was conducted among 180 patients with T2DM attending a tertiary care center. Data regarding sociodemographic characteristics, clinical profile, and anthropometric measurements were collected. Screening for OSA was performed using the STOP-BANG questionnaire and Epworth Sleepiness Scale. Glycemic parameters including fasting blood glucose, postprandial blood glucose, and HbA1c were assessed. Statistical analysis was performed using appropriate tests, with $p < 0.05$ considered significant. ***Results:*** The prevalence of OSA was 45.6% (95% CI: 38.3%-52.9%). Among all participants, 18.9% had mild OSA, 16.1% had moderate OSA, and 10.6% had severe OSA. OSA was significantly associated with older age, male gender, higher BMI, increased neck and waist circumference, hypertension, dyslipidemia, longer duration of diabetes, and poor glycemic control ($p < 0.05$). Mean HbA1c was significantly higher among OSA patients (8.9 ± 1.3%) compared to non-OSA patients (7.8 ± 1.2%, $p < 0.001$). A progressive increase in fasting blood glucose, postprandial blood glucose, and HbA1c was observed with increasing severity of OSA. ***Conclusion:*** OSA is highly prevalent among patients with T2DM and is strongly associated with adverse metabolic and anthropometric factors. The severity of OSA correlates with worsening glycemic control, highlighting the need for routine screening and integrated management in diabetic patients.

Keywords: Obstructive Sleep Apnea; Type 2 Diabetes Mellitus; Glycemic Control.

INTRODUCTION

Obstructive Sleep Apnea (OSA) is a common yet underdiagnosed sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia, fragmented sleep, and significant cardiometabolic consequences. Globally, OSA affects nearly 9-38% of the adult population, with higher prevalence reported among individuals with obesity and metabolic disorders. Type 2 Diabetes Mellitus (T2DM), a chronic metabolic disease characterized by insulin resistance and hyperglycemia, has emerged as one of the major comorbid conditions associated with OSA. The coexistence of OSA and T2DM is clinically significant due to their bidirectional relationship, where each condition exacerbates the other, contributing to poor glycemic control and increased risk of cardiovascular complications. [1]

The pathophysiological link between OSA and T2DM involves intermittent hypoxia, oxidative stress, sympathetic activation, and systemic inflammation, which impair insulin sensitivity and glucose metabolism. Repeated apneic episodes during sleep result in activation of stress pathways and release of pro-inflammatory cytokines, thereby worsening insulin resistance. Conversely, patients with T2DM often have obesity, neuropathy, and altered upper airway muscle function, which predispose them to the development of OSA. Several studies have reported that the prevalence of OSA among patients with T2DM ranges from 40% to 80%, significantly higher than in the general population. [2]

Early identification of OSA in diabetic patients is essential, as untreated OSA has been associated with poor glycemic control, increased HbA1c levels, resistant hypertension, cardiovascular morbidity, and decreased quality of life. Screening tools such as the STOP-BANG questionnaire and Epworth Sleepiness Scale (ESS) are

widely used for identifying high-risk individuals, while polysomnography remains the gold standard for diagnosis. However, due to limited accessibility of polysomnography, especially in resource-limited settings, clinical screening assumes great importance in tertiary care settings. [3]

Various risk factors have been identified for OSA in patients with T2DM, including obesity (especially central obesity), male gender, advancing age, hypertension, dyslipidemia, and duration of diabetes. Anthropometric measures such as Body Mass Index (BMI), neck circumference, and waist-hip ratio are strong predictors of OSA severity. Despite growing awareness, OSA remains underdiagnosed in diabetic populations in India, particularly in semi-urban and rural settings, where healthcare resources are limited and awareness is low. [4]

AIM

To assess the prevalence and risk factors of obstructive sleep apnea among patients with type 2 diabetes mellitus.

OBJECTIVES

1. To determine the prevalence of obstructive sleep apnea among patients with type 2 diabetes mellitus.
2. To identify clinical and anthropometric risk factors associated with obstructive sleep apnea in these patients.
3. To evaluate the association between severity of obstructive sleep apnea and glycemic control parameters.

MATERIALS AND METHODS

Source of Data

The data were collected from patients diagnosed with Type 2 Diabetes Mellitus attending the outpatient and inpatient departments of a tertiary care hospital.

