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Nocturnal Hypoxemia and Glycemic Status in Adults with Obstructive Sleep Apnea: A Retrospective Pulmonary Sleep Cohort with Positive Airway Pressure Titration Data

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Research Article

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Abstract

Background

Obstructive sleep apnea (OSA) and dysglycemia frequently coexist, but pulmonary sleep risk is not always captured by the apnea-hypopnea index (AHI) alone. Because nocturnal hypoxemia may be more closely linked to metabolic stress, we evaluated the relationship between glycemic status and oxygen desaturation burden in a real-world adult OSA cohort.

Methods

This retrospective observational study included adults evaluated in a tertiary pulmonary sleep laboratory between 2018 and 2020. Diagnostic polysomnography (PSG), capillary glucose measurements before and after PSG, and positive airway pressure (PAP) titration data when available were extracted. OSA was defined as AHI > = 5 events/h. Patients were classified as normoglycemic, prediabetic, or diabetic using American Diabetes Association glucose thresholds and documented diabetes. Between-group comparisons, paired PSG-to-titration analyses, Spearman correlations, and exploratory multivariable models were performed.

Results

Of 438 screened patients, 420 met OSA criteria and were analyzed (normoglycemia n = 193, prediabetes n = 139, diabetes n = 88). ODI increased stepwise across glycemic strata (median [IQR] 22.1 [10.2–47.2], 31.7 [15.1–67.3], and 37.9 [21.1–85.0] events/h; $p < 0.001$). AHI also differed across groups (32.5 [17.3–53.3], 39.7 [21.4–66.6], and 42.0 [23.8–79.3] events/h; $p = 0.010$). Post-PSG fasting glucose correlated with ODI (rho = 0.237; $p < 0.001$) more strongly than with AHI (rho = 0.151; $p = 0.002$). In available titration data, PAP reduced median AHI from 53.1 to 3.7 events/h (n = 165) and median ODI from 44.2 to 7.6 events/h (n = 205) (both $p < 0.001$). Residual titration AHI and ODI did not differ significantly by glycemic group. In adjusted exploratory analysis, diabetes remained associated with higher ODI after accounting for age, body mass index, and AHI.

Conclusions

In adults with OSA, worsening glycemic status was associated with greater nocturnal hypoxemia and higher AHI, with ODI showing the stronger glucose relationship. Effective PAP titration markedly reduced respiratory events and desaturation across glycemic strata. Incorporating hypoxemia metrics may improve pulmonary sleep phenotyping and cardiometabolic risk recognition in OSA.

Background

Obstructive sleep apnea (OSA) is a common pulmonary sleep disorder characterized by recurrent upper-airway obstruction during sleep, intermittent hypoxemia, sleep fragmentation, and sympathetic activation. These physiological stressors are clinically relevant beyond sleepiness because they may contribute to cardiometabolic disease, impaired glucose regulation, and type 2 diabetes mellitus [1].

The apnea-hypopnea index (AHI) remains the most widely used severity index in clinical sleep laboratories; however, AHI summarizes event frequency and does not fully capture the depth, duration, or cumulative burden of oxygen desaturation. Oxygen desaturation index (ODI) may therefore better reflect intermittent hypoxemic stress, a mechanism plausibly linked to insulin resistance and glucose dysregulation [3–5]. This distinction is particularly important in routine pulmonary sleep practice, where patients with similar AHI values may have different desaturation profiles and different metabolic risk.

Evidence from large cohorts and mechanistic studies supports a relationship between sleep-disordered breathing and dysglycemia [3–7]. Nevertheless, real-world data that jointly evaluate glycemic strata, diagnostic PSG indices, and PAP titration changes remain useful for clinical phenotyping and treatment planning. This study was designed to assess whether glycemic status is associated with nocturnal hypoxemia and OSA severity in adults referred to a pulmonary sleep laboratory, and whether PAP titration attenuates respiratory and desaturation indices across glycemic strata.

Methods

Study design and setting

This retrospective observational study used routinely collected clinical data from adults evaluated in a tertiary pulmonary sleep laboratory at Harran University between 2018 and 2020. The manuscript was prepared in accordance with the STROBE recommendations for observational research [8].

Participants

Consecutive adults with overnight diagnostic polysomnography (PSG) and capillary glucose measurements recorded before PSG and the morning after PSG were screened. Patients were eligible for the primary analysis if diagnostic AHI was > = 5 events/h. Patients with AHI < 5 events/h or missing diagnostic AHI were excluded from the primary OSA cohort. Patients with available PAP titration data were included in secondary analyses of PSG-to-titration change.

