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ASSOCIATION BETWEEN SLEEP DISORDERS AND CARDIOVASCULAR HEALTH – EXAMINING HOW SLEEP PATTERNS AFFECT HEART DISEASE RISKS

Original Article

Shazia Safdar[1]*, Shaista Ismail[1]*, Bashir Ullah[2]*, Tooba Shaikh[3], Aqib Dil Awaiz[4], Iqra Munir Qazi[5], Mashkoor Ahmed Ansari[6], Muhammad Naeem[7]

[1]Charge Nurse, Post RN BSN, New Life Institute of Nursing, Multan, UHS Lahore, Pakistan.
[2]Faculty of Cardiology and Cardiac Perfusion Technology, Institute of Paramedical Sciences, Khyber Medical University, Peshawar, Pakistan.
[3]Senior Registrar, Baqai Institute of Diabetes and Endocrine, Medicine Unit 4, Karachi, Pakistan.
[4]Associate Professor / Managing Director, New Life Institute of Nursing, Multan, Pakistan.
[5]Department of Zoology, University of Sindh, Jamshoro, Pakistan.
[6]Associate Professor, Department of Pharmacology, Liaquat University of Medical & Health Sciences, Jamshoro, Pakistan.
[7]Assistant Professor, UVAS, Lahore, Pakistan.

Corresponding Authors:
Shazia Safdar, Charge Nurse, Post RN BSN, New Life Institute of Nursing, Multan, UHS Lahore, Pakistan. bhattithe1@gmail.com
Shaista Ismail, Charge Nurse, Post RN BSN, New Life Institute of Nursing, Multan, UHS Lahore, Pakistan. shaistazeeshan3658@gmail.com
Bashir Ullah, Faculty of Cardiology and Cardiac Perfusion Technology, Institute of Paramedical Sciences, Khyber Medical University, Peshawar, Pakistan. bashir.ipms@kmu.edu.pk

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ABSTRACT

Background: Sleep disorders are increasingly recognized as modifiable risk factors for cardiovascular disease (CVD), yet remain underdiagnosed and underprioritized in clinical assessments, particularly in low- and middle-income countries.

Objective: To determine the association between sleep disorders and cardiovascular health by examining how variations in sleep patterns affect heart disease risk in adults.

Methods: A cross-sectional study was conducted over eight months at a tertiary care hospital in Lahore, Pakistan, including 422 adults aged 30–65 years. Sleep quality, daytime sleepiness, and obstructive sleep apnea risk were assessed using the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and Berlin Questionnaire, respectively. Cardiovascular risk was evaluated using the Framingham Risk Score (FRS). Statistical analyses included independent t-tests, ANOVA, chi-square tests, and multivariate linear regression, with significance set at $p<0.05$.

Results: Of 422 participants, 244 (57.8%) reported poor sleep quality. These individuals had significantly higher mean FRS scores (14.8 ± 5.3) compared to those with good sleep (9.3 ± 4.1). A graded increase in FRS was observed with increasing ESS scores: 10.1 ± 4.6 in the normal group, 12.9 ± 5.0 in mild, and 15.7 ± 5.8 in moderate-to-severe categories. Participants at high risk for obstructive sleep apnea also exhibited elevated FRS (15.1 ± 5.5) compared to the low-risk group (9.7 ± 3.9).

Conclusion: Sleep disturbances, including poor sleep quality, daytime sleepiness, and sleep apnea risk, are strongly associated with elevated cardiovascular risk. Early identification and management of sleep disorders may offer a valuable preventive strategy against CVD.

Keywords: Adult, Cardiovascular Diseases, Cross-Sectional Studies, Framingham Risk Score, Pakistan, Risk Assessment, Sleep Apnea Syndromes, Sleep Deprivation, Sleep Quality, Sleep Wake Disorders.

