Baseline coronary artery stenosis severity is an independent predictor of subsequent poor sleep quality in patients with acute coronary syndrome

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Aim **To investigate the relationship between coronary artery lesion severity determined using the baseline SYNTAX score and sleep problems that might occur after discharge determined using the Pittsburgh Sleep Quality Index (PSQI).**

Methods **This prospective study included patients with first acute coronary syndrome (ACS) who underwent percutaneous coronary angiography between February 2019 and August 2019. The severity of coronary artery stenosis was classified according to coronary angiography and SYNTAX scores. Patients were grouped as those with a SYNTAX score of ≤22 and >22. Sleep quality after discharge was classified according to the PSQI. PSQI ≤5 represented good sleep quality, and PSQI >5 represented poor sleep quality. Univariate and multivariate logistic regression was used to investigate the relationship between sleep quality and coronary artery stenosis severity.**

Results **A total of 424 patients were included in the study. Of these, 294 (69.34%) had a SYNTAX score of ≤22 and 130 (30.66%) had a SYNTAX score of >22. The mean age of all patients was 60.37 ± 12.23 years, 59.69 ± 11.85 years in the SYNTAX ≤22 groups and 61.90 ± 12.98 years in the SYNTAX >22 group ($P = 0.086$). The majority (78.54%) of the patients were male and there was no significant difference between the SYNTAX ≤22 group and the SYNTAX >22 group in terms of sex distribution ($P = 0.383$). According to the univariate logistic regression analysis, age ($P = 0.014$), diabetes ($P = 0.027$), left ventricular ejection fraction ($P = 0.001$), estimated glomerular filtration rate ($P = 0.039$), creatine kinase MB ($P = 0.040$) and SYNTAX scores ($P < 0.001$) were significantly associated with high PSQI global scores (>5). However, according to the multivariate logistic regression analysis results, high (>22) SYNTAX scores were the only factor independently associated with the high (>5) PSQI global scores [odds ratio, 3.477; 95% confidence interval (CI), (2.190–5.522); $P < 0.001$]. Complete revascularization group had significantly higher sleep latency and sleep duration time, sleep efficiency and the percentage of patients with PSQI global score of ≤5 than the incomplete revascularization group ($P < 0.001$ for all).**

Conclusion **Among patients with ACS, those with high SYNTAX scores should be monitored more carefully for sleep disorders that may occur later.** ***Coron Artery Dis*** **35: 299–308**

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Keywords: acute coronary syndrome, independent risk factor, Pittsburgh Sleep Quality Index, SYNTAX

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Introduction

Almost half of all deaths from cardiovascular diseases (CVD), the leading cause of death worldwide [1], are due to ischemic heart disease [2]. Acute coronary syndrome (ACS) is a heterogeneous entity of CVD and is the common name for unstable angina pectoris (USAP), ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI) [3]. Several well-known risk factors for ACS have been identified, including advanced age, smoking, hyperlipidemia, hyperglycemia high BMI, lack of physical activity, hypertension and exposure to air pollution [4,5]. Research has also shown that sleep duration and sleep quality are associated with an increased risk of developing coronary artery disease (CAD), acute myocardial infarction (AMI) or death [5–9].

Sleep disorders are increasingly common in the general population and are associated with many other diseases and socioeconomic situations [5]. They were also associated with poor health consequences such as CVD, respiratory diseases, gastrointestinal diseases, overall mortality and poor prognosis [5,10]. Research has shown that insufficient or poor-quality sleep can increase

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physical and mental fatigue, increase the risk of depression and hinder the ability of patients with CAD to perform daily activities [11,12]. Even though depression caused by sleep disorders can be controlled, poor sleep quality in patients with CAD has been found to be associated with a higher risk of AMI, heart-related surgeries and death [13]. In clinical settings, often with time and resource constraints, the Pittsburgh Sleep Quality Index (PSQI) is commonly used to evaluate sleep problems in patients [14]. Although it is a subjective tool, subsequent researchers have confirmed that the PSQI best represents multiple factors, including sleep efficiency, perceived sleep quality and daily disturbances, and has been associated with different health-related quality of life domains in patients with CAD [10,15].

The SYNergy between percutaneous intervention with TAXus drug-eluting stents and cardiac surgery (SYNTAX) score was developed to evaluate the complexity of coronary artery lesions quantitatively according to the lesion location, severity, bifurcation, calcification and other anatomic characteristics [16]. Many studies have shown that the SYNTAX score is a prognostic marker for short and long-term clinical outcomes [17,18]. However, the relationship between baseline SYNTAX scores and postdischarge sleep disorders has been investigated in a very limited number of studies [19,20]. Therefore, in this study, we aimed to investigate the relationship between coronary artery lesion severity determined using baseline SYNTAX scores and sleep problems that might occur after discharge determined using the PSQI.

Material and methods

Setting and ethics

This prospective study, some of the data which were obtained retrospectively, was conducted in the Cardiology Department of Ankara City Hospital Health Application and Research Center, Ankara, Turkey.

