Section: General Medicine

Original Research Article

# SEVERITY OF OBSTRUCTIVE SLEEP APNOEA IN OBESE PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH HBA1C LEVELS

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***Received*** *: 12/05/2026*
***Received in revised form*** *: 09/07/2026*
***Accepted*** *: 23/07/2026*

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**DOI:** 10.70034/ijmedph.2026.3.225

**Source of Support:** Nil,
**Conflict of Interest:** None declared

**Int J Med Pub Health**
2026; 16 (3); 1401-1405

ABSTRACT

**Background:** Obstructive sleep apnoea (OSA) is a common but under-diagnosed complication among obese patients with type 2 diabetes mellitus (T2DM) and is linked to poor glycaemic control and higher cardiovascular risk. **Objectives:** To determine the severity of OSA in obese T2DM patients and correlate it with HbA1c, body mass index (BMI), neck circumference, duration of diabetes and pulmonary artery systolic pressure (PASP).

**Materials and Methods:** A cross-sectional observational study was conducted on 40 obese T2DM patients at SSIMS hospital davangere, over a period of 18 months. Participants underwent clinical evaluation, HbA1c estimation, overnight polysomnography and 2D-echocardiography. Correlations were assessed using Pearson's coefficient and chi-square tests, with $p<0.05$ taken as significant.

**Results:** OSA was present in 92.5% of subjects (22.5% mild, 32.5% moderate, 37.5% severe). No significant correlation was found between OSA severity and either HbA1c ($r=0.169$, $p=0.298$) or duration of diabetes ($r=0.233$, $p=0.148$). OSA severity correlated positively with neck circumference ($r=0.342$, $p=0.03$) and with PASP ($r=0.552$, $p<0.001$). Pulmonary hypertension was present in 20% of patients, all with severe OSA.

**Conclusion:** OSA is highly prevalent among obese T2DM patients. Anthropometric measurements and pulmonary haemodynamics positively co-related with OSA severity, while glycaemic control and diabetes duration were not independently associated with severity in this cohort. Therefore, routine screening for OSA in obese diabetics is recommended to enable early detection and prevent OSA related complications.

**Keywords:** Obstructive sleep apnoea; Type 2 diabetes mellitus; HbA1c; Polysomnography; Apnoea-hypopnoea index; Pulmonary artery hypertension; Body mass index.

## INTRODUCTION

Obstructive sleep apnoea (OSA) is characterised by repeated episodes of partial or complete upper-airway obstruction during sleep, accompanied by increased respiratory effort, intermittent oxygen desaturation and sleep fragmentation. It has well-established associations with hypertension, heart failure, atrial fibrillation and pulmonary hypertension.[1-4] Type 2 diabetes mellitus (T2DM) is a heterogeneous metabolic disorder driven by insulin resistance and progressive β-cell dysfunction, and remains a global epidemic, accounting for more than 90% of all diabetes cases worldwide.[6]

Obesity is the strongest independent risk factor for OSA: excess adipose tissue around the pharynx narrows the upper airway and increases its

collapsibility, while sleep fragmentation and intermittent hypoxaemia in turn worsen insulin resistance through sympathetic activation, hypothalamic-pituitary-adrenal axis stimulation and pro-inflammatory cytokine release.[7-10] A substantial proportion of patients with T2DM therefore have unrecognised OSA, and conversely T2DM is more prevalent among patients with OSA than in the general population.[15-17] Several studies suggest that early initiation of continuous positive airway pressure (CPAP) therapy in these patients improves glycaemic control and may reduce long-term cardiovascular and microvascular complications.[5,23] Glycated haemoglobin (HbA1c) reflects average glycaemic control over the preceding three months and is central to the diagnosis and monitoring of diabetes18. Neck circumference, body mass index (BMI) and pulmonary artery systolic pressure (PASP) are simple, widely available parameters that have been proposed as correlates of OSA severity.[14] However, the relationship between OSA severity and glycaemic control remains inconsistent across studies, with some reporting a positive correlation and others showing none.[29,30] Given this uncertainty and the paucity of Indian data specific to obese diabetics, the present study was undertaken to determine the severity of OSA in obese T2DM patients and to correlate it with HbA1c, BMI, neck circumference, duration of diabetes and PASP, so that at-risk patients can be identified for early CPAP initiation.

## MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of General Medicine, SSIMS Davangere, from 1st December 2023 to 30th June 2024. Using Cochran's formula, with an anticipated OSA prevalence of 73% among obese T2DM patients, 95% confidence level and 10% allowable error, the calculated sample size of 37 was rounded up to 40. Participants were recruited by purposive sampling after obtaining informed consent (in the local language) and institutional ethical committee clearance.