Glycemic status

Patients were categorized into operational glycemic strata using American Diabetes Association glucose thresholds and documented diabetes when available [2]. Normoglycemia was defined as fasting glucose < 100 mg/dL and postprandial glucose < 140 mg/dL. Prediabetes was defined as fasting glucose 100–125 mg/dL and/or postprandial glucose 140–199 mg/dL. Diabetes was defined as fasting glucose

> = 126 mg/dL and/or postprandial glucose > = 200 mg/dL, or documented diabetes in the medical record. Because measurements were obtained as part of routine clinical care, these strata were used for research classification rather than as new clinical diagnoses.

Polysomnography and PAP titration variables

Diagnostic PSG-derived variables included AHI (events/h), ODI (events/h), REM sleep duration (minutes), and REM sleep percentage of total sleep time. ODI was defined as the number of oxygen desaturation events per hour of sleep. For patients who underwent non-invasive PAP titration, residual AHI, residual ODI, and REM sleep duration during titration were extracted. Anthropometric variables extracted from the clinical record included age, body mass index (BMI), waist circumference, and neck circumference.

Outcomes

The primary outcome was diagnostic ODI across glycemic strata. Secondary outcomes included diagnostic AHI, REM sleep indices, correlations between capillary glucose and PSG/PAP variables, and within-patient change in AHI and ODI from diagnostic PSG to PAP titration.

Statistical analysis

Continuous variables are summarized as mean ± standard deviation or median (interquartile range [IQR]), as appropriate; categorical variables are summarized as counts and percentages. Between-group comparisons across glycemic strata used Kruskal-Wallis tests, followed by pairwise non-parametric comparisons with correction for multiple testing where applicable. Within-patient PSG-to-titration changes were assessed using Wilcoxon signed-rank tests. Spearman rank correlations were used to evaluate associations between glucose measurements and sleep indices. Exploratory multivariable linear regression models were fitted for log-transformed ODI (log[ODI + 1]) to assess the independent association of glycemic status with desaturation burden after adjustment for age, BMI, and log-transformed AHI (log[AHI + 1]). Two-sided $p < 0.05$ was considered statistically significant.

Ethics

Ethical approval

was obtained from the Harran University Faculty of Medicine Ethics Committee (Decision No. 18/12/31; meeting date: 10 December 2018). The study was conducted in accordance with the Declaration of Helsinki. The need for informed consent was waived by the ethics committee because the study was a retrospective analysis of de-identified routine clinical records.

Results

Study sample

Among 438 screened patients with available capillary glucose measurements around diagnostic PSG, 420 met OSA criteria (diagnostic AHI > = 5 events/h) and comprised the primary analytic cohort; 18 were excluded due to AHI < 5 events/h or missing AHI. Glycemic status distribution was: normoglycemia (n = 193), prediabetes (n = 139), and diabetes (n = 88). Participants with diabetes were older and had higher BMI and waist circumference than normoglycemic participants (Table 1).

Table 1
Participant characteristics and capillary glucose measurements by glycemic status (OSA cohort).

Variable	Normoglycemia	Prediabetes	Diabetes	P value
Age, years	47.2 ± 12.5	54.1 ± 11.6	57.0 ± 9.6	< 0.001
Body mass index, kg/m²	32.0 ± 6.6	35.1 ± 8.9	37.1 ± 6.3	< 0.001
Neck circumference, cm	41.1 ± 3.5	42.1 ± 8.3	40.6 ± 3.4	0.343
Waist circumference, cm	110.6 ± 14.0	115.9 ± 15.7	123.7 ± 12.2	< 0.001
Capillary glucose before PSG (postprandial), mg/dL	110.1 ± 13.8	132.0 ± 23.8	208.5 ± 77.6	< 0.001
Capillary glucose after PSG (fasting), mg/dL	91.7 ± 5.2	107.4 ± 8.1	152.5 ± 43.9	< 0.001
Capillary glucose before PAP titration (postprandial), mg/dL	114.0 ± 21.2	130.2 ± 25.9	195.4 ± 79.1	< 0.001
Capillary glucose after PAP titration (fasting), mg/dL	98.5 ± 13.5	109.3 ± 21.0	146.0 ± 43.3	< 0.001

Values are mean ± SD unless otherwise stated. P values are from Kruskal-Wallis tests. OSA=obstructive sleep apnea; PAP=positive airway pressure; PSG=polysomnography.