The protocol for the study was approved by the local ethics committee (Date: 15.08.2019, No: 64). The study was designed respecting the expected ethical aspects and performed according to the Declaration of Helsinki and its later amendments. Verbal or written informed consent was obtained from all participants.

Participants

The study included patients with first ACS who were hospitalized, underwent percutaneous coronary angiography and were treated with percutaneous coronary intervention in our inpatient ward or coronary ICU and then discharged, between February 2019 and August 2019. Patients aged under 18 years, those diagnosed as having heart failure or recurrent AMI, patients with any infection within 1 month before the diagnosis of ACS, those with a left ventricular ejection fraction (LVEF) of <35%, those who could not undergo percutaneous coronary angiography for any reason, those with severe liver and renal dysfunction, patients with a diagnosis or history of cancer, patients with known valvular heart disease, sleep medication users, patients with proven obstructive sleep apnea (OSA) with polysomnography, and those with known any psychiatric or sleep disorders were excluded from the study.

Data collection

The patients' age, sex, BMI, comorbidity and drug use information, laboratory results, information regarding the diagnosis of ACS and the period of hospitalization, including whether they had atrial fibrillation, ACS type, LVEF information and percutaneous coronary angiography images were obtained from the hospital computer registers and patients' charts.

After obtaining ethical approval, the PSQI questionnaire was completed by phone or at the time of the first follow-up examination.

Acute coronary syndrome management

The diagnosis, classification and treatment management of ACS were performed according to the current European Society of Cardiology guidelines [21,22]. ACS subtypes were identified as USAP, STEMI and NSTEMI depending on the electrocardiography and the level of cardiac biomarkers. STEMI is defined by permanent electrocardiographic ST elevation with characteristic symptoms of myocardial ischemia, followed by the release of specific cardiac biomarkers. Positive ST-segment elevation was defined as ST elevation at the J point in at least two adjacent leads of 2 mm (0.2 mv) in men or 1.5 mm (0.15 mv) in women in leads v2–v3 or of 1 mm (0.1 mv) in other adjacent chest leads or limb leads [22,23]. USAP and NSTEMI were identified through electrocardiographic ST-segment depression or prominent T-wave inversion or positive cardiac enzymes in the absence of ST-segment elevation and an appropriate clinical setting (chest discomfort or anginal equivalent) for ACS. The presence of positive cardiac enzymes was defined as NSTEMI and negative cardiac enzymes as USAP [24].

Baseline LVEF values were obtained from echocardiography results performed and recorded before angiography.

The baseline and postprocedure SYNTAX scores were calculated by viewing the patients' coronary angiography images saved on the computer system and using the website http://www.syntaxscore.com/calculator/start.htm. Lesions with >50% diameter stenosis in vessels ≥1.5 mm in diameter were scored using the SYNTAX score algorithm described previously. A higher score indicates more severe vascular disease. Baseline severity was classified according to coronary angiography and baseline SYNTAX score as ≤22 and >22. Patients with postprocedure SYNTAX score = 0 were grouped as patients with complete revascularization, and patients with postprocedure

SYNTAX score >0 were grouped as patients with incomplete revascularization.

Laboratory analysis

All laboratory analyses were performed locally in certified laboratories using routine and calibrated devices in accordance with the manufacturer's recommendations. The following parameters, which were studied from the first venous blood taken after the patients were hospitalized for the diagnosis of ACS and before any intervention were included in the study: hemoglobin, creatinine, uric acid, C-reactive protein, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglyceride, fasting blood glucose, troponin, creatine kinase MB (CK-MB) levels and platelet and leukocyte counts. The first troponin level after ACS diagnosis (troponin fist) and the highest troponin level during hospitalization (troponin peak) were included.

The estimated glomerular filtration rate (eGFR; in ml/min per 1.73 m^2) was calculated for each cohort using the Modification of Diet in Renal Disease Study equation [25].

Sleep-related instruments

The sleep quality and sleep disturbance of the patients were evaluated using the PSQI questionnaire. This questionnaire was completed either through telephone interviews or face-to-face interviews at the first follow-up visits after ethical approval was obtained. This scale includes the following seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, sleep medication use and daytime dysfunction. There are a total of 19 questions in the questionnaire and the total score ranges from 0 to 21. A higher score indicates worse sleep quality. Those with a PSQI global score of >5 were categorized as having poor sleep quality and those with a score of ≤5 as having good sleep quality [14].

Sleep latency, sleep duration and sleep efficiency, which were calculated using the data obtained from the PSQI questions, were investigated as separate variables. Sleep latency was obtained in minutes as the time between going to bed and falling asleep and the results were divided into the following four categories: <15, 16–30, 31–60 and >60 min. Sleep duration was obtained as total sleep time in hours and the results were divided into the following four categories: >7, 6–7, 5–6 and <5 h. Sleep efficiency was obtained as the ratio of sleep duration to total time spent in bed multiplied by 100 and the results were divided into the following four categories: >85%, 75–84%, 65–74% and <65%.