Patients aged over 18 years, with T2DM and BMI >25 kg/m² were included. Patients with hypothyroidism, upper-airway disorders or prior upper-airway surgery, prior OSA treatment, pregnancy, or those already on home oxygen, positive airway pressure therapy or oral appliances, were excluded. Each participant underwent detailed history-taking, clinical examination, anthropometric measurement (BMI, neck circumference), and laboratory work-up including complete haemogram, renal and liver function tests, fasting lipid profile, HbA1c (by ion-exchange high-performance liquid chromatography), electrocardiography, overnight polysomnography and 2D-echocardiography for PASP estimation.

Data were entered in Microsoft Excel and analysed using SPSS version 22 (trial version). Categorical variables are presented as frequencies and percentages, and continuous variables as mean ± standard deviation. Associations between OSA severity and HbA1c, duration of diabetes, BMI, neck circumference and PASP were assessed using the chi-square test and Pearson's correlation coefficient, with scatter plots used for graphical representation. A p-value <0.05 was considered statistically significant.

## RESULTS

Forty obese T2DM patients were studied. Most subjects (55%) were aged between 50 and 69 years, and the female-to-male ratio was 1.2:1 (22 females, 18 males). Around 62.5% had a diabetes duration of 6-15 years, and 60% had a BMI between 31 and 40 kg/m².

Baseline characteristics are summarised in Table 1

**Table 1: Baseline demographic and clinical characteristics of study population (n=40)**

| Parameter | Category | n (%) |
|---|---|---|
| Age (years) | 50 – 69 | 22 (55.0%) |
| Gender | Female / Male | 22 (55.0%) / 18 (45.0%) |
| Duration of DM (years) | 6 – 15 | 25 (62.5%) |
| BMI (kg/m²) | 31 – 40 | 24 (60.0%) |
| HbA1c (g%) | 8.0 - 10.0 | 14 (35.0%) |
| Neck circumference (cm) | 41 – 45 | 20 (50.0%) |
| PASP (mmHg) | < 25 / 25-40 / > 40 | 16 (40.0%) / 16 (40.0%) / 8 (20.0%) |

On overnight polysomnography, OSA (AHI ≥5/hour) was present in 92.5% of the study population — 22.5% had mild, 32.5% moderate and 37.5% severe OSA. [Table 2, Figure 1]

**Table 2: Severity of OSA in study population (AHI-based classification)**

| Severity | AHI (events/hour) | Number | Percentage |
|---|---|---|---|
| Normal | < 5 | 3 | 7.5% |
| Mild | 5 – 15 | 9 | 22.5% |
| Moderate | 15 – 30 | 13 | 32.5% |
| Severe | > 30 | 15 | 37.5% |

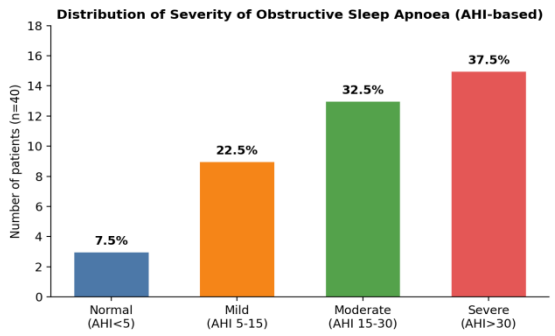


**Figure 1: Distribution of severity of obstructive sleep apnoea in the study population**

On correlation analysis, OSA severity did not correlate significantly with HbA1c (r=0.169, p=0.298) or with duration of diabetes (r=0.233, p=0.148). BMI showed a weak, non-significant positive trend (r=0.233, p=0.15). In contrast, neck circumference correlated positively with AHI (r=0.342, p=0.03), and PASP showed the strongest and most significant correlation with AHI (r=0.552, p<0.001) — all 8 patients with PASP >40 mmHg had severe OSA. [Table 3, Figure 2]

**Table 3: Correlation of AHI with glycaemic, anthropometric and haemodynamic parameters**

| Variable correlated with AHI | Correlation coefficient (r) | p-value | Significance |
|---|---|---|---|
| HbA1c (g%) | 0.169 | 0.298 | Not significant |
| Duration of diabetes (years) | 0.233 | 0.148 | Not significant |
| BMI (kg/m²) | 0.233 | 0.150 | Not significant |
| Neck circumference (cm) | 0.342 | 0.03 | Significant |
| PASP (mmHg) | 0.552 | <0.001 | Highly significant |

