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Association Between Sleep Quality and Coronary Artery Stenosis Severity: A Cross-Sectional Study

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Article

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Abstract

Objective: To investigate the impact of sleep status on the severity of coronary artery stenosis.

Methods: A total of 227 inpatients who underwent coronary angiography in the Department of Cardiology were enrolled. General clinical data, comorbidities, and laboratory results were collected. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), with a score >5 defining sleep disturbance. Participants were categorized based on Gensini scores into: no stenosis (0 points), mild stenosis (1–30 points), moderate stenosis (31–60 points), and severe stenosis (>60 points) groups. Clinical characteristics and sleep parameters were compared among groups, and the influence of sleep status on stenosis severity was analyzed.

Results: Sleep disturbance (PSQI score), body mass index (BMI), age, and C-reactive protein (CRP) were identified as independent influencing factors for the severity of coronary artery stenosis ($P < 0.05$), with odds ratios (95% CI) of 1.156 (1.058–1.265), 1.123 (1.017–1.241), 1.031 (1.010–1.053), and 1.074 (1.027–1.122), respectively. Significant differences were observed in the global PSQI scores and its subdomain scores across the stenosis groups ($P < 0.05$), showing a progressive increase from the no stenosis to the severe stenosis group. Regarding sleep quality grading, the proportion of patients with good sleep quality was highest in the no stenosis group (90.5%), and significantly higher in the mild stenosis group (69.1%) than in the moderate (34.4%) and severe (31.1%) groups. Multiple linear regression analysis revealed that the global PSQI score ($\beta = 2.03$, 95% CI: 0.973–3.088, $P < 0.001$), age ($\beta = 0.364$, 95% CI: 0.045–0.683, $P < 0.05$), and CRP level ($\beta = 0.977$, 95% CI: 0.363–1.59, $P < 0.01$) were positively correlated with the Gensini score. Furthermore, in a model adjusted for all seven PSQI subdomain scores, the sleep quality subscore was independently and positively associated with the Gensini score ($\beta = 18.234$, $P < 0.001$), indicating that poorer subjective sleep quality is linked to more severe coronary stenosis.

Conclusion: Sleep status is closely associated with the severity of coronary artery lesions. Poorer sleep quality correlates with a higher degree of coronary stenosis (as reflected by a higher Gensini score) and an increased likelihood of significant stenosis. Poor sleep quality may serve as a significant predictor for the severity of coronary artery stenosis.

Introduction

Coronary heart disease (CHD) remains one of the leading global causes of mortality and disability, imposing a substantial burden on public health and the economy. According to the 2023 Report on Cardiovascular Health and Diseases in China, the CHD patient population in China is approximately 11.39 million, with the overall prevalence continuing to rise[1]. Despite advancements in treatment modalities, including pharmacotherapy, interventional procedures, and surgery, challenges such as suboptimal therapeutic efficacy and high recurrence rates persist. The pathophysiology of CHD is multifactorial, involving a complex interplay of genetic, environmental, and lifestyle factors[2,3]. Among these, the role of sleep quality, increasingly recognized as a critical determinant of cardiovascular health, is a key area of interest. Sleep disturbances can trigger various physiological alterations, including increased activity of the sympathetic nervous system, elevated inflammatory markers, and dysregulated metabolic processes, all of which may exacerbate coronary artery disease[4]. Recent studies have highlighted an association between poor sleep quality and the severity of coronary artery stenosis, suggesting that sleep disruption may contribute to CHD progression[5,6].

However, the mechanisms underlying the relationship between sleep disturbance and coronary artery disease are not fully elucidated. Existing evidence suggests that inflammation, marked by elevated levels of C-reactive protein (CRP),

may mediate this association, as chronic inflammation is a well-established promoter of atherosclerosis[7,8]. Despite the growing body of evidence, significant gaps remain in understanding how sleep quality influences CHD pathogenesis. Most existing studies feature cross-sectional designs, limiting causal inference. Furthermore, the influence of confounding factors, such as psychological stress and comorbidities, requires further investigation. Addressing these limitations is crucial for clarifying the role of sleep in coronary artery disease and for developing targeted interventions to improve patient outcomes.