Statistical analysis

All analyses were performed using the IBM SPSS Statistics for Windows, Version 25.0 software (IBM Corp., Armonk, New York, USA). Histogram and Q–Q plots were used to evaluate the distribution of continuous variables. Data are given as mean ± SD or median (1st quartile–3rd quartile) for continuous variables according to the normality of distribution and as frequency (percentage) for categorical variables. Normally distributed continuous variables were analyzed using Student's *t*-test. Non-normally distributed continuous variables were analyzed using the Mann–Whitney *U* test. Categorical variables were analyzed using the Chi-square test or Fisher's exact test. Logistic regression analyses were performed to determine significant factors independently associated with high (>5) PSQI global scores. Variables were analyzed using univariate regression analysis and statistically significant variables were included in the multivariate analysis. *P* values of less than 0.05 were accepted as statistically significant.

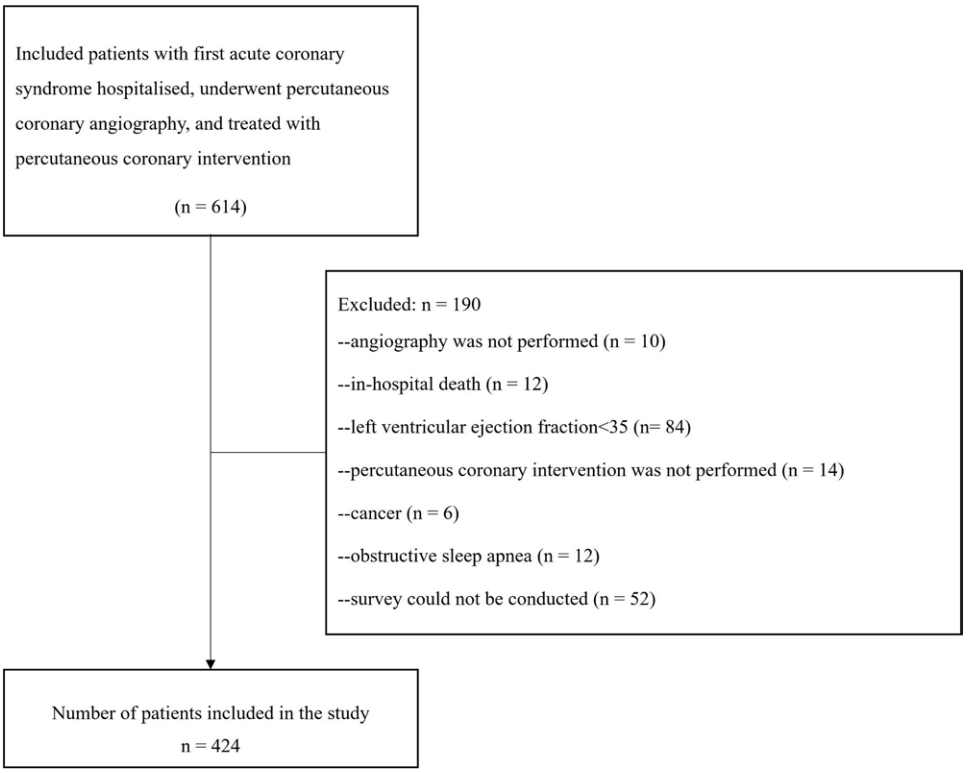
Results

A total of 614 patients were evaluated for eligibility. 190 patients were not included in the study because they met the exclusion criteria. A total of 424 patients were included in the study. A detailed flowchart of the study is presented in Fig. 1. Of these, 294 (69.34%) had a SYNTAX score of ≤*22* and 130 (30.66%) had a SYNTAX score of >*22*. The mean age of all patients was 60.37 ± 12.23 years, 59.69 ± 11.85 years in the SYNTAX ≤ *22* groups and 61.90 ± 12.98 years in the SYNTAX > *22* group ($P = 0.086$). The majority (78.54%) of the patients were male and there was no significant difference between the SYNTAX ≤ *22* group and the SYNTAX > *22* group in terms of sex distribution ($P = 0.383$). The percentage of patients with diabetes ($P = 0.032$) and median CK-MB levels ($P = 0.007$) in the SYNTAX > *22* groups were significantly higher than in the SYNTAX ≤ *22* group. The mean LVEF of the SYNTAX ≤ *22* group was significantly higher than that of the SYNTAX > *22* group ($P < 0.001$). The median postprocedure SYNTAX score ($P < 0.001$) and the percentage of patients with incomplete revascularization ($P < 0.001$) of the SYNTAX > *22* group were significantly higher than that of the SYNTAX ≤ *22* group (Table 1).

Although the sleep latency of the SYNTAX ≤ *22* group was significantly lower than that of the SYNTAX > *22* group ($P < 0.001$), sleep duration ($P < 0.001$) and sleep efficiency ($P < 0.001$) were significantly higher. The median PSQI global score of the SYNTAX ≤ *22* group was significantly lower than that of the SYNTAX > *22* group ($P < 0.001$). The percentage of patients with a PSQI global score of ≤5 was significantly higher in the SYNTAX ≤ *22* group compared with the SYNTAX > *22* group (Table 2, Fig. 2).

