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Prevalence And Determinants Of Poor Sleep Quality Among Patients With Type 2 Diabetes Mellitus

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ABSTRACT

Background
Cardiomyopathies are a heterogeneous group of myocardial disorders associated with significant morbidity, heart failure, ventricular arrhythmias, and sudden cardiac death. Accurate differentiation between ischemic and non-ischemic cardiomyopathy is essential because the underlying etiology influences therapeutic strategies, risk stratification, and prognosis. Although two-dimensional echocardiography remains the first-line imaging modality for the assessment of ventricular structure and function, cardiac magnetic resonance (CMR) provides superior tissue characterization, myocardial strain analysis, and detection of myocardial fibrosis through late gadolinium enhancement. The present study was undertaken to evaluate the clinical profile and compare echocardiographic and CMR findings among patients with cardiomyopathy in a tertiary care hospital, with emphasis on their complementary diagnostic value.
Aim: This study aimed to assess the prevalence of poor sleep quality and its associated factors among patients with T2DM.
Materials and Methods: This prospective cross-sectional study was conducted at a tertiary care center, India, over two months. Adult patients (≥18 years) with T2DM were enrolled. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), with a global score ≥5 defining poor sleep quality. Demographic and clinical variables, including age, sex, body mass index (BMI), duration of diabetes, treatment modality, random blood sugar (RBS), and presence of neuropathy, were collected. Logistic regression analysis was performed to identify factors associated with poor sleep quality.
Results: A total of 109 patients were included (mean age 59.85 ± 11.10 years; 40.4% female). Poor sleep quality was observed in 68.8% of participants (75/109; 95% CI: 59.6–76.7), with a mean PSQI score of 6.95 ± 3.79. Higher prevalence was noted among females (75.0% vs 64.6%) and those with neuropathy (76.9% vs 66.3%). However, no variables were significantly associated with poor sleep quality on multivariable analysis, including age (adjusted OR 1.13 per 10 years; p=0.566), female sex (adjusted OR 1.80; p=0.207), BMI (adjusted OR 0.95; p=0.300), RBS (adjusted OR 1.25 per 50 mg/dL; p=0.209), duration of diabetes (adjusted OR 0.89; p=0.512), and neuropathy (adjusted OR 1.56; p=0.425).
Conclusion: Poor sleep quality is highly prevalent among patients with T2DM, affecting nearly two-thirds of individuals. The absence of independent predictors suggests a multifactorial etiology, highlighting the need for routine sleep assessment as part of comprehensive diabetes care.

Keywords: diabetes mellitus; Karnataka; predictors; quality of life; sleep quality.

Introduction
Type 2 diabetes mellitus (T2DM) is a major global health challenge associated with morbidity and mortality, primarily driven by its metabolic and vascular complications. In addition to traditional risk

factors, increasing attention has been directed toward non-traditional contributors, such as sleep disturbances, which may significantly influence disease progression and overall health outcomes [1]. Sleep is a fundamental physiological process essential for metabolic homeostasis, and its disruption has been shown to adversely affect glucose metabolism, insulin sensitivity, and neuroendocrine regulation [2].

Poor sleep quality is increasingly recognized as a common yet underdiagnosed problem among individuals with T2DM [3]. Emerging evidence suggests a bidirectional relationship between sleep disturbances and diabetes, wherein impaired sleep may worsen glycemic control, while chronic hyperglycemia and diabetes-related complications may, in turn, negatively impact sleep quality [4]. This interaction may contribute to reduced quality of life and increased risk of adverse clinical outcomes. The Pittsburgh Sleep Quality Index (PSQI) is a widely validated and commonly used instrument for assessing subjective sleep quality in both clinical and research settings. It evaluates multiple domains of sleep, including duration, latency, efficiency, disturbances, and daytime dysfunction, providing a global measure of sleep quality [2,5]. Previous studies utilizing PSQI have demonstrated that poor sleep quality is highly prevalent among patients with T2DM, highlighting the clinical importance of routine sleep assessment in this population [6].

Despite increasing recognition of the relationship between sleep and diabetes, the determinants of poor sleep quality remain incompletely understood, with variability in findings across different populations and settings [7]. Furthermore, there is a relative paucity of data, particularly from real-world tertiary care settings, where demographic and clinical characteristics may differ from those reported in other regions. Therefore, the present study aims to assess the prevalence of poor sleep quality using the PSQI and to evaluate its associated factors among patients with T2DM attending a tertiary care hospital in North Karnataka, India.