INTRODUCTION

Sleep is a fundamental biological function, vital for maintaining physiological balance and supporting overall health. Despite its importance, sleep disturbances and disorders are increasingly prevalent worldwide, with their implications extending far beyond fatigue and reduced daytime performance. One growing area of concern is the potential relationship between sleep disorders and cardiovascular health. Mounting evidence suggests that disruptions in sleep quantity or quality may play a significant role in the development and progression of cardiovascular disease (CVD), a leading cause of mortality globally (1). In recent years, attention has shifted toward understanding how sleep habits may serve as modifiable risk factors for cardiovascular morbidity and mortality, and yet, this domain remains underexplored, particularly in cross-sectional community-based populations. Cardiovascular diseases encompass a range of conditions, including coronary artery disease, stroke, heart failure, and hypertension, each characterized by complex etiologies involving both genetic and environmental factors (2). Traditionally, known risk factors for CVD include poor diet, sedentary lifestyle, smoking, obesity, hypertension, and diabetes. However, a growing body of research has highlighted the significance of sleep as an additional, independent risk factor. Disturbances such as insomnia, obstructive sleep apnea (OSA), short or long sleep duration, and fragmented sleep have been consistently associated with increased cardiovascular risk. For example, a large cross-sectional study in Brazil found that adults experiencing sleep disturbances were significantly less likely to achieve ideal cardiovascular health, as measured by Life's Simple 7 metrics (3,4).

The mechanisms underlying this association are multifactorial. Sleep deprivation and poor sleep quality are known to disrupt metabolic processes, elevate blood pressure, and increase sympathetic nervous system activity, all of which contribute to cardiovascular strain. Additionally, inflammation—a known contributor to atherosclerosis and other vascular abnormalities—is exacerbated by inadequate or disturbed sleep. For instance, the Gutenberg Health Study found that individuals reporting significant sleep disturbances had higher levels of inflammatory biomarkers such as C-reactive protein and fibrinogen, alongside a higher prevalence of myocardial infarction and dyslipidemia (5,6). The pattern of sleep duration appears to play a particularly critical role. Both short (<6 hours) and long (>9 hours) sleep durations have been associated with increased cardiovascular risk, a U-shaped relationship that persists even after controlling for confounding variables. This phenomenon was clearly illustrated in a population-based study from Russia, which found that individuals reporting either very short or very long sleep durations had higher odds of developing coronary artery disease and myocardial infarction (7,8).

Sleep-disordered breathing, especially obstructive sleep apnea, has emerged as a particularly potent cardiovascular risk factor. It not only disrupts sleep architecture but also induces intermittent hypoxia, which in turn leads to oxidative stress and endothelial dysfunction. Findings from the Sleep Heart Health Study demonstrated that individuals with higher apnea-hypopnea indices had increased odds of heart failure and stroke compared to those with lower indices (9). Furthermore, studies have also suggested a role of mood disorders as mediators in the link between sleep and cardiovascular disease. For example, a recent analysis of U.S. NHANES data revealed that depressive symptoms partially mediated the relationship between sleep disorders and CVD, indicating that the psychosocial effects of poor sleep may compound physiological risks (10,11). Despite these compelling associations, there remains a need for more population-based studies to comprehensively evaluate the strength and nature of the relationship between sleep disorders and cardiovascular health. Particularly, there is a gap in understanding the nuanced effects of various types of sleep disturbances, as well as their interactions with demographic and behavioral variables. Cross-sectional data, while limited in causal inference, offer an important snapshot that can guide public health initiatives and clinical practice toward better sleep management as a preventive strategy against cardiovascular disease. Therefore, the current cross-sectional study is designed to assess the association between sleep disorders and cardiovascular health, specifically focusing on how variations in sleep patterns—such as duration, quality, and the presence of clinical sleep disorders—relate to the risk of cardiovascular disease in adults. The objective is to provide empirical evidence that could inform both preventive healthcare and targeted interventions aimed at improving sleep hygiene as a modifiable factor in cardiovascular risk reduction.

METHODS

This cross-sectional study was conducted over a period of eight months at a tertiary care hospital in Lahore, Pakistan. The primary objective was to investigate the association between sleep disorders and cardiovascular health, with a specific focus on how variations in sleep patterns influence the risk of heart disease. The study was designed with methodological rigor to ensure that findings would contribute meaningful insights to the growing body of evidence surrounding cardiovascular risk factors, particularly those related to sleep. The target population for this study comprised adult patients aged 30 to 65 years, representing a demographically diverse patient

group attending outpatient departments. Inclusion criteria required participants to be within the specified age range, willing to provide informed consent, and free from any diagnosed psychiatric or neurological conditions that could influence sleep or cardiovascular parameters. Patients with a history of diagnosed cardiovascular disease under current treatment, or those with secondary causes of sleep disturbances such as pregnancy, night-shift employment, or substance abuse, were excluded to minimize confounding factors.