There were no significant differences between patients with NSTEMI and STEMI in terms of sleep latency ($P = 0.7759$), sleep duration ($P = 0.075$), sleep efficiency

Fig. 1



Flowchart showing patient participation in the study.

(P = 0.067) and PSQI global score ≤5 (P = 0.487) and >5 (P = 0.221) (Table 3).

Complete revascularization group had significantly higher sleep latency and sleep duration time, sleep efficiency and the percentage of patients with PSQI global score of ≤5 than the incomplete revascularization group (P < 0.001 for all) (Table 4, Fig. 3).

According to the univariate logistic regression analysis, age (P = 0.014), diabetes (P = 0.027), LVEF (P = 0.001), eGFR (P = 0.039), CK-MB (P = 0.040), SYNTAX scores (P < 0.001) and incomplete revascularization (P < 0.001) were significantly associated with high PSQI global scores (>5). However, according to the multivariate logistic regression analysis results, high (>22) SYNTAX scores [odds ratio (OR), 2.220; 95% confidence interval (CI), (1.281–3.849); P = 0.004] and incomplete revascularization [OR, 2.189; 95% CI, (1.291–3.710); P = 0.004] were the only factors independently associated with the high (>5) PSQI global scores (Table 5).

Discussion

Recent studies have demonstrated an important link between sleep disorders and CVD such as chronic heart failure and CAD [26]. The majority of cardiac patients experience decreased sleep quality [27]. However, despite being a very well-known symptom in these patients, it has not yet been investigated routinely and has not been incorporated into clinical practice guidelines. Although predictability of the risk of sleep disorders after ACS is important, this issue has not been adequately researched. The current study revealed that although age, diabetes, LVEF, eGFR, CK-MB, SYNTAX scores and incomplete revascularization were significantly associated with high PSQI global scores (>5) in the univariate analysis, high baseline SYNTAX scores (>22) and incomplete revascularization were the only independent risk factors for subsequent poor sleep quality.

Calcaianu *et al.* [28] performed polysomnography on patients with ACS 2 months after AMI and used the apneic coefficient (AC), defined as the ratio between apnea index and apnea-hypopnea index (AHI), as the main outcome. The results showed that the patients with a higher AC (≥37% vs. <37%) had higher baseline levels of troponin-I, N-terminal prohormone brain natriuretic peptide and higher SYNTAX scores, lower LVEF, and were more likely to have a STEMI. Yılmaz *et al.* [29] investigated the relationship between angina severity and sleep quality in patients who underwent coronary artery bypass graft (CABG) surgery. The study showed that patients who had recently had AMI in the 2 weeks prior to CABG were more likely to experience sleep disturbances

Table 1 Summary of demographics, laboratory measurements and acute coronary syndrome-related features with regard to SYNTAX score

		SYNTAX score		
	Total ($n = 424$)	≤22 ($n = 294$)	>22 ($n = 130$)	*P* value
Age (years)	60.37 ± 12.23	59.69 ± 11.85	61.90 ± 12.98	0.086
Sex				
Male	333 (78.54%)	227 (77.21%)	106 (81.54%)	0.383
Female	91 (21.46%)	67 (22.79%)	24 (18.46%)	
BMI (kg/m^2)	27.76 ± 3.89	27.89 ± 3.99	27.44 ± 3.64	0.273
Hypertension	173 (40.80%)	127 (43.20%)	46 (35.38%)	0.131
Diabetes	102 (24.06%)	62 (21.09%)	40 (30.77%)	**0.032**
Stroke	6 (1.42%)	3 (1.02%)	3 (2.31%)	0.376
Atrial fibrillation	7 (1.65%)	5 (1.70%)	2 (1.54%)	0.999
Smoking	147 (34.67%)	110 (37.41%)	37 (28.46%)	0.074
Diuretics use	57 (13.44%)	37 (12.59%)	20 (15.38%)	0.532
Acute coronary syndrome				
NSTEMI	126 (29.72%)	92 (31.29%)	34 (26.15%)	0.425
STEMI	228 (53.77%)	152 (51.70%)	76 (58.46%)	
USAP	70 (16.51%)	50 (17.01%)	20 (15.38%)	
LVEF (%)	47.71 ± 8.45	48.91 ± 8.23	44.98 ± 8.32	**<0.001**
Hemoglobin (g/dl)	14.11 ± 1.83	14.21 ± 1.72	13.89 ± 2.04	0.125
Platelet (×10^3)	242 (206–290)	247 (206–295)	235 (206–281)	0.425
WBC (×10^3)	10.85 ± 3.19	10.80 ± 3.10	10.97 ± 3.40	0.621
Creatinine (mg/dl)	0.90 (0.77–1.05)	0.89 (0.77–1.01)	0.90 (0.77–1.10)	0.159
eGFR (ml/min per 1.73 m^2)	87.89 ± 23.41	89.20 ± 22.24	84.93 ± 25.69	0.102
Uric acid (mg/dl)	5.6 (4.6–6.6)	5.6 (4.6–6.6)	5.6 (4.7–6.6)	0.816
CRP (mg/l)	4.00 (2.54–8.47)	3.99 (2.70–7.00)	4.27 (2.40–10.90)	0.223
Total cholesterol (mg/dl)	176 (153–204)	176 (150–201)	177 (161–210)	0.110
HDL-C (mg/dl)	39 (32–46)	39 (32–45)	38 (32–46)	0.801
LDL-C (mg/dl)	112.67 ± 35.43	110.50 ± 34.09	117.57 ± 37.95	0.058
Triglyceride (mg/dl)	119 (78–185)	123 (79–185)	115.5 (78–182)	0.719
Fasting blood glucose (mg/dl)	105 (93–131)	102 (92–123)	109 (94–146)	0.061
Troponin, first (ng/ml)	0.350 (0.056–3.050)	0.410 (0.054–2.990)	0.328 (0.060–3.280)	0.783
Troponin, peak (ng/ml)	3.745 (1.10–13.36)	3.76 (1.08–16.90)	3.68 (1.39–9.76)	0.555
CK-MB (ng/ml)	35.4 (16.6–87.3)	31.9 (14.9–72.3)	46.2 (20.45–110.75)	**0.007**
Postprocedure SYNTAX score	2 (0–6)	0 (0–2)	7 (6–9)	**<0.001**
Complete revascularization	186 (44.60%)	184 (63.67%)	2 (1.56%)	**<0.001**
Incomplete revascularization	231 (55.40%)	105 (36.33%)	126 (98.44%)	