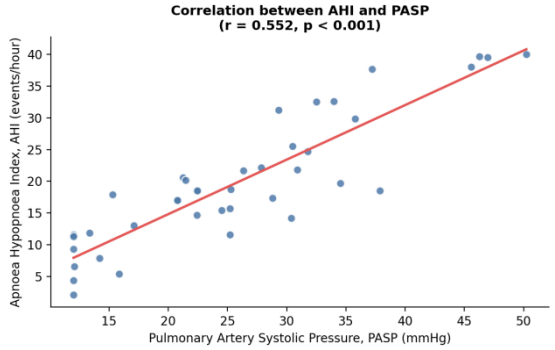


**Figure 2: Scatter plot showing correlation between**

AHI and pulmonary artery systolic pressure (PASP) Pulmonary hypertension (PASP >40 mmHg) was found in 20% (8/40) of the study population, and all of these patients had severe OSA, corresponding to 53% of all patients with severe OSA. Mean HbA1c was comparable across mild (10.25 ± 3.63%), moderate (9.35 ± 2.03%) and severe (10.61 ± 2.20%) OSA groups, without a consistent dose-response relationship.

## DISCUSSION

This study found a very high prevalence of OSA (92.5%) among obese T2DM patients, comparable to the 86% reported by Foster et al,[22] and higher than the 70-71% reported by Brooks et al,[27] and Einhorn et al,[28] reflecting the compounding effect of obesity and diabetes on upper-airway collapsibility.[11-13] The mean age (60.67 years) and female predominance (55%) in this cohort were broadly consistent with the studies of Bakker et al,[24] Nagayoshi et al,[25] and Appleton et al.[26]

Unlike Aronsohn et al,[29] who reported a graded rise in mean HbA1c with increasing OSA severity (7.3% in mild to 9.7% in severe OSA), the present study found no significant correlation between HbA1c and AHI (r=0.169, p=0.298), a finding that mirrors the report by Pillai et al,[30] who also found no consistent glycaemic gradient across OSA severity categories. This discrepancy may reflect differences in background glycaemic therapy, duration of poorly controlled diabetes, and the cross-sectional design of both studies, which limits causal inference between intermittent hypoxaemia and long-term glycaemic control. Similarly, duration of diabetes did not correlate significantly with OSA severity in this study, in contrast to Chen et al,[31] who reported a significant association, possibly reflecting differences in study population (diabetic foot ulcer patients) and comorbidity burden.

Neck circumference and PASP, however, showed statistically significant positive correlations with AHI, consistent with prior work by Ibrahim et al,[32] and Laldaya et al,[33] who similarly demonstrated that neck circumference rises progressively with OSA severity. The strong correlation between AHI and PASP (r=0.552, p<0.001), with pulmonary hypertension confined exclusively to the severe OSA subgroup, corroborates the mechanistic link between chronic intermittent hypoxia, endothelial dysfunction and pulmonary vascular remodelling described in the literature,[21] and is comparable to the prevalence of pulmonary hypertension reported by Chaouat et al,[19] (17%) and somewhat lower than that reported by Sajkov and McEvoy42 (34%). Similarly, the proportion of morbidly obese patients (BMI >40 kg/m²) with moderate-to-severe OSA in this study (73%) was comparable to figures reported by Leong et al,[34] (58%) and Daltro et al,[35] (68%).

**Table 4: Comparison of key findings with previously published studies**

| Parameter | Present study | Comparable study | Reported value |
|---|---|---|---|
| Prevalence of OSA | 92.5% | Foster et al. [22] | 86% |
| Mean HbA1c, severe OSA | 10.61% | Aronsohn et al. [29] | 9.7% |
| Mean neck circumference, severe OSA | 42.3 cm | Laldaya et al. [33] | 48.7 cm |
| Prevalence of pulmonary hypertension | 20% | Chaouat et al. [21] | 17% |

Taken together, these findings suggest that while OSA is nearly universal in obese T2DM patients, its severity in this cohort was driven more by mechanical/anthropometric factors (neck circumference, BMI) and its haemodynamic consequences (PASP) than by glycaemic control itself, which appeared to be an independent, parallel morbidity rather than a direct correlate of AHI. This has practical implications: bedside anthropometric measures such as neck circumference and BMI may be more immediately useful than HbA1c for flagging obese diabetics who warrant polysomnographic evaluation.

Limitations of this study include its single-centre, cross-sectional design, modest sample size (n=40), and referral-hospital setting, which may limit generalisability. Longitudinal studies with larger cohorts and pre-/post-CPAP glycaemic assessment are needed to clarify causal relationships and the therapeutic benefit of CPAP in this population.[5,23]

## CONCLUSION

OSA is highly prevalent (92.5%) among obese patients with type 2 diabetes mellitus, with over a third having severe disease. In this cohort, OSA severity did not correlate significantly with HbA1c or duration of diabetes, but correlated positively with neck circumference and, most strongly, with PASP, with pulmonary hypertension confined to patients with severe OSA. These findings support routine OSA screening in obese diabetic patients using simple anthropometric parameters, with polysomnography and early CPAP initiation in those at high risk, to help prevent cardiopulmonary and metabolic complications.

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