

Sleep Quality in Patients with Type 2 Diabetes Mellitus and Its Impact on Quality of Life and Glycemic Control: A Cross-sectional Study



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ABSTRACT

Background: Sleep quality is an underrecognized determinant of metabolic control and quality of life (QoL) in patients with type 2 diabetes mellitus (T2DM). Emerging evidence suggests a tridirectional relationship between glycemic control, sleep quality, and QoL.

Objectives: To assess the prevalence of poor sleep quality in patients with T2DM and its association with glycated hemoglobin (HbA1c) and QoL, assessed using the 36-Item Short Form Health Survey (SF-36), while identifying key predictors of sleep disturbances.

Materials and methods: This cross-sectional study was conducted over 3 months at SSG Hospital, Vadodara. A total of 375 adult patients with T2DM (duration ≥ 1 year) were assessed using the Pittsburgh Sleep Quality Index (PSQI), SF-36 questionnaire, and recent HbA1c levels. Exclusion criteria included major psychiatric illness, use of sleep medication, and other chronic comorbidities affecting sleep. Statistical analysis included correlation, subgroup, and multiple regression analyses.

Results: The mean age of the participants was 59.4 years (± 12.1), with a median age of 60 and a range of 24–95 years. The average HbA1c was 8.4% (± 1.46), indicating suboptimal glycemic control. Poor sleep quality was observed, with a mean PSQI score of 12.95 (± 5.36), while QoL, measured using SF-36, had a mean score of 42.52 (± 27.9). The average duration of diabetes was 9.1 years (± 6.4).

Conclusion: Poor sleep quality is highly prevalent among patients with T2DM and significantly impacts both glycemic control and QoL. Neuropathy emerged as the most influential factor. Incorporating routine sleep assessment and targeted interventions into diabetes management could improve holistic outcomes.

Keywords: Diabetic neuropathy, Glycemic control, Hemoglobin A, glycosylated, Pittsburgh Sleep Quality Index, Quality of life, Sleep disorders, Type 2 diabetes mellitus.

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INTRODUCTION

Sleep does much more than simply recharge our bodies; it is essential for overall health. Research indicates that insufficient sleep raises the risk of diminished life quality by about one-third and boosts the chance of developing long-term illnesses by nearly half.^{1,2} Those who get under 6 hours of sleep each night consistently experience lower satisfaction with life, greater emotional turmoil, and difficulty performing everyday tasks.³ Chronic lack of sleep has been associated with a higher likelihood of depression, anxiety, and weakened social connections, key aspects of overall well-being.⁴ Rather than a luxury, sleep is essential to our emotional state, cognitive performance, and daily functioning. Although often neglected, sleep is now recognized as a vital factor in managing type 2 diabetes mellitus (T2DM). It is far from passive downtime; sleep actively shapes metabolic health by

influencing insulin sensitivity, blood sugar control, and hormonal regulation. Mounting research indicates that poor sleep quality does not just accompany chronic illness; it helps drive its advancement. Indeed, sleep disturbances are exceedingly common among individuals with T2DM: one study in Jordan found that 81% of patients scored 8 or higher on the Pittsburgh Sleep Quality Index (PSQI), signifying significant sleep impairment.⁵ Notably, this rate is markedly higher than those observed in other groups, for example, 34.8% among Asian patients⁶ and between 49% and 71% in Korean and Indian cohorts,^{7,8} highlighting the influence of both cultural and clinical factors. The contributors to this elevated risk are diverse: female sex, cigarette smoking, unemployment, and suboptimal glycated hemoglobin (HbA1c) $\geq 7\%$ each independently correlate with impaired sleep.⁵ The impact is profound: disrupted or inadequate sleep is associated with greater insulin resistance, higher cortisol

secretion, and amplified inflammatory responses, physiological shifts that exacerbate diabetic complications.⁹ Yet, sleep is still largely overlooked in standard diabetes management. This oversight fuels a self-reinforcing cycle in which worsening diabetes disrupts sleep, poor sleep further undermines metabolic control, and together they erode patients' overall well-being.

This article aims to explore the tridirectional relationship between sleep quality, T2DM, and quality of life (QoL). By examining how each of these elements influences and amplifies the others, this article underscores the urgency of integrating sleep and quality-of-life assessments into routine diabetes management. It also highlights the potential of sleep optimization as a nonpharmacological, cost-effective strategy to enhance both metabolic control and holistic well-being in patients with T2DM.