Data are given as mean ± SD or median (1st quartile–3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.

Bold values indicate statistically significant.

CK-MB, creatine kinase MB; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; SYNTAX, The SYNergy between percutaneous intervention with TAXus drug-eluting stents and cardiac surgery; USAP, unstable angina pectoris; WBC, white blood cell.

immediately after CABG compared with those without AMI. They also reported that the severity of angina pectoris in the preoperative period was independently associated with worse sleep quality after elective isolated CABG surgery. Shaffer *et al.* [30] showed that patients with ACS-related post-traumatic stress disorder had significantly worse overall sleep quality scores on the PSQI. Nevertheless, these authors reported that ACS-induced symptoms of post-traumatic stress disorder were not significantly associated with sleep disturbance when the multivariate model was adjusted for BMI and depression. Another study reported that there was a significant deterioration in the sleep of patients in the early period after CABG and that this deterioration was most likely due to the temporary change in brain stem circulation [31]. Zhang *et al.* [32] showed that the PSQI scores of patients with USAP were higher than those of normal individuals, and the PSQI scores of patients with STEMI and NSTEMI were higher than those with USAP and normal individuals. Additionally, the circadian rhythms and clock genes were found to be correlated with the occurrence of ACS. In a descriptive and cross-sectional study, according to PSQIs completed 72 h after AMI, poor sleep quality was found in 71.7% of patients, and diabetes, depression and lack of physical activity were independent risk factors for high PSQI scores. However, SYNTAX scores were not investigated in this study [19]. On the other hand, in a retrospective study, it was reported that there was no significant association between the severity and complexity of coronary artery involvement determined using SYNTAX and Gensini scores and OSA risk classes determined by the STOP-BANG questionnaire [20]. In short, the common conclusion from most of the available studies and the present study is that the severity of the coronary artery lesion in ACS seems to be related to the severity of subsequent sleep disturbance.

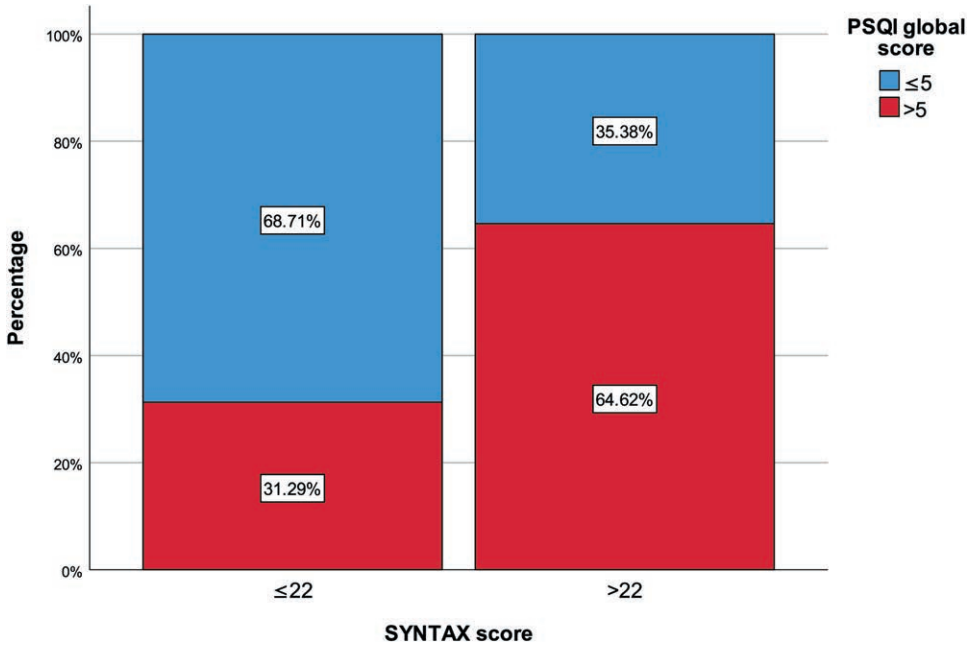
Some psychiatric disorders, circulatory diseases and gastrointestinal diseases may cause sleep disorders [33]. Conversely, patients with sleep disorders may be predisposed to cardiovascular and cerebrovascular risk factors [5]. It has been suggested that partial sleep deprivation