Diagnostic PSG findings by glycemic status

Diagnostic AHI differed across glycemic groups (median [IQR] 32.5 [17.3–53.3], 39.7 [21.4–66.6], and 42.0 [23.8–79.3] events/h for normoglycemia, prediabetes, and diabetes, respectively; p = 0.010). The glycemic gradient was stronger for ODI, which increased from 22.1 (10.2–47.2) to 31.7 (15.1–67.3) and 37.9 (21.1–85.0) events/h across the same groups (p < 0.001) (Table 2). REM sleep duration was lower in the diabetes group, whereas REM percentage did not reach statistical significance.

Table 2
Diagnostic polysomnography indices by glycemic status (OSA cohort).

Variable	Normoglycemia	Prediabetes	Diabetes	P value
Apnea-hypopnea index (AHI), events/h	32.5 (17.3–53.3)	39.7 (21.4–66.6)	42.0 (23.8–79.3)	0.010
Oxygen desaturation index (ODI), events/h	22.1 (10.2–47.2)	31.7 (15.1–67.3)	37.9 (21.1–85.0)	< 0.001
REM sleep duration, min	48.0 (28.5–79.0)	51.8 (29.0–78.0)	38.0 (22.4–66.8)	0.040
REM sleep, % of total sleep time	12.7 (6.8–18.5)	13.8 (7.3–19.0)	10.3 (5.9–15.5)	0.075

Values are median (interquartile range). P values are from Kruskal-Wallis tests. AHI=apnea-hypopnea index; ODI=oxygen desaturation index; REM=rapid eye movement.

Correlations between glucose and sleep indices

Capillary glucose values correlated positively with diagnostic PSG indices. Post-PSG fasting glucose correlated with ODI (rho = 0.237; p < 0.001) more strongly than with AHI (rho = 0.151; p = 0.002). Pre-PSG postprandial glucose also correlated with PSG ODI (rho = 0.162; p < 0.001) and PSG AHI (rho = 0.111; p = 0.023) (Table 4).

Table 4
Significant Spearman correlations between capillary glucose and polysomnography/positive airway pressure indices.

Context	Glucose variable	Sleep variable	n	Spearman rho	P value
Diagnostic PSG	Pre-PSG postprandial glucose	PSG AHI	420	0.111	0.023
Diagnostic PSG	Pre-PSG postprandial glucose	PSG ODI	420	0.162	< 0.001
Diagnostic PSG	Post-PSG fasting glucose	PSG AHI	420	0.151	0.002
Diagnostic PSG	Post-PSG fasting glucose	PSG ODI	420	0.237	< 0.001
PAP titration	Post-titration fasting glucose	Titration ODI	204	0.165	0.019

Only correlations with p < 0.05 are shown. AHI=apnea-hypopnea index; ODI=oxygen desaturation index; PAP=positive airway pressure; PSG=polysomnography.

PAP titration findings

In patients with available titration data (AHI n = 165; ODI n = 205), PAP titration markedly reduced event and desaturation burden. Median AHI decreased from 53.1 (34.2–72.3) to 3.7 (2.2–6.2) events/h ($p < 0.001$), and median ODI decreased from 44.2 (21.6–77.8) to 7.6 (3.4–14.0) events/h ($p < 0.001$). Within-group reductions were significant for normoglycemia, prediabetes, and diabetes (Table 3). Residual titration AHI and ODI did not differ significantly across glycemic strata.

Table 3
Paired changes from diagnostic polysomnography to positive airway pressure titration (subset with available data).

Outcome	Group	n	Diagnostic PSG	PAP titration	P value
AHI (events/h)	Overall	165	53.1 (34.2–72.3)	3.7 (2.2–6.2)	< 0.001
AHI (events/h)	Normoglycemia	67	42.4 (32.6–66.3)	3.9 (2.5–6.2)	< 0.001
AHI (events/h)	Prediabetes	62	54.6 (35.0-68.4)	3.8 (2.3–6.3)	< 0.001
AHI (events/h)	Diabetes	36	66.8 (40.2–86.9)	3.4 (2.0-5.3)	< 0.001
ODI (events/h)	Overall	205	44.2 (21.6–77.8)	7.6 (3.4–14.0)	< 0.001
ODI (events/h)	Normoglycemia	82	39.5 (19.9–74.9)	5.9 (3.0-11.9)	< 0.001
ODI (events/h)	Prediabetes	79	42.6 (20.6–70.5)	8.0 (3.8–14.9)	< 0.001
ODI (events/h)	Diabetes	44	65.7 (35.5-100.2)	9.8 (4.5–17.4)	< 0.001
REM duration (min)	Overall	205	43.5 (23.5–75.0)	72.0 (51.0-98.5)	< 0.001
REM duration (min)	Normoglycemia	82	43.2 (23.0-75.5)	71.5 (45.4–100.0)	< 0.001
REM duration (min)	Prediabetes	79	51.5 (26.8–78.0)	71.5 (54.5–93.5)	< 0.001
REM duration (min)	Diabetes	44	38.0 (21.2–65.6)	78.0 (51.4-105.5)	< 0.001

Values are median (interquartile range). P values are from Wilcoxon signed-rank tests. AHI=apnea-hypopnea index; ODI=oxygen desaturation index; PAP=positive airway pressure; PSG=polysomnography; REM=rapid eye movement.