MATERIALS AND METHODS

Objectives

- To assess the prevalence of sleep disturbances in patients with T2DM.
- To evaluate the impact of sleep quality on glycemic control, as measured by HbA1c levels.
- To investigate the association between sleep quality and QoL in patients with T2DM.
- To identify potential risk factors contributing to poor sleep quality in patients with T2DM.

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Aim

This cross-sectional study aims to evaluate the relationship between sleep quality and its impact on QoL and glycemic control in patients with T2DM.

Study Design

This study employed a cross-sectional design, using validated questionnaires to assess sleep quality and glycemic control in patients with T2DM.

Setting and Participants

Location

SSG Hospital, Vadodara.

Duration

3 months

Inclusion Criteria

Patients aged 18 years or older who had been diagnosed with T2DM for at least 1 year.

Exclusion Criteria

Patients with acute medical conditions, severe psychiatric disorders, or current use of sleep medication, and patients with other chronic conditions, such as hypertension, hyperlipidemia, rheumatoid arthritis (RA), or osteoarthritis (OA), that affect sleep or may cause sleep disturbances.

Variables

- **Primary outcome:** Sleep quality, assessed using the PSQI, and glycemic control.
- **Secondary outcomes:** QoL, assessed using the 36-Item Short Form Health Survey (SF-36) questionnaire; demographic and clinical variables, including age, sex, and duration of T2DM; and correlations among the variables.

Data Sources/Measurements

- **Data collection:** Data were collected through face-to-face interviews and self-administered questionnaires.
- **Sleep quality:** Measured using the PSQI, which provides a global score ranging from 0 to 21, with higher scores indicating poorer sleep quality.

- **Quality of life:** Assessed using the SF-36, which measures physical and mental health on a scale from 0 to 100, with higher scores indicating better QoL.
- **Glycemic control:** Assessed using the patient's clinical laboratory report for HbA1c.

Bias

Selection bias was minimized by including all eligible patients during the study period. Information bias was reduced by using standardized and validated questionnaires.

Study Size

The sample size was calculated based on the expected prevalence of poor sleep quality in patients with T2DM, with a confidence level of 95% and a margin of error of 5%. An estimated 320 patients were required. OpenEpi software was used.

Statistical Methods

- **Descriptive statistics:** Used to summarize demographic and clinical characteristics.
- **Subgroup analysis.**
- **Correlation analysis.**
- **Comparative analysis:** *t*-tests or analysis of variance (ANOVA) for continuous variables and chi-square tests for categorical variables were used to compare sleep quality and QoL between groups.
- **Regression analysis:** Multiple regression was performed to identify factors associated with poor sleep quality and its impact on QoL.
- **Significance level:** $p < 0.05$.

Ethical Considerations

- **Approval:** The study was approved by the Institutional Review Board (IRB) of SSG Hospital.
- **Informed consent:** Written informed consent was obtained from all participants.
- **Confidentiality:** All data were anonymized to protect patient confidentiality.

Results and Reporting

- **Preliminary analysis:** Initial analysis focused on the prevalence of poor sleep quality in the study population.

- **Secondary analysis:** Further analysis examined the relationship among sleep quality, T2DM, and QoL, as well as associated factors.
- **Reporting:** Findings were reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines, ensuring transparency and comprehensiveness in the reporting of cross-sectional studies.

RESULTS

In this study, the mean age of the participants was 59.4 years (± 12.1), with a median age of 60 and a range of 24–95 years. The average HbA1c was 8.4% (± 1.46), indicating suboptimal glycemic control. Poor sleep quality was observed, with a mean PSQI score of 12.95 (± 5.36), while QoL, measured using SF-36, had a mean score of 42.52 (± 27.9). The average duration of diabetes was 9.1 years (± 6.4) (Table 1). Among the participants, 54% were male, and 46% were female, with complications such as retinopathy (23%), nephropathy (28%), and neuropathy (43%) being prevalent (Table 2).

The frequency distribution analysis of sleep disturbances reveals notable patterns. Among the most common issues are bathroom visits (Table 3), with 70% of participants reporting occurrences at least once a week. Similarly, waking up at night affects 60% of individuals weekly or more frequently. By contrast, breathing discomfort is among the least frequent problems, with only 25% of participants experiencing it at least once per week.