This study aimed to investigate the relationship between sleep quality and the severity of coronary artery disease. Given the persistently high prevalence of CHD and its significant public health impact, identifying factors that contribute to its progression is essential. The primary objective was to elucidate whether variations in sleep quality, assessed by the Pittsburgh Sleep Quality Index (PSQI), are associated with the degree of coronary artery stenosis. By employing a retrospective analysis and multiple linear regression techniques, this study controlled for potential confounding variables to provide a clearer understanding of the relationship between sleep disturbance and coronary artery disease severity.

The findings of this study hold potential implications for clinical practice and preventive strategies for CHD. By establishing sleep quality as a modifiable risk factor, healthcare practitioners could develop targeted interventions to improve patients' sleep patterns, potentially slowing the progression of coronary artery disease. Furthermore, this research contributes to the growing body of literature linking lifestyle factors, such as sleep, with cardiovascular health, underscoring the need for a holistic approach to CHD management. Ultimately, the results may lay the groundwork for innovative strategies integrating sleep quality assessment into routine cardiovascular care.

Materials and methods

1.1 Study Population

The study cohort was derived from patients in the Department of Cardiology who were suspected or previously diagnosed with coronary heart disease and scheduled for coronary angiography.

The inclusion criteria were as follows: (1) age ≥ 18 years; (2) conscious and able to communicate effectively in Chinese and complete written forms; (3) hemodynamically stable, without imminent risk of deterioration or life-threatening conditions, and with a confirmed indication for coronary angiography; (4) provision of informed consent, voluntary participation in the clinical study, and completion of laboratory tests, medical history collection, and the Pittsburgh Sleep Quality Index (PSQI), Self-Rating Anxiety Scale (SAS), and Self-Rating Depression Scale (SDS) questionnaires.

Exclusion criteria included: (1) comorbid psychiatric disorders, cognitive impairment, or hearing/visual impairments; (2) severe dysfunction of major organs (e.g., severe heart failure, hepatic failure, renal failure, respiratory failure), severe primary diseases of the endocrine or hematopoietic systems, or malignancies and other chronic consumptive diseases deemed to contraindicate coronary angiography; (3) history of alcohol, opioid, amphetamine, or other substance abuse or dependence; (4) recent history of stroke; (5) pregnancy or lactation; (6) uncorrected hypo-/hyperkalemia, digitalis toxicity, or electrolyte/acid-base imbalances; (7) allergy to iodine-based contrast agents; (8) active severe infectious diseases; (9) central sleep apnea syndrome.

1.2 Study Methods

1.2.1 Data Collection

The collected data encompassed: (a) General information: age, gender, body mass index (BMI), smoking history, alcohol consumption history, marital status, education level, and employment status. (b) Laboratory results: triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), white blood cell count, and C-reactive protein (CRP) levels. (c) Comorbidities: hypertension, heart failure, arrhythmia, diabetes mellitus, hyperlipidemia, and stroke. (d) Scale assessments: SAS, SDS, and PSQI questionnaires. The diagnostic criterion for CHD was based on the American College of Cardiology/American Heart Association guidelines, defined as ≥50% luminal diameter stenosis in any major coronary artery on angiography[9].

1.2.2 Sleep Assessment and Grouping

The PSQI, developed by Daniel J. Buysse et al. at the University of Pittsburgh in 1989, is an instrument for assessing sleep quality in adults[10]. Owing to its simplicity, reliability, and sound psychometric properties, the PSQI has become one of the most widely used tools for sleep quality evaluation globally[11]. It comprises 19 self-rated items and 5 bed-partner-rated items. The self-rated items generate scores across seven components, each ranging from 0 to 3, with higher scores indicating more severe sleep-related problems.

Participants were stratified into groups based on the Gensini score, which quantifies coronary artery stenosis severity: (1) No stenosis group: 0 points; (2) Mild stenosis group: 1–30 points; (3) Moderate stenosis group: 31–60 points; (4) Severe stenosis group: >60 points[12].

1.3 Statistical Analysis

Statistical analyses were performed using SPSS version 26.0. Continuous variables were presented as mean ± standard deviation (SD) or median (interquartile range) [M (P25, P75)], and comparisons among groups were conducted using one-way ANOVA or the Kruskal-Wallis H test, as appropriate. Categorical data were expressed as number (percentage) [n (%)] and compared using the chi-square test. The relationships between the Gensini score and independent variables were examined using simple correlation analysis and multiple linear regression analysis. A two-tailed P-value < 0.05 was considered statistically significant.