Table 2 Summary of sleep characteristics with regard to SYNTAX score

		SYNTAX score		
	Total ($n = 424$)	≤22 ($n = 294$)	>22 ($n = 130$)	*P* value
Sleep latency				
<15 min	159 (37.50%)	130 (44.22%)	29 (22.31%)	**<0.001**
16–30 min	173 (40.80%)	110 (37.41%)	63 (48.46%)	
31–60 min	73 (17.22%)	41 (13.95%)	32 (24.62%)	
>60 min	19 (4.48%)	13 (4.42%)	6 (4.62%)	
Sleep duration				
>7 h	198 (46.70%)	164 (55.78%)	34 (26.15%)	**<0.001**
6–7 h	117 (27.59%)	73 (24.83%)	44 (33.85%)	
5–6 h	88 (20.75%)	42 (14.29%)	46 (35.38%)	
<5 h	21 (4.95%)	15 (5.10%)	6 (4.62%)	
Sleep efficiency				
>85%	246 (58.02%)	197 (67.01%)	49 (37.69%)	**<0.001**
75–84%	114 (26.89%)	65 (22.11%)	49 (37.69%)	
65–74%	49 (11.56%)	23 (7.82%)	26 (20.00%)	
<65%	15 (3.54%)	9 (3.06%)	6 (4.62%)	
PSQI global score	5 (3–7)	4 (3–6)	6.5 (4–9)	**<0.001**
≤5	248 (58.49%)	202 (68.71%)	46 (35.38%)	**<0.001**
>5	176 (41.51%)	92 (31.29%)	84 (64.62%)	

Data are given as median (1st quartile–3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables. Bold values indicate statistically significant.
PSQI, Pittsburgh Sleep Quality Index; SYNTAX, The SYNergy between percutaneous intervention with TAXus drug-eluting stents and cardiac surgery.

Fig. 2



PSQI score with regard to SYNTAX score. PSQI, Pittsburgh Sleep Quality Index.

in healthy subjects, even for only five nights, may lead to endothelial dysfunction, impaired cardiovascular autonomic control, increased sympathetic activity and impaired blood pressure variability [10]. More than 50% of patients with stroke have been shown to have OSA, which is recognized as an independent risk factor for CVD and cerebrovascular diseases [34]. Also, many studies have shown that even in sleep disorders without apnea, the risk of ACS increases [5–7]. A Chinese study in young patients with CAD showed that after adjustment for confounding factors, patients with prolonged poor sleep quality (PSQI > 5) had an increased risk of complex coronary lesions as determined using coronary angiography and SYNTAX scores. In addition, the higher the PSQI score as a continuous variable, the higher the risk of complex coronary lesions [35]. Zhang *et al*. [36] investigated patients with OSA who had ACS, reporting that patients with severe OSA had a higher SYNTAX score than the mild group and the moderate group and that SYNTAX scores were positively correlated with AHI.

Table 3 Summary of sleep characteristics with regard to acute coronary syndrome

	Acute coronary syndrome		
	NSTEMI ($n = 126$)	STEMI ($n = 228$)	*P* value
Sleep latency			
<15 min	44 (34.92%)	88 (38.60%)	0.775
16–30 min	57 (45.24%)	90 (39.47%)	
31–60 min	20 (15.87%)	40 (17.54%)	
>60 min	5 (3.97%)	10 (4.39%)	
Sleep duration			
>7 h	69 (54.76%)	93 (40.79%)	0.075
6–7 h	27 (21.43%)	72 (31.58%)	
5–6 h	23 (18.25%)	49 (21.49%)	
<5 h	7 (5.56%)	14 (6.14%)	
Sleep efficiency			
>85%	70 (55.56%)	130 (57.02%)	0.067
75–84%	37 (29.37%)	59 (25.88%)	
65–74%	10 (7.94%)	33 (14.47%)	
<65%	9 (7.14%)	6 (2.63%)	
PSQI global score	5 (3–7)	5 (3–7)	0.487
≤5	77 (61.11%)	124 (54.39%)	0.221
>5	49 (38.89%)	104 (45.61%)	

Data are given as median (1st quartile–3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.
Bold values indicate statistically significant.
NSTEMI, non-ST-elevation myocardial infarction; PSQI, Pittsburgh Sleep Quality Index; STEMI, ST-elevation myocardial infarction.