Patients with neuropathy had higher PSQI scores (14.2 ± 4.1 vs 10.1 ± 3.8 ; $p < 0.001$) and lower SF-36 scores compared to patients without neuropathy, which makes neuropathy a major cause for disturbance in sleep quality and QoL (38.4 ± 20.5 vs 57.2 ± 25.6 ; $p < 0.01$). Among those with neuropathy, the most severe sleep disturbances were bathroom visits (mean score: 2.5 ± 0.7), followed by trouble sleeping (mean score: 2.3 ± 0.9) and waking up at night (mean score: 2.2 ± 0.8). This indicates that certain issues disproportionately affect patients

Table 1: Descriptive statistics

Variable	Mean $\pm$ SD	Median	Range
Age	59.8 $\pm$ 12.1	60	24–95
HbA1c	8.4 $\pm$ 1.8	8.1	6.1–13.2
PSQI	12.6 $\pm$ 4.9	12	2–24
SF-36	46.2 $\pm$ 25.7	42.5	0–100
DM Duration	8.1 $\pm$ 6.3	7	1–35

DM, diabetes mellitus; HbA1c, glycated hemoglobin; PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; SF-36, 36-Item Short Form Health Survey

Table 2: Categorical variables

Category	Variable	Percentage
Sex	Male	54
	Female	46
Complications	Retinopathy	23
	Nephropathy	28
	Neuropathy	43

with neuropathy, contributing to their poor sleep quality overall. Among participants with neuropathy, 78% reported experiencing trouble sleeping three or more times a week, compared to only 15% of those without neuropathy. Additionally, neuropathy patients were more likely to face this issue once or twice a week (53%) compared to non-neuropathy individuals (42%). Those who did not experience trouble

sleeping in the past month were predominantly non-neuropathy individuals (45%), whereas only 12 neuropathy patients reported experiencing trouble sleeping. The chi-square test ($\chi^2 = 68.3$, $p < 0.001$) confirms a strong association between neuropathy and trouble sleeping frequency.

The data also reveal a clear relationship between pain affecting sleep and neuropathy

(Table 1 and Fig. 1). Among individuals with neuropathy, 57% reported experiencing pain during sleep three or more times a week, compared with only 13% of those without neuropathy. This indicates that pain during sleep is more prevalent in patients with neuropathy at higher frequencies. However, at lower frequencies, such as “less than once a week,” the prevalence is similar between patients with neuropathy (40%) and individuals without neuropathy (40%). Patients with neuropathy had worse sleep quality ($p < 0.001$) and lower QoL ($p < 0.01$) (Fig. 2 and Table 4).

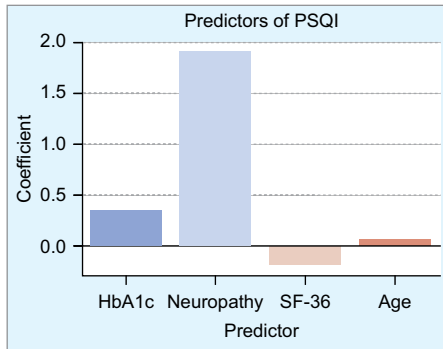


Fig. 1: Sleep disturbance vs neuropathy prevalence

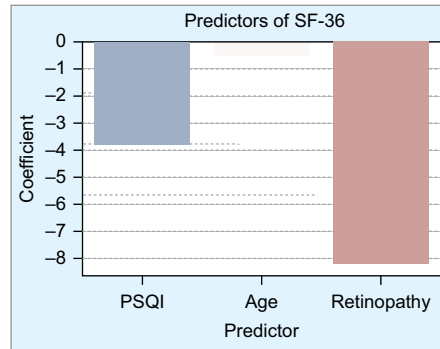


Fig. 2: Correlation of patients with and without neuropathy vs PSQI and SF-36

Table 4: Subgroup analysis by complications

Complication	PSQI (mean $\pm$ SD)	SF-36 (mean $\pm$ SD)
Neuropathy	14.2 $\pm$ 4.1	38.4 $\pm$ 20.5
No neuropathy	10.1 $\pm$ 3.8	57.2 $\pm$ 25.6

PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; SF-36, 36-Item Short Form Health Survey

Table 3: Frequency distribution and neuropathy prevalence by sleep disturbance

Sleep issue	Frequency	Neuropathy (Yes)	Neuropathy (No)	Neuropathy prevalence	Chi-square (χ^2)	p-value	Significance
Trouble sleeping	Not during past month	12	45	21%	68.3	<0.001	Strong correlation
	Less than once a week	25	78	24%			
	Once or twice a week	48	42	53%			
	Three or more times a week	53	15	78%			
Waking up at night	Not during past month	18	30	38%	14.2	0.003	Moderate correlation
	Less than once a week	32	48	40%			
	Once or twice a week	55	57	49%			
	Three or more times a week	33	25	57%			
Bathroom visits	Not during past month	8	24	25%	32.6	<0.001	Strong correlation
	Less than once a week	22	42	34%			
	Once or twice a week	65	63	51%			
	Three or more times a week	43	21	67%			
Breathing discomfort	Not during past month	62	82	43%	1.1	0.78	No significant association
	Less than once a week	45	51	47%			
	Once or twice a week	23	25	48%			
	Three or more times a week	8	12	40%			
Coughing/snoring	Not during past month	30	40	43%	5.8	0.12	Weak association
	Less than once a week	38	52	42%			
	Once or twice a week	45	51	47%			
	Three or more times a week	25	17	60%			
Bad dreams	Not during past month	52	70	43%	4.3	0.23	No significant association
	Less than once a week	35	45	44%			
	Once or twice a week	38	32	54%			
	Three or more times a week	13	13	50%			
Pain during sleep	Not during past month	41	55	43%	3.6	0.31	Weak association
	Less than once a week	45	67	40%			
	Once or twice a week	35	45	44%			
	Three or more times a week	17	13	57%			

Older adults (>60 years) exhibited worse sleep quality (PSQI: 14.5 vs 10.1, $p < 0.001$). Longer diabetes duration (>10 years) was associated with significantly poorer sleep quality (PSQI: 15.1 $\pm$ 3.9 vs 10.4 $\pm$ 4.2; $p < 0.001$), suggesting that as diabetes persists over time, its progressive nature exacerbates health issues, including sleep disturbances.

A significant difference associated with sex was reported. Females reported worse sleep quality (PSQI: 13.7 $\pm$ 5.2 vs 11.8 $\pm$ 4.5; $p = 0.02$) and lower QoL (SF-36: 42.1 $\pm$ 22.3 vs 48.5 $\pm$ 24.1; $p = 0.01$) compared with males, although no significant difference in glycemic control (HbA1c: 8.6% in females vs 8.4% in males; $p = 0.45$) was observed (Table 5).

Other complications, such as retinopathy, were associated with older age (64.2 $\pm$ 11.3 vs 56.8 $\pm$ 12.1 years; $p < 0.001$) and poorer glycemic control (HbA1c: 9.4 $\pm$ 1.9% vs 8.1 $\pm$

1.5%; $p < 0.001$), while nephropathy correlated with older age (61.5 $\pm$ 10.8 vs 57.3 $\pm$ 12.4 years; $p = 0.003$) and moderate glycemic dysfunction (HbA1c: 8.9 $\pm$ 1.7% vs 8.3 $\pm$ 1.6%; $p = 0.02$) (Table 6).

Multiple complications (≥ 2) were linked to significantly poorer outcomes, including higher PSQI (16.1 vs 10.3; $p < 0.001$) and lower SF-36 (28.7 vs 58.9; $p < 0.001$).

Correlation analysis revealed a weak positive correlation between HbA1c and PSQI ($r = 0.29$, $p < 0.001$), suggesting that

poor glycemic control is associated with worse sleep quality, and a strong negative correlation between HbA1c and SF-36 ($r = -0.41$, $p < 0.001$) (Table 7 and Figs 3 to 5).

Correlation analysis comparing the PSQI and SF-36 scores ($r = -0.63$, $p < 0.001$) indicated a significant association between poorer sleep quality and reduced QoL.