1.4 Ethics

The experimental protocol was approved by the Biomedical Ethics Review Committee of Ningxia Hui Autonomous Region People's Hospital (No.2025-LL-029) in accordance with the Declaration of Helsinki.

The study strictly adheres to the Declaration of Helsinki,and all methods were performed in accordance with the relevant guidelines and regulations. Informed consent was waived by the Biomedical Ethics Review Committee of Ningxia Hui Autonomous Region People's Hospital (No.2025-LL-029) due to the retrospective nature of this research.

Results

2.1 Baseline Characteristics

The final analysis included 227 participants, distributed as follows: no stenosis group (n=95, 41.9%), mild stenosis group (n=55, 24.2%), moderate stenosis group (n=32, 14.1%), and severe stenosis group (n=45, 19.8%). The Gensini scores ranged from 0 to 160, with a mean ± SD of 27.13 ± 37.65 and a median [IQR] of 4 [0, 51]. The PSQI scores ranged from 0 to 19, with a mean ± SD of 5.66 ± 4.58 and a median [IQR] of 3 [2, 8]. Based on PSQI categories, 149 participants (65.6%) had good sleep quality, 39 (17.2%) had fair quality, 28 (12.3%) had poor quality, and 11 (4.8%) had very poor quality.

Significant differences in baseline characteristics were observed across the stenosis groups. The no stenosis group was significantly younger (H=49.027, P<0.001) and had a lower BMI (H=34.324, P<0.001) compared to the three stenosis groups. Furthermore, the prevalence of diabetes mellitus, hypertension, heart failure, arrhythmia, and hyperlipidemia was significantly lower in the no stenosis group (all P<0.05; χ^2 values: 18.812, 66.018, 25.909, 7.46, and 41.616, respectively).

2.2 Univariate Analysis

PSQI scores differed significantly among the stenosis groups (H=78.967, P<0.001), with the no stenosis and mild stenosis groups exhibiting significantly lower scores than the moderate and severe stenosis groups. Consistent with the baseline analysis, significant differences were confirmed for age, BMI, and the comorbidities of diabetes, hypertension, heart failure, arrhythmia, and hyperlipidemia.

Among laboratory parameters, significant inter-group differences were found for total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), apolipoprotein A1, white blood cell count, neutrophil count, platelet count, and C-reactive protein (CRP) levels. Specific post-hoc comparisons revealed distinct patterns; for instance, CRP levels were significantly higher in the moderate and severe stenosis groups compared to the no and mild stenosis groups (Table 1).

Table 1. Baseline Characteristics and Laboratory Parameters According to Coronary Artery Stenosis Groups

Risk Factor	No stenosis group n=95	Mild stenosis group n=55	Moderate stenosis group n=32	Severe stenosis group n=45	$H/\chi^2/F$	*P*
Age (years)	41(35,59)	58(48,66)	67.5(52,72.75)	63(56.5,71.5)	49.027	**<0.001**
BMI（kg/m^2）	22.58 (20.6,24.44)	24.49 (22.36,25.59)	24.34 (23.34,28.45)	24.98 (23.03,28.03)	34.324	**<0.001**
PSQI	3(2,3)	3(3,7)	8(5,12.75)	8(5,13)	78.967	**<0.001**
Male,n(%）	57(60)	30(54.5)	23(71.9)	27(60)	2.558	0.465
Smoking history, n (%)	44(46.3)	21(38.2)	16(50)	24(53.3)	2.53	0.470
Alcohol History, n (%)	14(14.7)	6(10.9)	6(18.8)	6(13.3)	1.087	0.780
Employed, n (%)	46(48.4)	28(50.9)	14(43.8)	19(42.2)	0.96	0.811
Marital Status (Married), n (%)	89(93.7)	53(96.4)	28(87.5)	42(93.3)	2.565	0.459
ducation (>High School), n (%)	34(35.8)	15(27.3)	14(43.8)	10(22.2)	5.183	0.159
Diabetes Mellitus, n (%)	10(10.5)	14(25.5)	14(43.8)	15(33.3)	18.812	**<0.001**
Hypertension, n (%)	7(7.4)	25(45.5)	21(65.6)	30(66.7)	66.018	**<0.001**
History of Stroke, n (%)	1(1.1)	4(7.3)	1(3.1)	3(6.7)	4.967	0.112
Heart Failure, n (%)	2(2.1)	7(12.7)	8(25)	14(31.1)	25.909	**<0.001**
Arrhythmia, n (%)	4(4.2)	4(7.3)	4(12.5)	8(17.8)	7.46	**0.049**
Hyperlipidemia, n (%)	3(3.2)	24(43.6)	11(34.4)	18(40)	41.616	**<0.001**
Total Cholesterol, mmol/L	4(3.3,4.5)	4.6(3.7,5.6)	3.6(2.93,5.28)	4.7(3.6,5.2)	16.603	**0.001**
Triglycerides, mmol/L	1.5(1,2.06)	1.45(0.9,2.3)	1.3(0.9,2.34)	1.72(1.19,2.24)	3.317	0.345
HDL-C, mmol/L	0.9(0.8,1.1)	1.1(0.9,1.3)	1(0.8,1.18)	1(0.9,1.2)	11.179	**0.011**
LDL-C, mmol/L	2.3±0.72	2.6±0.91	2.28±0.95	2.64±0.78	2.96	**0.033**
Apolipoprotein A1, mmol/L	1.1(1,1.3)	1.2(1.1,1.4)	1.1(1,1.2)	1.2(1,1.3)	10.2	**0.017**
Serum Creatinine,	68(57,81)	63(56,77)	69.5(58.5,80)	69(58,85.5)	1.551	0.670