Table 4 Summary of sleep characteristics with regard to revascularization

	Revascularization		
	Complete ($n = 186$)	Incomplete ($n = 231$)	*P* value
Sleep latency			
<15 min	95 (51.08%)	62 (26.84%)	**<0.001**
16–30 min	71 (38.17%)	100 (43.29%)	
31–60 min	18 (9.68%)	52 (22.51%)	
>60 min	2 (1.08%)	17 (7.36%)	
Sleep duration			
>7 h	113 (60.75%)	82 (35.50%)	**<0.001**
6–7 h	51 (27.42%)	64 (27.71%)	
5–6 h	18 (9.68%)	69 (29.87%)	
<5 h	4 (2.15%)	16 (6.93%)	
Sleep efficiency			
>85%	141 (75.81%)	103 (44.59%)	**<0.001**
75–84%	33 (17.74%)	77 (33.33%)	
65–74%	11 (5.91%)	38 (16.45%)	
<65%	1 (0.54%)	13 (5.63%)	
PSQI global score	4 (3–5)	6 (4–9)	**<0.001**
≤5	142 (76.34%)	103 (44.59%)	**<0.001**
>5	44 (23.66%)	128 (55.41%)	

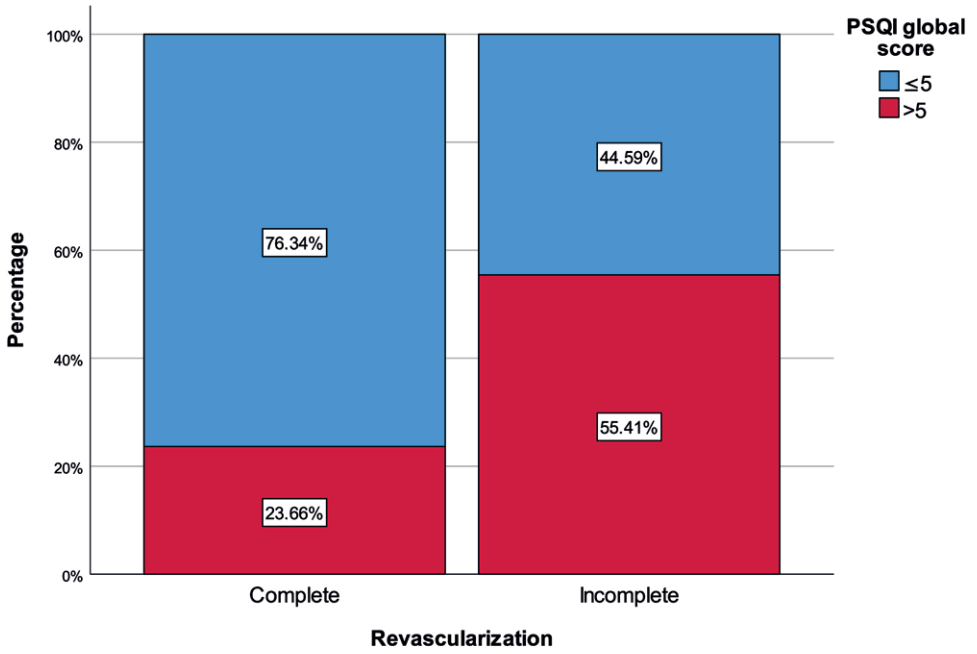
Data are given as median (1st quartile–3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.
Bold values indicate statistically significant.
PSQI, Pittsburgh Sleep Quality Index.

In a prospective study, patients with OSA had significantly higher SYNTAX scores than those without OSA, and OSA was independently associated with CAD after adjustment for traditional risk factors [37]. In a nationwide population-based cohort study, it was reported that patients with nonapnea sleep disorders were at higher risk of developing ACS, which increases with age and the presence of more than three comorbidities compared with healthy individuals. The nonapnea sleep disorders cohort in this study had a 1.43-fold increased risk of subsequent ACS compared with the nonsleep disorders cohort after controlling for age, sex and comorbidities [5]. Grandner *et al.* [8] also demonstrated the relationship between increased risk of AMI and sleep disorders regardless of the presence of apnea. In a prospective study, at a median follow-up of 1 year, the incidence of major adverse cardiovascular and cerebrovascular events was significantly higher in patients with CAD and OSA compared with those without OSA. adequate revascularization was recommended for better clinical outcomes in patients with ACS and OSA [17].