Regression analysis identified HbA1c ($\beta = 0.35$, $p < 0.001$), neuropathy ($\beta = 1.9$, $p < 0.001$), SF-36 ($\beta = -0.18$, $p < 0.001$), and age ($\beta = 0.06$, $p = 0.03$) as significant predictors of PSQI, with an R^2

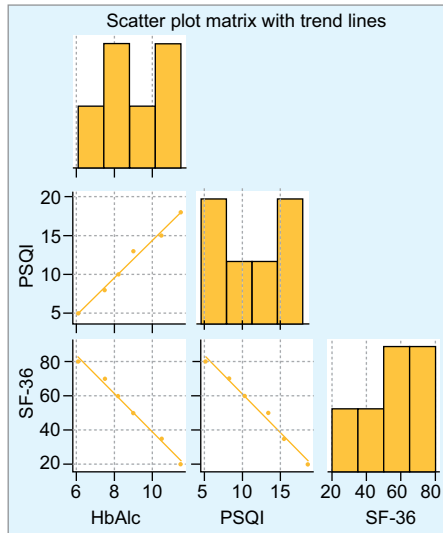


Fig. 3: Correlation plots: PSQI vs SF-36; HbA1c vs PSQI; HbA1c vs SF-36

Table 5: Subgroup analysis by sex

Sex	Mean PSQI $\pm$ SD	Mean SF-36 $\pm$ SD
Male	11.8 $\pm$ 4.5	48.5 $\pm$ 24.1
Female	13.7 $\pm$ 5.2	42.1 $\pm$ 22.3

PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; SF-36, 36-Item Short Form Health Survey

Table 6: Complications and glycemic control

Complication	Prevalence	Mean HbA1c $\pm$ SD	Mean age $\pm$ SD
Retinopathy	23%	9.4 $\pm$ 1.9	64.2 $\pm$ 11.3
Nephropathy	28%	8.9 $\pm$ 1.7	61.5 $\pm$ 10.8
Neuropathy	43%	9.2 $\pm$ 1.8	63.7 $\pm$ 10.5
Retinopathy + neuropathy	18%	10.3 $\pm$ 2.1	68.2 $\pm$ 8.9
Retinopathy + nephropathy	12%	10.1 $\pm$ 2.0	67.5 $\pm$ 9.8
Nephropathy + neuropathy	22%	9.8 $\pm$ 1.9	65.4 $\pm$ 10.1
All three complications	8%	11.5 $\pm$ 2.3	70.1 $\pm$ 7.5

HbA1c, glycated hemoglobin; SD, standard deviation

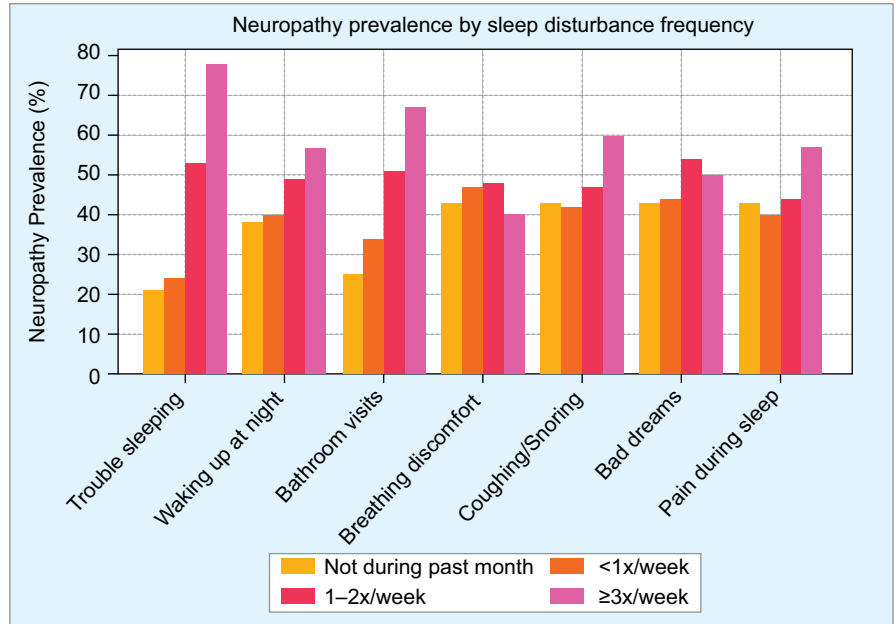


Fig. 4: Predictors of PSQI (bar graph)