Study Design

The study was a hospital-based cross-sectional observational study.

Study Location

The study was conducted at a tertiary care teaching hospital.

Study Duration

The study was carried out over a period of 12 months.

Sample Size

A total of 180 patients with Type 2 Diabetes Mellitus were included in the study.

Inclusion Criteria

- Patients aged ≥18 years diagnosed with Type 2 Diabetes Mellitus (as per ADA criteria).
- Patients who provided informed consent.

Exclusion Criteria

- Patients with previously diagnosed obstructive sleep apnea on treatment.
- Patients with Type 1 Diabetes Mellitus.
- Patients with severe comorbid conditions (e.g., advanced cardiac, respiratory, or neurological illness).
- Pregnant women.
- Patients unwilling to participate.

Procedure and Methodology

All eligible patients were enrolled after obtaining informed written consent. A detailed clinical history was obtained, including duration of diabetes, treatment history, and associated comorbidities such as hypertension and dyslipidemia. Anthropometric measurements including height, weight, BMI, neck circumference, and waist-hip ratio were recorded using standard techniques.

Patients were screened for obstructive sleep apnea using validated questionnaires such as the STOP-BANG questionnaire and Epworth Sleepiness Scale (ESS). Based on questionnaire scores, patients were categorized into low-risk and high-risk groups for OSA.

Patients identified as high-risk were further advised for confirmatory evaluation using overnight polysomnography wherever feasible. The severity of OSA was classified based on Apnea-Hypopnea Index (AHI) into mild, moderate, and severe categories.

Laboratory investigations including fasting blood glucose, postprandial blood glucose, and HbA1c levels were performed to assess glycemic control.

Sample Processing

Blood samples were collected under aseptic conditions. Fasting venous blood samples were analyzed for glucose levels using standard biochemical methods. HbA1c estimation was performed using standardized laboratory techniques such as high-performance liquid chromatography (HPLC).

Statistical Methods

Data were entered into Microsoft Excel and analyzed using SPSS software. Descriptive statistics were expressed as mean ± standard deviation for continuous variables and as frequencies and percentages for categorical variables.

The Chi-square test was applied to assess associations between categorical variables. Independent t-test or ANOVA was used for comparison of means between groups. Pearson correlation analysis was used to assess the relationship between OSA severity and glycemic parameters. A p-value <0.05 was considered statistically significant.

Data Collection

Data were collected using a pre-designed and pre-tested structured proforma. Information regarding demographic details, clinical history, anthropometric measurements, questionnaire scores, and laboratory findings were systematically recorded for each participant.

RESULTS

Table 1: Baseline sociodemographic, clinical and anthropometric profile of study participants (N = 180)

Variable	Category / Value	n (%) / Mean ± SD	95% CI	Test of significance	p value
Age (years)	Mean ± SD	54.8 ± 9.7	53.4 - 56.2	One-sample t = 75.84	<0.001
Age group	<45 years	29 (16.1)	10.7 - 21.5	χ^2 = 41.62	<0.001
	45-54 years	58 (32.2)	25.4 - 39.0		
	55-64 years	63 (35.0)	28.0 - 42.0		
	≥65 years	30 (16.7)	11.2 - 22.1		
Sex	Male	104 (57.8)	50.6 - 65.0	χ^2 = 4.36	0.037
	Female	76 (42.2)	35.0 - 49.4		
Duration of T2DM (years)	Mean ± SD	8.9 ± 4.6	8.2 - 9.6	One-sample t = 25.94	<0.001
BMI (kg/m²)	Mean ± SD	28.7 ± 4.2	28.1 - 29.3	One-sample t = 91.64	<0.001
BMI category	Normal (<23)	21 (11.7)	7.0 - 16.4	χ^2 = 66.84	<0.001
	Overweight (23-24.9)	34 (18.9)	13.2 - 24.6		
	Obese I (25-29.9)	71 (39.4)	32.3 - 46.6		
	Obese II (≥30)	54 (30.0)	23.3 - 36.7		
Neck circumference (cm)	Mean ± SD	38.9 ± 3.7	38.4 - 39.5	One-sample t = 141.03	<0.001
Waist circumference (cm)	Mean ± SD	96.7 ± 10.8	95.1 - 98.3	One-sample t = 120.14	<0.001
Hypertension	Present	97 (53.9)	46.6 - 61.2	χ^2 = 1.09	0.296
	Absent	83 (46.1)	38.8 - 53.4		
Dyslipidemia	Present	88 (48.9)	41.6 - 56.2	χ^2 = 0.36	0.548
	Absent	92 (51.1)	43.8 - 58.4		
HbA1c (%)	Mean ± SD	8.3 ± 1.4	8.1 - 8.5	One-sample t = 79.52	<0.001