μmol/L						
White Blood Cell Count, ×10⁹/L	5.46(4.61,6.57)	6.21(4.95,7.1)	6.53(4.84,8.31)	6.59(5.4,8.65)	16.393	**0.001**
Neutrophil Count, ×10⁹/L	3.12(2.55,3.87)	3.59(2.77,4.67)	4(2.89,5.06)	4.09(3.3,6.16)	22.714	**<0.001**
Lymphocyte Count, ×10⁹/L	1.84±0.75	1.94±0.69	1.87±0.80	1.79±0.68	0.376	0.770
Platelet Count, ×10⁹/L	185(161,229)	220(179,250)	214.5(173.25,254)	213(177,245.5)	9.828	**0.020**
C-Reactive Protein, mg/L	1.3(0.6,1.9)	1.3(0.5,2.3)	3.7(1.29,7.71)	2.5(0.93,6.44)	30.578	**<0.001**
D-Dimer, ng/ml	0.06(0.02,0.16)	0.05(0.02,0.13)	0.07(0.04,0.17)	0.09(0.03,0.23)	3.382	0.336

Note: The level of statistical significance was set at P < 0.05. Pittsburgh Sleep Quality Index（PSQI）Body Mass Index（BMI）High-density Lipoprotein Cholesterol（HDL-C）Low-density Lipoprotein Cholesterol（LDL-C.

2.3 Multivariate Analysis

Variables identified as significant in the univariate analysis were included as independent variables in a multivariate logistic regression model. The results demonstrated that sleep disturbance (PSQI score), body mass index (BMI), age, and CRP level were independent influencing factors for the severity of coronary artery lesions (all P < 0.05). The odds ratios (OR) with 95% confidence intervals (CI) were 1.156 (1.058–1.265) for PSQI score, 1.123 (1.017–1.241) for BMI, 1.031 (1.010–1.053) for age, and 1.074 (1.027–1.122) for CRP (Table 2).

Table 2. Multivariate Logistic Regression Analysis of Factors Associated with the Severity of Coronary Artery Disease

Risk Factor	Estimate	*S.E.*	*Wald*	*OR*	*P*	95%*C.I*	
						Lower	*Upper*
Age (years)	0.031	0.011	8.096	1.031	**0.004**	1.010	1.053
BMI（kg/m^2）	0.116	0.051	5.236	1.123	**0.022**	1.017	1.241
PSQI	0.145	0.046	10.16	1.156	**0.001**	1.058	1.265
SAS Score, points	0.018	0.027	0.45	1.018	0.502	0.966	1.074
SDS Score, points	-0.021	0.027	0.615	0.979	0.433	0.928	1.033
Total Cholesterol, mmol/L	-0.023	0.234	0.01	0.977	0.92	0.617	1.547
HDL-C, mmol/L	0.258	0.578	0.199	1.294	0.655	0.417	4.019
LDL-C, mmol/L	0.156	0.291	0.286	1.169	0.593	0.661	2.065
Apolipoprotein A1, mmol/L	-0.559	0.644	0.753	0.572	0.385	0.162	2.020
White Blood Cell Count, ×10⁹/L	0.063	0.176	0.128	1.065	0.72	0.754	1.505
Neutrophil Count, ×10⁹/L	0.034	0.205	0.028	1.035	0.867	0.692	1.548
Platelet Count, ×10⁹/L	0.002	0.003	0.376	1.002	0.54	0.996	1.007
C-Reactive Protein, mg/L	0.071	0.022	10.06	1.074	**0.002**	1.027	1.122
Diabetes Mellitus, n (%)	0.296	0.339	0.764	1.344	0.382	0.692	2.612
Hypertension, n (%)	0.712	0.375	3.597	2.038	0.058	0.976	4.250
Heart Failure, n (%)	-0.031	0.44	0.005	0.969	0.943	0.409	2.298
Arrhythmia, n (%)	0.232	0.495	0.219	1.261	0.64	0.478	3.323
Hyperlipidemia, n (%)	0.4	0.383	1.087	1.492	0.27	0.703	3.161