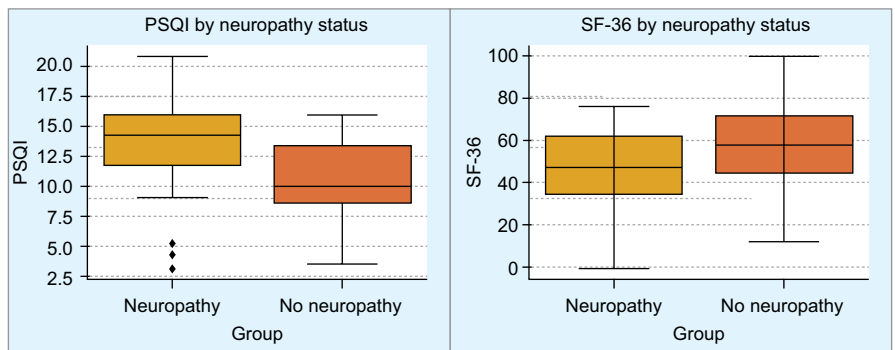


Fig. 5: Predictors of SF-36 (bar graph)

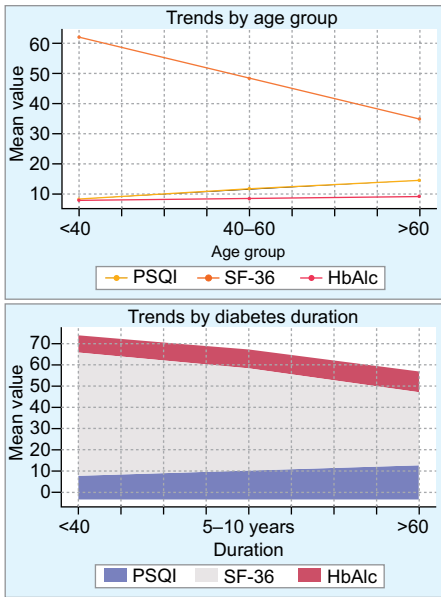


Fig. 6: Trends in PSQI, SF-36, and HbA1c by age group and diabetes duration

Table 7: Correlation analysis

Pair	Pearson's <i>r</i>	<i>p</i> -value
HbA1c vs PSQI	0.29	< 0.001
HbA1c vs SF-36	-0.41	< 0.001
PSQI vs SF-36	-0.63	< 0.001

HbA1c, glycated hemoglobin; PSQI, Pittsburgh Sleep Quality Index; SF-36, 36-Item Short Form Health Survey

Table 8: Regression analysis

Predictors of PSQI		
Predictor	Coefficient	<i>p</i> -value
HbA1c	0.35	<0.001
Neuropathy	1.9	<0.001
SF-36	-0.18	<0.001
Age	0.06	<0.03
Predictors of SF-36		
Predictor	Coefficient	<i>p</i> -value
PSQI	-3.8	<0.001
Retinopathy	-8.2	<0.001
Age	-0.5	<0.03

PSQI, Pittsburgh Sleep Quality Index; SF-36, 36-Item Short Form Health Survey

of 0.42. SF-36 scores were significantly predicted by PSQI ($\beta = -3.8$, $p < 0.001$), age ($\beta = -0.5$, $p = 0.02$), and retinopathy ($\beta = -8.2$, $p = 0.004$), with an R^2 of 0.65 (Table 8).

Neuropathy emerged as the strongest predictor of poor sleep and diminished QoL, underscoring the importance of comprehensive, targeted interventions to improve these outcomes in patients with T2DM. Older age and longer diabetes duration correlated with worse sleep quality ($\uparrow$ PSQI),

Table 9: Age-group and diabetes-duration analysis

Age group	Mean PSQI $\pm$ SD	Mean SF-36 $\pm$ SD	HbA1c (%)
<40	8.2	62.1	7.9
40-60	11.7	48.4	8.3
>60	14.5	34.8	9.1
DM duration	Mean PSQI $\pm$ SD	Mean SF-36 $\pm$ SD	HbA1c (%)
<5 years	10.4 $\pm$ 4.2	54.8 $\pm$ 25.3	7.8
5-10 years	12.8 $\pm$ 4.5	45.3 $\pm$ 21.4	8.5
>10 years	15.1 $\pm$ 3.9	32.5 $\pm$ 18.7	9.4

DM, diabetes mellitus; HbA1c, glycated hemoglobin; PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; SF-36, 36-Item Short Form Health Survey

lower QoL ($\downarrow$ SF-36), and higher HbA1c (Fig. 6 and Table 9).