Table 1 shows the baseline sociodemographic, clinical, and anthropometric profile of the 180 study participants with type 2 diabetes mellitus. The mean age of the participants was 54.8 ± 9.7 years (95% CI: 53.4-56.2), and this was statistically significant (t = 75.84, p < 0.001). According to age-group distribution, the largest proportion of patients belonged to the 55-64 years category (63, 35.0%), followed by the 45-54 years group (58, 32.2%). Participants aged ≥65 years constituted 30 (16.7%), while those aged <45 years accounted for 29 (16.1%). This age distribution was statistically significant (χ^2 = 41.62, p < 0.001), indicating that middle-aged and elderly diabetic patients formed the major part of the study population. With regard to sex distribution, 104 (57.8%) participants were male and 76 (42.2%) were female, showing a significant male predominance (χ^2 = 4.36, p = 0.037). The mean duration of type 2 diabetes mellitus was 8.9 ± 4.6 years (95% CI: 8.2-9.6), which was also statistically significant (t = 25.94, p < 0.001). The mean body mass index (BMI) was 28.7 ± 4.2 kg/m² (95% CI: 28.1-29.3), suggesting that most patients were in the overweight to obese range. This was further supported by the BMI category distribution, where 71 (39.4%) participants were in Obese I, 54 (30.0%) in Obese II, 34 (18.9%) were overweight, and only 21 (11.7%) had normal BMI. This distribution was highly significant (χ^2 = 66.84, p < 0.001).

The mean neck circumference was 38.9 ± 3.7 cm (95% CI: 38.4-39.5) and the mean waist circumference was 96.7 ± 10.8 cm (95% CI: 95.1-98.3), both of which were highly significant ($p < 0.001$). Hypertension was present in 97 (53.9%) participants, while 83 (46.1%) did not have hypertension; however, this difference was not statistically significant ($\chi^2 = 1.09$, $p = 0.296$). Similarly, dyslipidemia was present in 88 (48.9%) and absent in 92 (51.1%), with no statistically significant difference ($\chi^2 = 0.36$, $p = 0.548$). The mean HbA1c was 8.3 ± 1.4% (95% CI: 8.1-8.5), showing poor glycemic control overall, and this was highly significant ($t = 79.52$, $p < 0.001$).

Table 2: Prevalence and severity of obstructive sleep apnea among patients with type 2 diabetes mellitus (N = 180)

Variable	Category	n (%)	95% CI	Test of significance	p value
OSA status	Present	82 (45.6)	38.3 - 52.9	$\chi^2 = 1.42$	0.233
	Absent	98 (54.4)	47.1 - 61.7		
OSA severity (among total N=180)	Mild	34 (18.9)	13.2 - 24.6	$\chi^2 = 59.87$	<0.001
	Moderate	29 (16.1)	10.7 - 21.5		
	Severe	19 (10.6)	6.1 - 15.1		
	No OSA	98 (54.4)	47.1 - 61.7		
STOP-BANG score	Mean ± SD	4.6 ± 1.9	4.3 - 4.9	One-sample t = 32.47	<0.001
Epworth Sleepiness Scale	Mean ± SD	9.8 ± 4.1	9.2 - 10.4	One-sample t = 32.09	<0.001
AHI (events/hour) among OSA cases (n=82)	Mean ± SD	19.7 ± 8.6	17.8 - 21.6	One-sample t = 20.75	<0.001

Table 2 depicts the prevalence and severity of obstructive sleep apnea among the 180 patients with type 2 diabetes mellitus. OSA was present in 82 (45.6%) participants and absent in 98 (54.4%). The estimated prevalence of OSA was therefore 45.6% with a 95% confidence interval of 38.3% to 52.9%. Although OSA was common in this diabetic population, the difference in overall OSA status distribution was not statistically significant ($\chi^2 = 1.42$, $p = 0.233$).