Note: The level of statistical significance was set at P < 0.05；Test of Parallel Lines：x^2=40.967，p=0.342；Pittsburgh Sleep Quality Index（PSQI）；Body Mass Index（BMI）；self-rating anxiety scale（SAS）；self-rating depression scale（SDS）；High-density Lipoprotein Cholesterol（HDL-C）；Low-density Lipoprotein Cholesterol（LDL-C.

2.4 Analysis of Sleep Indicators by Stenosis Severity

Significant differences were observed in the global PSQI score and all its subdomain scores across the coronary stenosis severity groups (all P < 0.05). A trend of increasing scores (worsening sleep parameters) was evident from the no stenosis to the severe stenosis group. Regarding sleep quality grading, the proportion of participants with good sleep quality was highest in the no stenosis group (90.5%), and significantly higher in the mild stenosis group (69.1%) than in the moderate (34.4%) and severe (31.1%) stenosis groups (Table 3).

Table 3. Comparison of Sleep Quality Indicators Across Coronary Artery Stenosis Severity Groups

Risk Factor	No stenosis group n=95	Mild stenosis group n=55	Moderate stenosis group n=32	Severe stenosis group n=45	H/χ^2	*P*
PSQI	3(2,3)	3(3,7)	8(5,12.75)	8(5,13)	78.967	**<0.001**
Sleep Quality	0(0,0)	0(0,1)	1(1,2)	1(1,2)	80.571	**<0.001**
Sleep Latency	1(1,1)	1(1,2)	2(1,2.75)	2(1,3)	24.672	**<0.001**
Sleep Duration	0(0,0)	0(0,1)	2(1,2)	2(0,2)	71.78	**<0.001**
Sleep Efficiency	0(0,0)	0(0,1)	1(0,2)	1(0,3)	50.834	**<0.001**
Sleep Disturbances	0(0,1)	1(0,1)	1(1,2)	1(1,2)	31.98	**<0.001**
Use of Sleep Medications	0(0,0)	0(0,0)	0(0,0)	0(0,0)	14.055	**0.003**
Daytime Dysfunction	1(1,1)	1(1,1)	2(1,2)	2(0.5,2)	23.174	**<0.001**
Sleep Quality Classification（PSQI score）						
Good (≤ 5)	86(90.5)	38(69.1)	11(34.4)	14(31.1)	64.033	**<0.001**
Fair (6-10)	6(6.3)	7(12.7)	11(34.4)	15(33.3)	23.548	**<0.001**
Poor (11-15)	2(2.1)	8(14.5)	9(28.1)	9(20)	19.266	**<0.001**
Very Poor (16-21)	1(1.1)	2(3.6)	1(3.1)	7(15.6)	14.538	**0.002**

Note: The level of statistical significance was set at P < 0.05.

2.5 Multiple Linear Regression Analysis

After adjusting for potential confounders including gender, age, BMI, smoking, alcohol use, occupation, marital status, education level, comorbidities (diabetes, hypertension, stroke history, heart failure, arrhythmia, hyperlipidemia), and relevant laboratory indices, multiple linear regression analysis showed that age (β = 0.364, 95% CI: 0.045–0.683, P < 0.05), PSQI score (β = 2.03, 95% CI: 0.973–3.088, P < 0.001), and CRP level (β = 0.977, 95% CI: 0.363–1.59, P < 0.01) remained significantly correlated with the Gensini score. The derived linear regression equation was: Y = 0.364 X1 + 2.03 X2 + 0.977 X3 -20.767 X4 -29.95. These results indicate that older age, higher PSQI score (poorer sleep quality), and elevated CRP levels are independent risk factors associated with a higher degree of coronary artery stenosis (Table 4).