DISCUSSION

Diabetes has been consistently linked to diminished QoL and impaired sleep among affected individuals. This association is evident in our study, where the mean PSQI score was 12.6, and the mean SF-36 quality-of-life score was 46.2, both indicative of significant impairment. Similar findings have been reported in a large comparative study involving 292 patients with diabetes and 291 nondiabetic controls. In that study, poor sleep quality was reported by 50.7% of individuals with diabetes, markedly higher than the 31.8% observed in the nondiabetic group.¹⁰ Additionally, certain demographic factors appear to exacerbate sleep disturbances in populations with diabetes. Notably, female patients tend to report poorer sleep quality compared with their male counterparts.¹¹ Variations in hormone levels throughout the menstrual cycle, pregnancy, and menopause significantly contribute to these differences. Moreover, societal expectations and caregiving responsibilities can intensify stress and sleep disturbances, particularly affecting women. In this study, sleep quality among females was lower (PSQI = 13.7) than that among males (PSQI = 11.8). In addition, QoL among females was lower than that among males. These findings highlight the need for sex-specific strategies to enhance sleep and QoL in patients with diabetes.

Poor sleep quality has been implicated in the dysregulation of glycemic control through multiple physiological pathways. Disrupted hormonal balance, particularly involving cortisol and growth hormone, may contribute to increased insulin resistance and elevated blood glucose levels.^{12,13} Additionally, sleep deprivation can trigger proinflammatory responses that further compromise glucose metabolism.¹⁴ In the present study, a modest positive correlation was observed between PSQI scores and HbA1c levels ($r = 0.29$), lending support to these mechanistic associations.

This study identified a strong inverse correlation ($r = -0.63$) between sleep quality and overall QoL. Poor sleep quality and sleep-related disorders are associated with disruptions in critical physiological functions, including metabolic slowing and impairments in motor control, regulatory balance, transport mechanisms, and the body's protective responses. These disruptions may increase vulnerability to injuries or even life-threatening events, particularly in cases involving sleepwalking or narcoleptic episodes.¹⁵⁻¹⁷

A diabetes duration exceeding 10 years is associated with significantly poorer sleep quality (mean PSQI: 15.1 $\pm$ 3.9), highlighting the progressive and cumulative burden of T2DM.¹⁸ This finding aligns with previous literature and underscores the well-established link between prolonged hyperglycemia and the development of diabetes-related complications.¹⁹ This study also found that the presence of complications—especially neuropathy—was strongly associated with poorer sleep quality. Moreover, the coexistence of multiple complications appeared to exacerbate this impact, further impairing sleep and overall health, consistent with findings reported in previous research.²⁰

Neuropathy emerged as the primary factor influencing sleep quality in this study. Its impact may be mediated through several mechanisms, particularly neuropathic pain and discomfort, which can significantly interfere with restful sleep. Previous studies have also highlighted these pathways, demonstrating that neuropathy not only disrupts nocturnal rest but also worsens QoL, thereby compounding sleep disturbances.^{20,21} Patients with neuropathy in this study exhibited significantly higher rates of sleep disturbances—such as frequent awakenings and pain—compared with those without neuropathy. Neuropathic symptoms, including pain, tingling, and burning sensations in the extremities, often intensify at night when external distractions diminish. This increased nocturnal discomfort can hinder sleep onset

and lead to recurrent awakenings, thereby substantially impairing sleep continuity.²² The cornerstone of management is strict glycemic control, which has been shown to slow disease progression and alleviate symptom severity, even after the onset of neuropathic manifestations.²³ Managing neuropathy-related sleep disturbances requires a comprehensive approach that integrates lifestyle modifications and nutritional support. A balanced diet and regular physical activity, combined with neurotropic B vitamins (B1, B6, and B12), can enhance nerve function and overall well-being. Maintaining a consistent sleep schedule, reducing intake of caffeine and alcohol, and avoiding heavy meals in the evening are essential behavioral strategies. Establishing calming pre-sleep routines—such as reading, listening to soothing music, or enjoying herbal teas—can signal the body to wind down. Creating a sleep-conducive environment with low lighting, comfortable bedding, and an optimal room temperature further supports restful sleep. Finally, unplugging from screens before bedtime and practicing relaxation techniques such as deep breathing, progressive muscle relaxation, meditation, or taking a warm bath can significantly improve sleep quality in individuals with neuropathy.²²