Regarding severity distribution among the total study population, 34 (18.9%) had mild OSA, 29 (16.1%) had moderate OSA, and 19 (10.6%) had severe OSA, while 98 (54.4%) had no OSA. This distribution of OSA severity was highly statistically significant ($\chi^2 = 59.87$, $p < 0.001$), indicating a meaningful spread across severity classes. The mean STOP-BANG score was 4.6 ± 1.9 (95% CI: 4.3-4.9), and this was statistically significant ($t = 32.47$, $p < 0.001$), suggesting that a considerable proportion of participants were at moderate to high risk for OSA on screening. The mean Epworth Sleepiness Scale score was 9.8 ± 4.1 (95% CI: 9.2-10.4), which was also statistically significant ($t = 32.09$, $p < 0.001$), reflecting substantial daytime sleepiness in the study group.

Among the 82 OSA-positive cases, the mean Apnea-Hypopnea Index (AHI) was 19.7 ± 8.6 events/hour with a 95% CI of 17.8-21.6, and this finding was highly significant ($t = 20.75$, $p < 0.001$). This mean AHI suggests that, on average, OSA-positive patients had disease in the mild-to-moderate range, though a substantial fraction also had severe OSA. Thus, Table 2 demonstrates that obstructive sleep apnea was highly prevalent among patients with type 2 diabetes mellitus, with nearly half of the study population affected and a notable proportion having moderate-to-severe disease.

Table 3 presents the association of clinical and anthropometric risk factors with obstructive sleep apnea by comparing OSA-positive patients (n = 82) with OSA-negative patients (n = 98). Patients with OSA were significantly older, with a mean age of 58.1 ± 8.7 years, compared with 52.0 ± 9.6 years in those without OSA. This difference was statistically significant ($t = 4.47$, $p < 0.001$) with a mean difference of 3.4 to 8.8 years, indicating that increasing age was an important risk factor for OSA in diabetic patients.

Male sex was significantly associated with OSA. Among OSA-positive patients, 56 (68.3%) were male compared to 48 (49.0%) in the OSA-negative group. This association was statistically significant ($\chi^2 = 6.76$, $p = 0.009$) with an odds ratio (OR) of 2.24 (95% CI: 1.21-4.16), suggesting that males had more than twice the risk of OSA compared with females. The mean duration of diabetes was significantly higher in the OSA group (10.3 ± 4.8 years) than in the non-OSA group (7.7 ± 4.1 years) ($t = 3.93$, $p < 0.001$), with a mean difference of 1.3 to 3.9 years.

Table 3: Association of clinical and anthropometric risk factors with obstructive sleep apnea (N = 180)

Risk factor	OSA Present (n=82)	OSA Absent (n=98)	Test of significance	95% CI	p value
Age (years), Mean ± SD	58.1 ± 8.7	52.0 ± 9.6	t = 4.47	Mean difference: 3.4 - 8.8	<0.001
Male sex, n (%)	56 (68.3)	48 (49.0)	χ^2 = 6.76	OR 2.24 (1.21 - 4.16)	0.009
Duration of T2DM (years), Mean ± SD	10.3 ± 4.8	7.7 ± 4.1	t = 3.93	Mean difference: 1.3 - 3.9	<0.001
BMI (kg/m²), Mean ± SD	30.9 ± 4.1	26.9 ± 3.5	t = 7.10	Mean difference: 2.9 - 5.1	<0.001
Neck circumference (cm), Mean ± SD	40.8 ± 3.3	37.4 ± 3.1	t = 7.09	Mean difference: 2.5 - 4.3	<0.001
Waist circumference (cm), Mean ± SD	102.6 ± 9.8	91.8 ± 8.7	t = 7.83	Mean difference: 8.1 - 13.5	<0.001
Hypertension present, n (%)	56 (68.3)	41 (41.8)	χ^2 = 12.58	OR 2.99 (1.61 - 5.54)	<0.001
Dyslipidemia present, n (%)	49 (59.8)	39 (39.8)	χ^2 = 7.15	OR 2.25 (1.23 - 4.11)	0.008
HbA1c (%), Mean ± SD	8.9 ± 1.3	7.8 ± 1.2	t = 5.93	Mean difference: 0.7 - 1.5	<0.001
Poor glycemic control (HbA1c ≥8%), n (%)	58 (70.7)	39 (39.8)	χ^2 = 17.08	OR 3.65 (1.92 - 6.95)	<0.001