Exploratory adjusted analysis

In an exploratory model adjusted for age, BMI, and diagnostic AHI (log-transformed), diabetes remained associated with higher ODI compared with normoglycemia (exp(beta) = 1.16 for ODI + 1; 95% CI 1.02–1.31; $p = 0.025$), supporting the presence of disproportionate desaturation burden in patients with diabetic-range glycemia.

Discussion

This retrospective pulmonary sleep cohort demonstrates that worsening glycemic status is associated with greater nocturnal hypoxemia in adults with OSA. Although AHI was higher in patients with prediabetes and diabetes, the gradient was more pronounced for ODI, and fasting glucose showed a stronger correlation with ODI than with AHI. These findings support the clinical value of looking beyond event frequency alone when assessing cardiometabolic risk in OSA.

The observed association is biologically plausible. Intermittent hypoxemia can activate sympathetic pathways, oxidative stress, systemic inflammation, and neuroendocrine responses, all of which may contribute to impaired insulin sensitivity and glucose dysregulation [1, 5]. Cohort data also show a high prevalence of diabetes in sleep-disordered breathing populations and associations between sleep disturbances and abnormal glucose metabolism [3, 4]. In this context, ODI may function as a clinically accessible marker of hypoxemic stress in pulmonary sleep practice.

An important strength of this study is the inclusion of PAP titration data. PAP titration substantially reduced AHI and ODI across glycemic strata, and residual between-group differences were largely attenuated. These findings cannot establish long-term metabolic benefit because follow-up adherence and longitudinal glycemic outcomes were not available. However, they suggest that effective titration can acutely control the respiratory and desaturation burden that may contribute to cardiometabolic stress.

The study also has implications for the interpretation of single-center real-world data. The cohort reflects routine clinical practice in an underrepresented regional pulmonary sleep population, with standardized PSG and PAP titration variables and simultaneous glucose measurements. Rather than relying solely on AHI, incorporating ODI and glycemic risk strata may help identify patients who require more integrated pulmonary, sleep, and metabolic management.

Several limitations should be considered. The retrospective design precludes causal inference. Glycemic status was based on capillary glucose measurements and medical record documentation rather than standardized laboratory plasma glucose, HbA1c, or oral glucose tolerance testing. Medication use, OSA symptoms, comorbidity burden, inflammatory markers, and long-term PAP adherence were incompletely available. Titration data were available for a subset of patients, introducing possible selection bias. Prospective studies using standardized metabolic phenotyping and longitudinal PAP adherence data are needed to validate these findings.

Conclusions

In adults with OSA, worsening glycemic status was associated with greater nocturnal hypoxemia and higher AHI on diagnostic PSG, with ODI showing the stronger relationship to glucose. PAP titration substantially reduced respiratory event and desaturation burden across glycemic strata. These results support the use of hypoxemia metrics such as ODI to complement AHI when evaluating pulmonary sleep patients with cardiometabolic risk.

Abbreviations

AHI
apnea-hypopnea index
BMI
body mass index
IQR
interquartile range
ODI
oxygen desaturation index
OSA
obstructive sleep apnea
PAP
positive airway pressure
PSG
polysomnography
REM
rapid eye movement
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the Harran University Faculty of Medicine Ethics Committee (Decision No. 18/12/31; meeting date: 10 December 2018). The need for informed consent was waived by the ethics committee because this was a retrospective analysis of de-identified routine clinical records.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Use of AI-assisted language tools

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Author Contribution

Z.S. admitted patients to the sleep clinic.Z.S. collected sleep-related data.F.S. performed statistical analysis.Z.S. and F.S. evaluated the data.Z.S. and F.S. wrote the main article text.All authors reviewed the article.

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Availability of data and materials

The datasets generated and/or analysed during the current study are not publicly available because they contain clinical record data subject to institutional and privacy restrictions, but de-identified data may be available from the corresponding author on reasonable request and with appropriate institutional approvals.

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