A calculated sample size of 384 participants was determined using Cochran's formula for prevalence studies, assuming a 50% prevalence rate of sleep disorders with a 5% margin of error and 95% confidence interval. The sample size was further inflated by 10% to accommodate possible non-responses, bringing the total to approximately 422 participants. A systematic random sampling technique was used to recruit patients who met the eligibility criteria (12). Data collection involved structured, face-to-face interviews administered by trained healthcare professionals. The Pittsburgh Sleep Quality Index (PSQI) was used to assess overall sleep quality, while the Epworth Sleepiness Scale (ESS) captured daytime sleepiness, and the Berlin Questionnaire was applied to evaluate the risk of obstructive sleep apnea. These validated instruments allowed for comprehensive categorization of sleep disturbances, which were then stratified into poor sleep quality, excessive daytime sleepiness, and high risk for sleep-disordered breathing.

Cardiovascular health was assessed using the Framingham Risk Score (FRS), a widely accepted tool that estimates the 10-year risk of developing cardiovascular disease based on parameters such as age, sex, total cholesterol, HDL cholesterol, systolic blood pressure, and smoking status. Blood pressure was measured using a calibrated sphygmomanometer, while fasting blood samples were collected to determine lipid profiles (12,13). Anthropometric measurements, including body mass index (BMI), were also recorded using standard procedures. All data were entered into SPSS version 26 for statistical analysis. Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables were presented as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. The normality of data distribution was confirmed using the Shapiro-Wilk test. To evaluate associations between sleep disorder categories and cardiovascular risk, independent sample t-tests and one-way ANOVA were employed for continuous variables, while chi-square tests were used for categorical variables. Multivariate linear regression analysis was conducted to control for potential confounding variables such as age, gender, BMI, smoking status, and physical activity level. A p-value of less than 0.05 was considered statistically significant for all analyses.

Ethical approval for the study was granted by the Institutional Review Board of the host institution. Participants were fully informed about the nature, scope, and aims of the research, and written informed consent was obtained prior to inclusion in the study. Confidentiality was strictly maintained, and all procedures adhered to the ethical principles outlined in the Declaration of Helsinki. Throughout the study, efforts were made to maintain methodological consistency and minimize measurement bias. All tools used were previously validated in similar populations, and data collection protocols were standardized across all participants. Regular training and inter-observer reliability checks were conducted for the data collection team to ensure consistency in administering questionnaires and performing clinical measurements. In sum, this cross-sectional study was meticulously designed to explore the relationship between various dimensions of sleep disturbance and cardiovascular risk. By integrating validated assessment tools, a robust sampling framework, and appropriate statistical analyses, the study aims to contribute reliable, locally relevant evidence that can inform preventive and therapeutic strategies targeting modifiable sleep-related risk factors in cardiovascular health.

RESULTS

A total of 422 participants were enrolled in the study, with a mean age of 46.3 years (SD ± 9.1). Among these, 226 (53.6%) were male and 196 (46.4%) were female. The mean BMI was 27.5 kg/m² (SD ± 3.8). Smoking was reported in 134 individuals (31.8%), 157 (37.2%) were hypertensive, and 119 (28.2%) were diabetic. Out of the total participants, 244 individuals (57.8%) were categorized as having poor sleep quality based on PSQI scores ≥5, while 178 (42.2%) had good sleep quality (PSQI <5). The mean Framingham Risk Score (FRS) among those with poor sleep was significantly higher at 14.8 (SD ± 5.3), compared to 9.3 (SD ± 4.1) in the good sleep quality group. High cardiovascular risk (FRS >20%) was present in 61.1% of participants with poor sleep, whereas only 29.8% of those with good sleep showed high risk. Analysis using the Epworth Sleepiness Scale revealed that 242 participants (57.3%) had normal levels of daytime sleepiness (ESS 0–10), while 97 (23.0%) had mild sleepiness (ESS 11–12), and 83 (19.7%) experienced moderate to severe sleepiness (ESS 13–24). A gradual increase in cardiovascular risk was observed across ESS categories, with mean FRS scores of 10.1 (SD ± 4.6), 12.9 (SD ± 5.0), and 15.7 (SD ± 5.8) for normal, mild, and moderate to severe groups, respectively. Correspondingly, the percentage of high-risk FRS increased from 33.5% in the normal group to 66.2% in the moderate to severe group. Regarding the Berlin Questionnaire, 229 participants (54.3%) were classified as high risk for obstructive sleep apnea, and 193 (45.7%) as low risk. Individuals