Materials and Methods

Study design and population

This was a prospective cross-sectional study which was conducted at tertiary care centre India, over a period of two months. The study was approved by the Institutional Ethics Committee DHR Reg. No. EC/NEW/INST/2024/KA/0548 (Ref No. MDC/JNMCIEC/342), and all participants provided written informed consent prior to enrollment. Adult patients (≥18 years) with a known diagnosis of T2DM presenting to outpatient or inpatient services were enrolled in this study. Patients with Type 1 diabetes, known obstructive sleep apnea, diagnosed insomnia or other sleep disorders, psychiatric disorders, gestational diabetes, or morbid obesity as per WHO Asia-Pacific criteria were excluded.

Data collection

Baseline demographic and clinical data, including age, sex, body mass index (BMI), duration of diabetes, treatment modality (oral hypoglycemic agents [OHAs], insulin, or combination therapy), and random blood sugar (RBS) levels, were collected using a structured case record form. Additionally, the presence of neuropathy as a perceived obstacle to sleep was documented.

Assessment of sleep quality

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), assessing seven components: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The sum of these components brings about a global score. A score above 5 indicates poor sleep quality while a score below 5 indicates good sleep quality with 89.6% sensitivity and 86.5% specificity. The PSQI yields a global score ranging from 0 to 21, with higher scores indicating poorer sleep quality[5,8]. Based on standard cut-offs, participants were categorized into good sleep quality (PSQI <5) and poor sleep quality (PSQI ≥5).

Statistical analysis

Continuous variables were expressed as mean ± SD or median (IQR), and categorical variables as n (%). Group comparisons were performed using t-tests or Mann–Whitney U tests for continuous variables and chi-square or Fisher's exact tests for categorical variables. Logistic regression was used to evaluate factors associated with poor sleep quality. Univariate and multivariable models were fitted,

and results are reported as odds ratios with 95% confidence intervals. Continuous predictors were scaled to clinically meaningful units prior to regression analysis. A two-sided p-value < 0.05 was considered statistically significant. All statistical analyses were performed using R software (version 4.3.3).

Results

A total of 109 participants were included in the analysis. Of these, 34 (31.2%) were classified as having good sleep quality (PSQI <5), while 75 (68.8%) had poor sleep quality (PSQI ≥5). The mean age of the cohort was 59.85 ± 11.10 years and was comparable between the good and poor sleep groups (59.32 ± 10.48 vs 60.09 ± 11.43 years; p=0.731). Similarly, no significant differences were observed in body mass index (25.70 ± 4.65 vs 24.72 ± 5.02 kg/m²; p=0.323), median random blood sugar levels (163.5 [132.5–218.8] vs 185.0 [140.5–231.5] mg/dL; p=0.162), or duration of diabetes (8.54 ± 7.96 vs 9.07 ± 6.91 years; p=0.739) between the two groups. Baseline characteristics of patients stratified based on sleep quality was described in Table 1.

Table 1: Baseline characteristics of study participants stratified by sleep quality

Variable	Overall (N=109 patients)	Good sleep (PSQI<5) (n=34 patients)	Poor sleep (PSQI≥5) (n=75 patients)	P value
Age, years	59.85 ± 11.10	59.32 ± 10.48	60.09 ± 11.43	0.731
BMI, kg/m²	25.03 ± 4.91	25.70 ± 4.65	24.72 ± 5.02	0.323
RBS value, mg/dL	175.0 (140.0, 220.0)	163.5 (132.5, 218.8)	185.0 (140.5, 231.5)	0.162
Duration of diabetes/treatment, years	8.90 ± 7.22	8.54 ± 7.96	9.07 ± 6.91	0.739
Global PSQI score	6.95 ± 3.79	2.97 ± 1.09	8.76 ± 3.14	<0.001
Male	65 (59.6)	23 (67.6)	42 (56.0)	0.251
Female	44 (40.4)	11 (32.4)	33 (44.0)	
Insulin	46 (42.2)	14 (41.2)	32 (42.7)	0.884
OHAs	98 (89.9)	28 (82.4)	70 (93.3)	0.078
Treatment category				
OHAs only	54 (49.5)	15 (44.1)	39 (52.0)	0.367
OHAs + Insulin	44 (40.4)	13 (38.2)	31 (41.3)	
Insulin only	2 (1.8)	1 (2.9)	1 (1.3)	
Neither	9 (8.3)	5 (14.7)	4 (5.3)	
Neuropathy as obstacle	26 (23.9)	6 (17.6)	20 (26.7)	0.306

Data are presented as mean ± standard deviation or median (interquartile range) for continuous variables, and frequency (percentage) for categorical variables. P values <0.05 were considered statistically significant. BMI: Body mass index; OHAs: Oral hypoglycemic agents; PSQI: Pittsburgh sleep quality index; RBS: Random blood sugar.