The results of all these studies show that the presence or severity of sleep disturbance is highly related to the presence or severity of CAD. OSA has significant effects on the cardiovascular system; it affects many systems. The pathophysiologic mechanism blamed for this relationship is based on endothelial dysfunction, coronary plaque burden, chronic inflammation and sympathetic activation secondary to intermittent hypoxemia [28]. Although several potential mechanisms have been proposed, the pathophysiology of the relationship between apnea-free sleep disorders and ACS remains unclear [5,38]. Stress caused due to poor sleep quality and quantity increases the release of epinephrine and norepinephrine, leading to increased heart rate, respiratory rate, blood pressure levels, myocardial oxygen demand, cardiac dysrhythmia and renal hypoperfusion disorder [39]. Conditions such as dyslipidemia, atherosclerosis and hypertension, which have been shown to be caused by apnea-free sleep disorders [5–7], may predispose coronary arteries to sudden reduced or occluded blood flow [5], which in turn increases the risk of developing ACS. Metabolic diseases caused by poor sleep quality and quantity as a result of various inflammatory mediators and hormones also constitute a risk factor for ACS [40]. In sum, the risk of ischemic heart attack is higher in people with sleep disturbances [26]. For this reason, it is important to diagnose sleep disorders, investigate their causes and treatment, and take necessary precautions in terms of cardiac outcomes. The current study has shown that patients with high baseline or postprocedure SYNTAX scores have a higher risk of having a higher PSQI score, that is, lower sleep quality, which has been shown to be highly associated with high ACS risk. Therefore, we suggest that in patients with ACS with high SYNTAX scores, proactively investigating sleep disorders, taking necessary precautions, and performing interventions may reduce residual cardiovascular risk and improve quality of life.

The limitations of the study are as follows. Being a single-center study limited the external validity of the results. The time between ACS diagnosis and completing the PSQI questionnaire, hospital stay duration, physical activity, socioeconomic status and family history were ignored. Although sleep quality was evaluated using a subjective test, the fact that the PSQI has been shown to be

Fig. 3



Global score distribution according to revascularization status.

Table 5 Odds ratios for high Pittsburgh Sleep Quality Index global score (>5), logistic regression analysis results

	Univariate		Multivariate	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Age	1.020 (1.004–1.037)	**0.014**	1.012 (0.991–1.033)	0.274
Sex, male	0.831 (0.521–1.327)	0.439		
BMI, kg/m^2	1.015 (0.966–1.067)	0.555		
Hypertension	1.446 (0.976–2.141)	0.066		
Diabetes	1.661 (1.061–2.600)	**0.027**	1.413 (0.859–2.326)	0.174
Stroke	1.416 (0.282–7.100)	0.672		
Atrial fibrillation	1.899 (0.420–8.594)	0.405		
Smoking	1.043 (0.695–1.564)	0.839		
Diuretics use	1.213 (0.692–2.127)	0.499		
Acute coronary syndrome[a]				
NSTEMI	1.300 (0.704–2.403)	0.402		
STEMI	1.714 (0.976–3.009)	0.061		
LVEF	0.960 (0.938–0.983)	**0.001**	0.978 (0.951–1.005)	0.103
Hemoglobin	0.953 (0.857–1.059)	0.368		
Platelet (×10^3)	1.000 (0.997–1.002)	0.689		
WBC (×10^3)	1.011 (0.952–1.075)	0.713		
Creatinine	1.303 (0.693–2.450)	0.412		
eGFR	0.991 (0.983–1.000)	**0.039**	0.997 (0.986–1.008)	0.595
Uric acid	1.016 (0.971–1.062)	0.494		
CRP	1.004 (0.994–1.014)	0.463		
Total cholesterol	1.001 (0.997–1.006)	0.547		
HDL-C	1.012 (0.998–1.027)	0.104		
LDL-C	1.000 (0.994–1.005)	0.982		
Triglyceride	1.000 (0.999–1.002)	0.673		
Fasting blood glucose	1.003 (0.999–1.006)	0.147		
Troponin, first	0.990 (0.971–1.009)	0.299		
Troponin, peak	0.995 (0.986–1.005)	0.328		
CK-MB	1.002 (1.000–1.005)	**0.040**	1.001 (0.999–1.004)	0.376
SYNTAX score, >22	4.009 (2.593–6.200)	**<0.001**	2.220 (1.281–3.849)	**0.004**
Incomplete revascularization	4.011 (2.619–6.142)	**<0.001**	2.189 (1.291–3.710)	**0.004**
Nagelkerke R^2	–		0.188	

Bold values indicate statistically significant.

CI, confidence interval; CK-MB, creatine kinase MB; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; NSTEMI, non-ST-elevation myocardial infarction; OR, odds ratio; STEMI, ST-elevation myocardial infarction; SYNTAX, The SYNergy between percutaneous intervention with TAXus drug-eluting stents and cardiac surgery; USAP, unstable angina pectoris; WBC, white blood cell.

[a]Reference category: USAP.

associated with depression, anxiety and CAD caused by sleep disorders, independent of OSA, minimizes this limitation [10,15]. Although patients with polysomnography-proven OSA diagnosis were excluded, it cannot be said with certainty that the results were only related to nonapnea sleep disorders because study-specific polysomnography testing could not be performed on all participants. The lack of a sleep disorder evaluation before ACS makes it difficult to make definitive comments about the cause-and-effect relationship between SYNTAX and PSQI.

In conclusion, a high baseline or postprocedure SYNTAX score (>22) was an independent predictor of poor sleep quality that might occur after ACS. Among patients diagnosed as having ACS, those with high SYNTAX scores should be monitored more carefully for sleep disorders that may occur later. Thus, with early interventions, increased cardiac and other risks that may occur due to sleep disturbance can be reduced. However, it should be emphasized that the relationship between SYNTAX score and sleep disorders is an association not necessarily causal.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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