in the high-risk group had a significantly elevated mean FRS of 15.1 (SD ± 5.5), compared to 9.7 (SD ± 3.9) in the low-risk group. Furthermore, 63.7% of the high-risk participants had FRS values indicating high cardiovascular risk, while only 27.4% in the low-risk group met that threshold.

Table 1: Demographic Characteristics of Participants (n = 422)

Variable	Value
Age (mean ± SD)	46.3 ± 9.1
Male (%)	226 (53.6%)
Female (%)	196 (46.4%)
BMI (mean ± SD)	27.5 ± 3.8
Smokers (%)	134 (31.8%)
Hypertensive (%)	157 (37.2%)
Diabetic (%)	119 (28.2%)

Table 2: Framingham Risk Score by Pittsburgh Sleep Quality Index (PSQI) Categories

Sleep Quality	Participants (n)	Mean FRS Score (±SD)	High Risk FRS (%)
Good (PSQI < 5)	178	9.3 ± 4.1	29.8%
Poor (PSQI ≥ 5)	244	14.8 ± 5.3	61.1%

Table 3: Framingham Risk Score by Epworth Sleepiness Scale (ESS) Categories

ESS Category	Participants (n)	Mean FRS Score (±SD)	High Risk FRS (%)
Normal (0–10)	242	10.1 ± 4.6	33.5%
Mild (11–12)	97	12.9 ± 5.0	47.4%
Moderate to Severe (13–24)	83	15.7 ± 5.8	66.2%

Table 4: Framingham Risk Score by Berlin Questionnaire Categories

Berlin Risk	Participants (n)	Mean FRS Score (±SD)	High Risk FRS (%)
Low Risk	193	9.7 ± 3.9	27.4%
High Risk	229	15.1 ± 5.5	63.7%

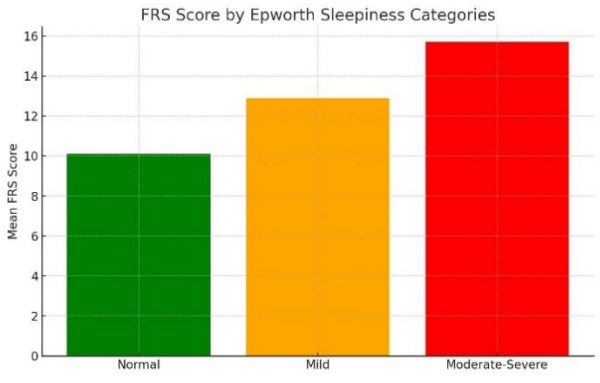

Figure 1 FRS Scores by Epworth Sleepiness Categories

Figure 2Framingham Risk Score by Sleep Quality

DISCUSSION

The present cross-sectional study sought to explore the association between sleep disorders and cardiovascular health among adult patients in a tertiary care setting in Lahore, Pakistan. The findings demonstrated a strong correlation between disturbed sleep patterns—including poor sleep quality, excessive daytime sleepiness, and high risk of sleep-disordered breathing—and elevated Framingham Risk Scores. These outcomes reinforce the growing evidence that sleep disorders may act as independent risk factors for cardiovascular morbidity, particularly in populations with other concurrent risk factors such as obesity, hypertension, and diabetes. Consistent with earlier population-based studies, individuals with poor sleep quality (PSQI ≥5) exhibited significantly higher mean FRS values and a greater proportion of high cardiovascular risk profiles. This trend is in line with previous findings that poor sleep efficiency and duration are associated with increased systemic inflammation, autonomic dysregulation, and metabolic disturbances that contribute to the development of atherosclerosis and cardiovascular disease (14,15). Furthermore, this study's identification of a stepwise increase in cardiovascular risk with escalating scores on the Epworth Sleepiness Scale suggests that daytime somnolence may serve as a clinical marker for underlying cardiovascular compromise, potentially mediated by fragmented nocturnal sleep or undiagnosed obstructive sleep apnea (16,17).