The overall prevalence of poor sleep quality (PSQI ≥5) was 68.8% (75/109; 95% CI: 59.6–76.7). Poor sleep quality was more prevalent among females compared to males (75.0% vs 64.6%). Across age categories, prevalence ranged from 62.5% in the 40–49 year group to 72.2% in the 60–69 year group. When stratified by body mass index, the highest prevalence was observed among underweight participants (85.7%), while prevalence in other BMI categories ranged from 61.3% to 71.4%. Patients

reporting neuropathy as an obstacle had a higher prevalence of poor sleep quality compared to those without neuropathy (76.9% vs 66.3%). Prevalence of poor sleep quality across clinical and demographic variables are detailed in Table 2.

Table 2: Prevalence of poor sleep quality across clinical and demographic variables

Subgroup	Poor sleep (n=75 patients)	95% CI
PQSI ≥ 5	75/109 (68.8)	59.6–76.7
Sex		
Female	33/44 (75.0)	60.6–85.4
Male	42/65 (64.6)	52.5–75.1
Age group, years		
18–39	2/3 (66.7)	20.8–93.9
40–49	10/16 (62.5)	38.6–81.5
50–59	21/30 (70.0)	52.1–83.3
60–69	26/36 (72.2)	56.0–84.2
≥70	16/24 (66.7)	46.7–82.0
BMI category, kg/m²		
Underweight (<18.5)	6/7 (85.7)	48.7–97.4
Normal (18.5–22.9)	21/30 (70.0)	52.1–83.3
Overweight (23–24.9)	15/21 (71.4)	50.0–86.2
Obese I (25–29.9)	19/31 (61.3)	43.8–76.3
Obese II (≥30)	14/20 (70.0)	48.1–85.5
Treatment category		
Insulin only	1/2 (50.0)	9.5–90.5
Neither	4/9 (44.4)	18.9–73.3
OHAs only	39/54 (72.2)	59.1–82.4
OHAs + Insulin	31/44 (70.5)	55.8–81.8
Neuropathy as obstacle	20/26 (76.9)	57.9–89.0

Data are presented as number/total (percentage) with corresponding 95% confidence intervals (CIs). Poor sleep quality was defined as PSQI ≥5. BMI: Body mass index; CI: Confidence interval; OHAs: Oral hypoglycemic agents; PSQI: Pittsburgh sleep quality index

Table 3: Univariate and multivariable logistic regression analysis of factors associated with poor sleep quality

Predictor	Unadjusted OR (95% CI)	P value	Adjusted OR (95% CI)	P value
Age (per 10-year increase)	1.07 (0.74–1.54)	0.736	1.13 (0.74–1.72)	0.566
Sex (Female vs Male)	1.64 (0.70–3.85)	0.253	1.80 (0.72–4.49)	0.207

BMI (per 1 kg/m²)	0.96 (0.88–1.04)	0.333	0.95 (0.87–1.04)	0.3
RBS value (per 50 mg/dL)	1.25 (0.91–1.72)	0.176	1.25 (0.88–1.76)	0.209
Duration of diabetes (per 5 years)	1.05 (0.79–1.40)	0.722	0.89 (0.64–1.25)	0.512
Neuropathy obstacle (Yes vs No)	1.70 (0.61–4.70)	0.309	1.56 (0.52–4.66)	0.425

P values <0.05 were considered statistically significant. BMI: Body mass index; CI: Confidence interval; OR: Odds ratio; PSQI: Pittsburgh sleep quality index; RBS: Random blood sugar

On univariate logistic regression analysis, age (OR 1.07 per 10-year increase; 95% CI: 0.74–1.54; p=0.736), female sex (OR 1.64; 95% CI: 0.70–3.85; p=0.253), BMI (OR 0.96 per 1 kg/m²; 95% CI: 0.88–1.04; p=0.333), random blood sugar (OR 1.25 per 50 mg/dL increase; 95% CI: 0.91–1.72; p=0.176), duration of diabetes (OR 1.05 per 5-year increase; 95% CI: 0.79–1.40; p=0.722), and presence of neuropathy (OR 1.70; 95% CI: 0.61–4.70; p=0.309) were not significantly associated with poor sleep quality. In the multivariable model, none of the variables demonstrated a statistically significant independent association with poor sleep quality. Female sex (adjusted OR 1.80; 95% CI: 0.72–4.49; p=0.207) and higher random blood sugar levels (adjusted OR 1.25 per 50 mg/dL increase; 95% CI: 0.88–1.76; p=0.209) showed a non-significant trend toward increased odds, while BMI (adjusted OR 0.95; 95% CI: 0.87–1.04; p=0.300) and duration of diabetes (adjusted OR 0.89; 95% CI: 0.64–1.25; p=0.512) were not associated with the outcome. Univariate and multivariable logistic regression analysis of factors associated with poor sleep quality are showed in Table 3.