Anthropometric parameters were also significantly higher among patients with OSA. The mean BMI was 30.9 ± 4.1 kg/m² in the OSA group versus 26.9 ± 3.5 kg/m² in the non-OSA group (t = 7.10, p < 0.001), with a mean difference of 2.9 to 5.1 kg/m². Similarly, mean neck circumference was 40.8 ± 3.3 cm in OSA patients compared to 37.4 ± 3.1 cm in non-OSA patients (t = 7.09, p < 0.001), and mean waist circumference was 102.6 ± 9.8 cm versus 91.8 ± 8.7 cm respectively (t = 7.83, p < 0.001). These findings clearly indicate that obesity and central adiposity were strongly associated with OSA.

Comorbid conditions also showed significant associations. Hypertension was present in 56 (68.3%) of OSA patients compared with 41 (41.8%) of non-OSA patients (χ^2 = 12.58, p < 0.001), with an OR of 2.99 (95% CI: 1.61-5.54). Dyslipidemia was present in 49 (59.8%) OSA cases and 39 (39.8%) non-OSA cases (χ^2 = 7.15, p = 0.008), with an OR of 2.25 (95% CI: 1.23-4.11). The mean HbA1c was significantly higher among OSA patients (8.9 ± 1.3%) than non-OSA patients (7.8 ± 1.2%) (t = 5.93, p < 0.001). Likewise, poor glycemic control (HbA1c ≥8%) was observed in 58 (70.7%) OSA patients versus 39 (39.8%) non-OSA patients, which was highly significant (χ^2 = 17.08, p < 0.001) with an OR of 3.65 (95% CI: 1.92-6.95). Overall, Table 3 shows that advancing age, male sex, longer duration of diabetes, obesity-related parameters, hypertension, dyslipidemia, and poor glycemic control were all important and statistically significant risk factors for OSA.

Table 4: Association between severity of obstructive sleep apnea and glycemic control parameters (N = 180)

Variable	No OSA (n=98)	Mild OSA (n=34)	Moderate OSA (n=29)	Severe OSA (n=19)	Test of significance	95% CI	p value
Fasting blood sugar (mg/dL), Mean ± SD	136.8 ± 24.7	148.3 ± 28.1	161.9 ± 30.8	176.4 ± 35.2	ANOVA F = 18.64	26.1 - 52.7*	<0.001
Postprandial blood sugar (mg/dL), Mean ± SD	198.5 ± 33.4	224.1 ± 36.7	242.6 ± 38.9	268.7 ± 40.1	ANOVA F = 29.83	46.8 - 93.6*	<0.001
HbA1c (%), Mean ± SD	7.8 ± 1.2	8.3 ± 1.1	8.9 ± 1.2	9.6 ± 1.3	ANOVA F = 19.27	1.1 - 2.5*	<0.001
Duration of T2DM (years), Mean ± SD	7.7 ± 4.1	8.9 ± 4.0	10.5 ± 4.4	12.2 ± 4.8	ANOVA F = 9.48	2.0 - 6.7*	

Table 4 shows the association between severity of obstructive sleep apnea and glycemic control parameters. A clear progressive worsening of glycemic indices was observed with increasing severity of OSA. The mean fasting blood sugar (FBS) increased from 136.8 ± 24.7 mg/dL in patients without OSA to 148.3 ± 28.1 mg/dL in mild OSA, 161.9 ± 30.8 mg/dL in moderate OSA, and 176.4 ± 35.2 mg/dL in severe OSA. This trend was highly significant (ANOVA F = 18.64, p < 0.001) with a 95% CI for difference between no OSA and severe OSA of 26.1-52.7 mg/dL.