The results also aligned with evidence from the Berlin Questionnaire, where individuals categorized as high risk for sleep-disordered breathing demonstrated substantially higher FRS scores. This correlation likely reflects the cardiovascular strain caused by intermittent nocturnal hypoxia and oxidative stress—hallmarks of obstructive sleep apnea. Such physiological disruptions have been shown to promote sympathetic nervous system overactivity, endothelial dysfunction, and systemic inflammation, thereby accelerating cardiovascular pathology (18,19). One of the study's major strengths lies in its use of validated, widely recognized assessment tools such as the PSQI, ESS, Berlin Questionnaire, and Framingham Risk Score, ensuring reliability and comparability with international literature. Moreover, the diverse sample from a real-world clinical population strengthens the external validity of the results, offering insights applicable to broader South Asian communities with comparable socio-demographic and lifestyle characteristics. However, several limitations must be acknowledged. First, the cross-sectional design precludes any causal inferences. While associations were strong and statistically significant, temporal relationships between sleep disturbances and cardiovascular risk development cannot be established. Second, reliance on self-reported sleep measures introduces the potential for recall bias and misclassification. Objective methods such as actigraphy or polysomnography would have offered more precise evaluations of sleep architecture. Additionally, while confounding factors such as age, BMI, smoking status, and comorbidities were statistically controlled, residual confounding from unmeasured variables such as diet, psychological stress, or physical activity patterns cannot be fully excluded.

Another limitation includes the exclusion of individuals with pre-existing cardiovascular diagnoses, which, while methodologically justified to assess predictive risk, may underestimate the overall burden of disease in relation to sleep disturbances. Furthermore, the study was conducted at a single urban tertiary hospital, limiting generalizability to rural or less medically resourced populations, where sleep health may differ due to environmental and occupational variables. Despite these limitations, the findings hold valuable implications. They underscore the need for incorporating routine sleep assessments in cardiovascular risk stratification protocols. Integrating sleep evaluations into primary care settings could facilitate early identification of individuals at elevated risk and provide a cost-effective avenue for preventive interventions. Moreover, these results advocate for public health strategies aimed at sleep hygiene promotion, particularly in regions with increasing rates of cardiovascular disease and limited access to specialist sleep medicine services (20).

Future research should aim to validate these findings through longitudinal designs that can ascertain temporal causality. Prospective cohort studies incorporating both subjective and objective sleep measures will be instrumental in establishing the directionality of associations. Interventional trials assessing the impact of sleep disorder treatments—such as continuous positive airway pressure (CPAP) for obstructive sleep apnea—on cardiovascular risk profiles may also offer translational benefits (21). In conclusion, this study adds to the growing body of literature suggesting that sleep disorders are significantly associated with increased cardiovascular risk. The robust associations observed across multiple validated sleep assessment tools support the recognition of sleep health as a critical component of cardiovascular disease prevention frameworks. There remains a compelling need to elevate the clinical prioritization of sleep disturbances, both as symptomatic complaints and as potentially modifiable determinants of long-term cardiovascular health.

CONCLUSION

This study demonstrated a strong and consistent association between sleep disorders and increased cardiovascular risk, as measured by the Framingham Risk Score. Findings highlight the clinical value of incorporating sleep assessments into routine cardiovascular evaluations. Addressing sleep disturbances may serve as a practical and cost-effective strategy to mitigate long-term cardiovascular morbidity, particularly in high-risk populations.

AUTHOR CONTRIBUTION

Author	Contribution
Shazia Safdar*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Shaista Ismail*	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Bashir Ullah*	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Tooba Shaikh	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Aqib Dil Awaiz	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Iqra Munir Qazi	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Mashkoor Ahmed Ansari	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Muhammad Naeem	Writing - Review & Editing, Assistance with Data Curation

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