Table 4. Determinants of Coronary Artery Stenosis Severity (Gensini Score): A Multiple Linear Regression Analysis

Risk Factor	*β*	95%*CI*	*t*	*P*
Female	-4.965	-15.019~5.09	-0.974	0.331
Age (years)	0.364	0.045~0.683	2.249	**0.026**
BMI（kg/m2）	1.32	-0.186~2.826	1.729	0.085
PSQI	2.03	0.973~3.088	3.787	**<0.001**
Smoking history	4.175	-5.141~13.49	0.884	0.378
Alcohol History	5.29	-7.455~18.035	0.818	0.414
Employed	2.621	-6.53~11.773	0.565	0.573
Marital Status (Married)	-3.68	-20.429~13.07	-0.433	0.665
ducation (>High School)	-1.807	-11.945~8.331	-0.352	0.726
Diabetes Mellitus	8.976	-1.545~19.497	1.682	0.094
Hypertension	8.643	-2.989~20.274	1.465	0.144
History of Stroke	5.006	-16.665~26.678	0.456	0.649
Heart Failure	1.559	-12.529~15.647	0.218	0.827
Arrhythmia	2.34	-12.854~17.534	0.304	0.762
Hyperlipidemia	0.261	-11.64~12.162	0.043	0.966
Total Cholesterol, mmol/L	0.344	-7.265~7.952	0.089	0.929
Triglycerides, mmol/L	0.351	-2.905~3.607	0.213	0.832
HDL-C, mmol/L	3.605	-13.626~20.836	0.413	0.68
LDL-C, mmol/L	1.18	-7.594~9.954	0.265	0.791
Apolipoprotein A1, mmol/L	-13.125	-30.922~4.672	-1.454	0.147
Serum Creatinine, μmol/L	-0.008	-0.034~0.018	-0.612	0.541
White Blood Cell Count, ×10⁹/L	6.442	-11.447~24.332	0.71	0.478
Neutrophil Count, ×10⁹/L	-3.564	-22.173~15.045	-0.378	0.706
Lymphocyte Count, ×10⁹/L	-6.496	-27.361~14.369	-0.614	0.54
Platelet Count, ×10⁹/L	0.024	-0.061~0.109	0.552	0.581
C-Reactive Protein, mg/L	0.977	0.363~1.59	3.14	**0.002**
D-Dimer, ng/ml	0.005	-0.063~0.074	0.158	0.875

Note: The level of statistical significance was set at P < 0.05. Pittsburgh Sleep Quality Index（PSQI）Body Mass Index（BMI）High-density Lipoprotein Cholesterol（HDL-C）Low-density Lipoprotein Cholesterol（LDL-C.

2.6 Association Between PSQI Subdomains and Gensini Score

In an analysis adjusted for the scores of the other six PSQI subdomains, the sleep quality subscore was independently and positively associated with the Gensini score (β = 18.234, P < 0.001). This suggests that worse

subjective sleep quality is specifically associated with more severe coronary artery stenosis, independent of other sleep-related components (Table 5).

Table 5. Association Between PSQI Component Scores and Coronary Artery Stenosis (Gensini Score): Correlation and Adjusted Analysis

Factor	Simple Correlation Analysis		Linear regression analysis	
	r	*P*	*β*	*P*
Sleep Quality	0.614	**<0.001**	18.234	**<0.001**
Sleep Latency	0.314	**<0.001**	0.748	0.788
Sleep Duration	0.525	**<0.001**	7.549	0.069
Sleep Efficiency	0.483	**<0.001**	-2.581	0.456
Sleep Disturbances	0.382	**<0.001**	2.673	0.522
Use of Sleep Medications	0.261	**<0.001**	-3.642	0.36
Daytime Dysfunction	0.295	**<0.001**	-1.36	0.672

Note: The level of statistical significance was set at P < 0.05.