A similar increasing trend was observed for postprandial blood sugar (PPBS). The mean PPBS was 198.5 ± 33.4 mg/dL in patients without OSA, rising to 224.1 ± 36.7 mg/dL in mild OSA, 242.6 ± 38.9 mg/dL in moderate OSA, and 268.7 ± 40.1

mg/dL in severe OSA. This association was highly significant (ANOVA F = 29.83, p < 0.001) with a 95% CI difference of 46.8-93.6 mg/dL between the no OSA and severe OSA groups. The mean HbA1c also progressively increased from 7.8 ± 1.2% in those without OSA to 8.3 ± 1.1%, 8.9 ± 1.2%, and 9.6 ± 1.3% in mild, moderate, and severe OSA respectively. This difference was statistically significant (ANOVA F = 19.27, p < 0.001) with a 95% CI difference of 1.1-2.5%.

In addition, the mean duration of T2DM increased with worsening OSA severity, from 7.7 ± 4.1 years in patients without OSA to 8.9 ± 4.0 years in mild OSA, 10.5 ± 4.4 years in moderate OSA, and 12.2 ± 4.8 years in severe OSA. This trend was also significant (ANOVA F = 9.48, p < 0.001) with a 95% CI difference of 2.0-6.7 years between no OSA and severe OSA. Thus, Table 4 clearly demonstrates that increasing severity of obstructive sleep apnea was significantly associated with poorer glycemic control and longer duration of diabetes, suggesting that more severe sleep-disordered breathing may contribute to worsening metabolic dysfunction in patients with type 2 diabetes mellitus.

DISCUSSION

Table 1 demonstrates that the study population with type 2 diabetes mellitus was predominantly middle-aged to elderly, with a mean age of 54.8 ± 9.7 years, and most participants clustered in the 55-64 years age group (35.0%). A modest male predominance was observed (57.8% males). The mean duration of diabetes was 8.9 ± 4.6 years, while the mean BMI was 28.7 ± 4.2 kg/m², indicating that the majority of patients were overweight or obese. In fact, only 11.7% had normal BMI, whereas 39.4% were classified as Obese I and 30.0% as Obese II. The mean neck circumference (38.9 ± 3.7 cm) and waist circumference (96.7 ± 10.8 cm) were also elevated, supporting the presence of central obesity. Hypertension and dyslipidemia were found in 53.9% and 48.9% of participants respectively, while the mean HbA1c was 8.3 ± 1.4%, reflecting suboptimal glycemic control. These findings are in agreement with earlier studies showing that patients with T2DM and suspected OSA are typically older, predominantly male, and have higher adiposity indices. Umoh et al. (2020)[1] reported that the risk of obstructive sleep apnea was considerable among patients with type 2 diabetes mellitus, especially in those with obesity and higher cardiometabolic burden. Abdissa (2020)[2] observed that increasing age, obesity, and anthropometric indices such as neck circumference were common characteristics among diabetic patients at risk for OSA. Andayeshgar et al. (2022)[3], in their systematic review and meta-analysis, also emphasized that OSA is particularly frequent among patients with T2DM who are overweight, centrally obese, and metabolically compromised.

Table 2 shows that the prevalence of obstructive sleep apnea in the present study was 45.6% (95% CI: 38.3%-52.9%), with 18.9% mild, 16.1% moderate, and 10.6% severe OSA. The mean STOP-BANG score was 4.6 ± 1.9, the mean Epworth Sleepiness Scale score was 9.8 ± 4.1, and the mean AHI among OSA-positive patients was 19.7 ± 8.6 events/hour. These findings indicate that nearly half of diabetic patients in this cohort had sleep-disordered breathing, with a substantial number having moderate-to-severe disease. This prevalence falls well within the range reported in earlier literature. Worku et al. (2023)[4] documented a high prevalence of obstructive sleep apnea risk among patients with type 2 diabetes mellitus and identified a substantial burden of disease in chronic illness clinics. Wondie et al. (2022)[5] also reported a high frequency of OSA risk among T2DM patients in Ethiopia, demonstrating that the coexistence of diabetes and OSA is a major public health concern. Tassew et al. (2024)[6], in their systematic review and meta-analysis from Africa, concluded that the pooled burden of OSA risk among patients with T2DM was high and supported routine screening in this group. Ding et al. (2022)[8] similarly reported a high prevalence of obstructive sleep apnea syndrome among hospitalized patients with T2DM in Beijing, China. The somewhat lower prevalence observed in the present study compared with some hospital-based and polysomnography-based reports may reflect differences in ethnicity, obesity profile, healthcare setting, referral patterns, and the threshold used for diagnosis. Nevertheless, the current findings strongly support the concept that OSA is very common in T2DM and warrants routine screening.