Older patients in this study demonstrated poorer sleep quality compared with their younger counterparts. This observation aligns with the understanding that prolonged periods of poor glycemic control contribute to a higher risk of complications. Additionally, age-related changes in circadian rhythms and the presence of comorbidities further exacerbate sleep disturbances in older adults, thereby supporting our findings.²⁴ These trends highlight the importance of tailored interventions for older populations with diabetes.

Strengths, Limitations, and Future Directions

This study benefits from the use of validated tools such as PSQI and SF-36, which bolster its methodological robustness. However, its cross-sectional design limits causal inferences, and self-reported data may introduce recall bias. Future research should explore longitudinal and interventional designs to establish causality and evaluate the efficacy of integrated care models for improving sleep and diabetes outcomes.

CONCLUSION AND IMPLICATIONS

The findings of this study reinforce the growing evidence that poor sleep quality is a significant concern among individuals with

T2DM, contributing directly to worsening QoL. With a mean PSQI score of 12.6 and an SF-36 score of 46.2 in the population with diabetes, it is evident that sleep disturbances are highly prevalent and impactful. The data revealed that multiple demographic and clinical variables, including female sex, advanced age, longer diabetes duration, and the presence of complications—especially neuropathy—are associated with worsened sleep quality. This creates a vicious interdependent cycle in which diabetes worsens sleep, compromised sleep deteriorates metabolic health, and both together impair overall QoL.

Diabetic neuropathy emerged as the most critical determinant of disrupted sleep. The discomfort, pain, and sensory abnormalities associated with neuropathy result in frequent awakenings and difficulty initiating or maintaining sleep. This not only compromises sleep continuity but also creates a negative cycle that exacerbates glycemic instability and further leads to deterioration of overall health and daily functioning.

Although the correlation between HbA1c levels and sleep quality was weaker ($r = 0.29$), the impact of poor sleep on hormonal regulation and metabolic pathways remains clinically relevant. Chronic sleep disturbances can contribute to heightened insulin resistance and systemic inflammation, ultimately undermining glycemic control and increasing the risk of diabetic complications.

Gender-specific differences were also notable, with females reporting significantly poorer sleep and lower QoL, further emphasizing the necessity for sex-sensitive approaches in the management of T2DM-related sleep issues. Hormonal fluctuations, caregiving responsibilities, and psychosocial stressors may amplify sleep disturbances in women, necessitating holistic and tailored interventions.

Furthermore, the association between advancing age and poorer sleep among diabetic individuals reflects the cumulative burden of prolonged disease duration, circadian rhythm disruptions, and age-related comorbidities. These findings stress the importance of early intervention and continuous monitoring to mitigate the progressive decline in sleep and health outcomes in elderly patients.

Overall, this study underscores the importance of a multidisciplinary management strategy for diabetic patients, one that not only focuses on glycemic control but also incorporates sleep quality assessment and intervention. Strategies such as optimizing neuropathy management, promoting sleep hygiene, addressing psychosocial stressors, and encouraging lifestyle modifications such

as regular physical activity and balanced nutrition can collectively improve sleep and enhance overall QoL in this vulnerable population.

To improve sleep quality and QoL, controlling the glycemic index remains the mainstay approach; however, other hindrances to uninterrupted sleep can also be addressed, such as (1) polyuria, a common denominator in patients with T2DM that causes frequent awakenings at night, and (2) neuropathic pain, which makes sleep initiation difficult. By reducing these smaller but significant symptoms, uninterrupted, quality sleep and improved QoL may be promoted.

ETHICAL STATEMENT

The study was conducted according to IRB ethical policies and involved less than minimal risk.

CONFLICT OF INTEREST

None.

SOURCE OF SUPPORT

The study was self-funded; no external funding was received.

TIMELINE

- Data collection: 2 months.
- Data analysis: 1 week.
- Manuscript preparation: 1 week.
- Submission for publication: 1 week.

BUDGET

- Materials: Questionnaires and statistical software.
- Other costs: Administrative costs and publication fees.

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