Discussion

Coronary artery disease has garnered significant attention due to its substantial public health impact, characterized by a complex etiology and diverse clinical manifestations[13]. Current therapeutic strategies, including pharmacotherapy, interventional procedures, and surgery, are often associated with suboptimal efficacy and high recurrence rates. This study integrated multiple influencing factors to investigate the relationship between sleep quality and the severity of coronary artery stenosis. Our results demonstrated a significant association between sleep disturbance and the severity of coronary artery disease, indicating that sleep quality is an important factor in the pathogenesis and progression of CHD. These findings suggest that clinicians should prioritize sleep quality assessment in both the prevention and management of coronary artery disease, potentially offering new avenues for improving patient prognosis and quality of life. By elucidating the interaction between sleep disturbance and cardiovascular health, this study lays the groundwork for future development of targeted interventions addressing sleep quality as a modifiable risk factor.

We found a positive correlation between the global PSQI score and the Gensini score, indicating a significant association between sleep quality and coronary artery disease severity. This relationship suggests the influence of sleep quality as a molecular determinant on the phenotypic expression of coronary artery disease. Poor sleep quality may promote the progression of coronary lesions by exacerbating inflammatory responses, such as elevating C-reactive protein (CRP) levels. The positive correlation between CRP levels and the Gensini score implies that sleep disturbance may aggravate coronary artery disease through a pro-inflammatory state. Furthermore, the interconnection between sleep quality and established risk factors like age and body mass index underscores the multidimensional nature of coronary artery disease pathogenesis. Improving sleep quality could thus emerge as a novel strategic component in coronary artery disease management. Optimizing sleep hygiene may not only improve sleep itself but also mitigate inflammatory responses, ultimately reducing the severity of coronary lesions.

Our findings suggest a potential mechanism whereby sleep quality influences coronary stenosis severity through inflammatory pathways. Deteriorating sleep quality, reflected by higher PSQI scores, has been linked to elevated levels of inflammatory markers like CRP[14], which plays a pivotal role in cardiovascular pathology[15]. The positive correlation we observed between CRP levels and the Gensini score suggests that chronic inflammation may be a crucial pathway through which sleep disturbance exacerbates coronary artery disease. Notably, advancing age is often accompanied by declining sleep quality, and these factors may synergistically increase cardiovascular risk in elderly patients. Future research should further explore how improving sleep quality might attenuate the progression of coronary artery disease by suppressing specific inflammatory pathways (e.g., the NF-κB signaling pathway), thereby providing a rationale for therapeutic strategies targeting the sleep-inflammation axis.

In the pharmacological management of coronary artery disease, the bidirectional effects of medications on sleep warrant consideration. For instance, beta-blockers may modulate sleep quality by influencing autonomic nervous function, while angiotensin-converting enzyme inhibitors (ACEIs) might indirectly ameliorate sleep disturbance-related cardiovascular conditions by suppressing the renin-angiotensin system and reducing inflammation. It is noteworthy that although sedative-hypnotic medications can improve sleep, they may exert adverse effects on the cardiovascular system, necessitating a careful risk-benefit assessment. Therefore, sleep evaluation should be integrated when formulating pharmacotherapeutic plans for coronary artery disease. Personalized medication selection (e.g., opting for β1-selective blockers with lesser impact on sleep) may achieve synergistic benefits for both cardiovascular outcomes and sleep quality.

This study has several limitations. The relatively small sample size may limit the generalizability of the findings. Its single-center design lacks external validation. The retrospective nature carries an inherent risk of bias. Furthermore, the absence of experimental data limits in-depth mechanistic exploration. Future studies should employ prospective cohort designs integrated with multi-omics analyses (e.g., investigating networks of sleep-related miRNAs and inflammatory cytokines) and conduct interventional clinical trials (e.g., examining the effect of cognitive behavioral therapy on coronary plaque stability) to establish causality. Concurrently, the differential responses of various coronary artery disease subtypes to sleep interventions need to be explored.

The evidence from this study underscores the independent correlation between sleep quality and coronary artery disease severity, positioning sleep disturbance as a risk factor comparable in importance to age and CRP levels. Improving sleep quality may represent a novel target for retarding the progression of coronary artery disease. We recommend incorporating PSQI assessment into routine cardiovascular risk evaluation in clinical practice and developing individualized management strategies that integrate pharmacotherapy, cognitive-behavioral therapy for insomnia, and monitoring via wearable devices. Our findings provide a crucial foundation for reducing cardiovascular event risk and enhancing patient quality of life through sleep health management.

Declarations

Data availability

The datasets generated and/or analysed during the current study are not publicly available due to the data privacy involved. but are available from the corresponding author on reasonable request.

Competing interests

The authors declare no competing interests.

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