Table 3 reveals that OSA was significantly associated with older age, male sex, longer duration of diabetes, higher BMI, larger neck circumference, larger waist circumference, hypertension, dyslipidemia, higher HbA1c, and poor glycemic control. Patients with OSA were significantly older (58.1 ± 8.7 vs 52.0 ± 9.6 years; p < 0.001) and had longer duration of diabetes (10.3 ± 4.8 vs 7.7 ± 4.1 years; p < 0.001). Male sex conferred a more than twofold higher odds of OSA (OR 2.24), while hypertension (OR 2.99) and dyslipidemia (OR 2.25) were also significant risk factors. Anthropometric variables showed some of the strongest relationships, with markedly higher BMI, neck circumference, and waist circumference in OSA-positive patients. Poor glycemic control was especially important: HbA1c was higher in OSA cases (8.9 ± 1.3%) than non-OSA cases (7.8 ± 1.2%), and patients with HbA1c ≥8% had 3.65 times higher odds of OSA. These results are highly consistent with other studies. Abdissa (2020)[2] reported that older age, obesity, increased neck circumference, and longer duration of diabetes were significantly associated with high OSA risk in diabetic patients. Worku et al. (2023)[4] likewise noted that obesity-related measures, neck circumference, male sex, and hypertension were major determinant factors for OSA risk among T2DM patients. Adderley et al. (2020)[7] further demonstrated in a population-based cohort that obstructive sleep apnea in patients with T2DM was linked with a greater burden of cardiovascular and

microvascular disease, supporting the importance of associated metabolic and vascular risk factors. Ding et al. (2022)[8] also identified a high burden of OSA among hospitalized T2DM patients and highlighted obesity and related metabolic parameters as important contributors. The present study therefore supports the view that OSA in T2DM is driven by a combination of demographic, metabolic, and anthropometric factors, with central obesity playing a particularly important role.

Table 4 shows a clear dose-response relationship between OSA severity and glycemic dysfunction. Fasting blood sugar rose progressively from 136.8 ± 24.7 mg/dL in those without OSA to 176.4 ± 35.2 mg/dL in severe OSA ($p < 0.001$). A similar pattern was observed for postprandial blood sugar, which increased from 198.5 ± 33.4 mg/dL in the no-OSA group to 268.7 ± 40.1 mg/dL in the severe OSA group ($p < 0.001$). Mean HbA1c also showed a graded rise from 7.8 ± 1.2% to 9.6 ± 1.3%, while duration of T2DM increased from 7.7 ± 4.1 years in those without OSA to 12.2 ± 4.8 years in those with severe OSA. This strongly suggests that worsening sleep apnea is associated with increasingly poor glycemic control and longer-standing diabetes. These observations are supported by multiple prior studies. Singh et al. (2021)[9] concluded that obstructive sleep apnea was highly prevalent among diabetic patients and that increasing severity of OSA was associated with worsening diabetic status. Andayeshgar et al. (2022)[3], in their systematic review and meta-analysis, summarized that OSA is consistently associated with adverse metabolic parameters in patients with T2DM. Chiang et al. (2021)[10] further demonstrated that obstructive sleep apnea in patients with type 2 diabetes was associated with diabetes-related complications such as diabetic macular edema, indicating the broader systemic impact of OSA in poorly controlled diabetes. Wondie et al. (2022)[5] and Tassew et al. (2024)[6] also highlighted that poor metabolic control and longer diabetes duration were frequently associated with greater OSA risk.

CONCLUSION

The present cross-sectional study was conducted to assess the prevalence and risk factors of obstructive sleep apnea (OSA) among patients with type 2 diabetes mellitus (T2DM) and to evaluate its association with glycemic control. The findings of this study clearly demonstrate that OSA is highly prevalent among individuals with T2DM and is closely associated with multiple clinical, anthropometric, and metabolic risk factors.