Discussion

In this study, poor sleep quality (PSQI ≥5) was highly prevalent among patients with T2DM, affecting 68.8% of patients (95% CI: 59.6–76.7). The mean global PSQI score of 6.95 ± 3.79 further reflects a substantial burden of sleep disturbance in this population. Although several demographic and clinical variables showed clinically relevant trends, none demonstrated a statistically significant association with poor sleep quality on univariate or multivariable analysis. These findings suggest that sleep disturbance in T2DM may not be driven by a single identifiable clinical factor but rather represents a broader and more pervasive feature of the disease.

The high prevalence observed in our study is consistent with findings from other Indian and South Asian populations. A study from a tertiary care center in South India reported that approximately 69% of patients with T2DM had poor sleep quality, closely mirroring our results [9]. Similar studies from West Bengal and North India have reported prevalence rates ranging from 55% to 57%, indicating a consistently high burden across different regions of India [10,11]. In contrast, community-based data from the Longitudinal Ageing Study in India reported a substantially lower prevalence, with nearly one in four individuals with diabetes experiencing poor sleep [12]. This difference is expected, as tertiary care settings typically include patients with more advanced disease and greater symptom burden. International data also show considerable variability. A hospital-based study from Somalia reported a prevalence of 54% [13], whereas a tertiary care study from Pakistan reported a higher prevalence of 82% [6]. These differences likely reflect variations in study populations, healthcare settings, and the inclusion of important variables such as psychological factors, comorbidities, and lifestyle behaviors, all of which are known to influence sleep quality.

A higher proportion of poor sleep was observed among females compared to males. This trend is consistent with existing literature, which suggests that women, particularly in older age groups, are more likely to experience sleep disturbances. The underlying mechanisms are likely multifactorial and may include hormonal changes, especially in postmenopausal women, as well as higher levels of psychological stress and depression. These findings highlight the importance of considering sex-specific factors when evaluating sleep quality in patients with T2DM [14].

Participants reporting neuropathy as an obstacle had a higher prevalence of poor sleep, which is biologically plausible. Neuropathic pain and nocturnal discomfort are well-recognized contributors to sleep fragmentation in diabetes. In addition, other mechanisms such as nocturia, restless legs syndrome, and sleep-disordered breathing may further contribute to impaired sleep [15,16]. In our study, neuropathy

was assessed based on patient-reported perception rather than objective clinical grading, which may have underestimated its true impact and partially explains the lack of statistical significance.

Higher random blood sugar levels were observed among participants with poor sleep. This finding is consistent with the known bidirectional relationship between sleep and glycemic control. Sleep deprivation has been shown to increase insulin resistance and disrupt metabolic regulation, while hyperglycemia can contribute to nocturnal symptoms such as frequent urination and discomfort, further impairing sleep [14,17]. The use of random blood sugar rather than HbA1c in this study is an important limitation, as HbA1c better reflects long-term glycemic control and may have provided a stronger association with sleep quality.

The absence of statistically significant predictors should not be interpreted as a negative result but rather as evidence of the complex, multifactorial nature of sleep disturbances in T2DM. Diabetes can disrupt sleep through symptoms such as nocturia, neuropathic pain, and glycaemic fluctuations, while poor sleep can, in turn, worsen metabolic control. Given this close interaction, the overall burden of diabetes may exert a relatively uniform effect on sleep quality, limiting the discriminatory ability of individual clinical variables. The relatively small sample size may also have limited statistical power to detect modest associations. Trends observed in our study, higher odds of poor sleep among females and those with elevated blood glucose are consistent with previous literature and may reach significance in larger cohorts. Finally, the absence of key variables such as psychological factors, physical activity, and socioeconomic status, all well-established determinants of sleep quality, may have further contributed to the lack of significant associations.

Limitations

This study has several limitations. First, it was conducted at a single tertiary care center with a relatively small sample size, which may limit generalizability and reduce statistical power to detect modest associations. Second, the cross-sectional nature of the analysis precludes any inference of causality. Third, glycemic control was evaluated using random blood sugar levels rather than HbA1c, which may better represent long-term metabolic status. Finally, important confounders such as psychological factors, physical activity, and socioeconomic status were not included, which may have influenced the observed findings.

Conclusion

Poor sleep quality was highly prevalent among patients with type 2 diabetes mellitus, affecting 68.8% of patients. Despite this substantial burden, no independent clinical predictors were identified, suggesting that sleep disturbances in T2DM are likely multifactorial and not solely explained by conventional demographic or metabolic variables. These findings underscore the importance of routine assessment of sleep quality in patients with diabetes, irrespective of baseline characteristics.

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