In this study, the prevalence of OSA was found to be 45.6%, indicating that nearly one out of every two patients with T2DM had some degree of sleep-disordered breathing. This high prevalence highlights the substantial burden of undiagnosed OSA in diabetic populations, particularly in tertiary care settings. Furthermore, a considerable proportion of patients had moderate to severe OSA, emphasizing the clinical significance of the problem. The findings reinforce the need for routine screening for OSA in patients with T2DM, especially those presenting with risk factors such as obesity and poor glycemic control.

The study population predominantly consisted of middle-aged to elderly individuals, with a mean age of 54.8 ± 9.7 years, and a slight male predominance. Increasing age and male gender were found to be significant determinants of OSA. These findings are consistent with existing evidence suggesting that age-related anatomical and neuromuscular changes in the upper airway, along with hormonal differences, contribute to a higher risk of OSA in males and older individuals.

Anthropometric parameters emerged as some of the strongest predictors of OSA in this study. Patients with OSA had significantly higher BMI, neck circumference, and waist circumference compared to those without OSA. Central obesity, reflected by increased waist circumference and neck circumference, plays a critical role in upper airway collapsibility and reduced airway patency during sleep. The observation that the majority of participants were overweight or obese further underscores the importance of obesity as a modifiable risk factor in the development and progression of OSA.

Clinical comorbidities such as hypertension and dyslipidemia were also significantly associated with OSA. These findings highlight the clustering of cardiometabolic risk factors in patients with T2DM and OSA, suggesting a shared pathophysiological pathway involving insulin resistance, systemic inflammation, and sympathetic overactivity. The coexistence of these conditions increases the risk of adverse cardiovascular outcomes and necessitates a comprehensive approach to patient management.

A key finding of this study was the strong association between OSA and poor glycemic control. Patients with OSA had significantly higher HbA1c levels, and the prevalence of poor glycemic control (HbA1c ≥8%) was markedly higher among OSA patients. Moreover, a progressive worsening of glycemic parameters, including fasting blood glucose, postprandial blood glucose, and HbA1c, was observed with increasing severity of OSA. This dose-response relationship strongly suggests that OSA contributes to impaired glucose metabolism, possibly through mechanisms such as intermittent hypoxia, oxidative stress, and increased sympathetic activity.

The study also found that the duration of diabetes was significantly longer in patients with OSA, indicating that chronic metabolic dysregulation may predispose individuals to sleep-disordered breathing. Alternatively, untreated OSA may accelerate the progression of diabetes, further complicating disease management. This

bidirectional relationship between OSA and T2DM highlights the importance of early detection and intervention.

Screening tools such as the STOP-BANG questionnaire and Epworth Sleepiness Scale were useful in identifying individuals at high risk for OSA. Given the limited availability of polysomnography in many healthcare settings, these tools can serve as effective and practical alternatives for initial screening, particularly in resource-limited environments.

In conclusion, this study establishes that OSA is highly prevalent among patients with T2DM and is strongly associated with age, male gender, obesity, central adiposity, hypertension, dyslipidemia, longer duration of diabetes, and poor glycemic control. The severity of OSA correlates with worsening metabolic parameters, indicating a significant impact on disease progression and overall health outcomes. These findings underscore the need for routine screening for OSA in diabetic patients, early diagnosis, and integrated management strategies, including lifestyle modification, weight reduction, and appropriate therapeutic interventions such as continuous positive airway pressure (CPAP) therapy. Addressing OSA in patients with T2DM may lead to improved glycemic control, reduced cardiovascular risk, and enhanced quality of life.

LIMITATIONS OF THE STUDY

1. The study was conducted in a single tertiary care center, which may limit the generalizability of the findings to the wider population.
2. The cross-sectional study design precludes establishing a causal relationship between OSA and type 2 diabetes mellitus.
3. Diagnosis of OSA was partly based on screening questionnaires (STOP-BANG, ESS), and not all patients underwent polysomnography, which may lead to underestimation or misclassification.
4. The sample size, although adequate, was relatively limited (N = 180) for subgroup analysis.
5. Potential confounding factors such as lifestyle habits (physical activity, diet, smoking, alcohol consumption) were not extensively evaluated.
6. The study did not assess long-term outcomes or the effect of OSA treatment (e.g., CPAP) on glycemic control.
7. Selection bias may be present as participants were hospital-based patients, who may have more severe disease compared to the general population.
8. Measurement variability in anthropometric parameters and laboratory values cannot be completely excluded.

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