Association of acute coronary syndrome-induced posttraumatic stress disorder symptoms with self-reported sleep

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Abstract

Background—Symptoms of posttraumatic stress disorder (PTSD) after acute coronary syndrome (ACS) are associated with recurrent ACS events and mortality. Poor sleep may be a mechanism, but the association between PTSD and sleep after ACS is unknown.

Purpose—To estimate the association between ACS-induced PTSD symptoms and self-reported sleep.

Methods—ACS-induced PTSD symptoms were assessed 1-month post-ACS in 188 adults using the Impact of Events Scale-Revised. Sleep was assessed using the Pittsburgh Sleep Quality Index. Linear and logistic regression models were used to determine whether PTSD symptoms were associated with self reported sleep, independent of sociodemographic and clinical covariates.

Results—In adjusted models, ACS-induced PTSD symptoms were associated with worse overall sleep (= 0.22, $p = 0.003$) and greater impairment in six of seven components of sleep (all $p's <$ 0.05).

Conclusions—ACS-induced PTSD symptoms may be associated with poor sleep, which may explain why PTSD confers increased cardiovascular risk after ACS.

Keywords

posttraumatic stress disorder; sleep; acute coronary syndrome

Acute coronary syndrome (ACS)-induced symptoms of post-traumatic stress disorder (PTSD) are prevalent among patients with ACS, and are associated with adverse clinical outcomes. A recent meta-analysis of ACS patients found an aggregate prevalence estimate for PTSD of 12% across 24 studies, and a doubling of risk for a combined outcome of mortality and ACS recurrence in patients with clinically significant PTSD symptoms compared to those without PTSD symptoms (1). It is well known that exposure to a traumatic event can interfere with sleep and that PTSD contributes to the development of sleep problems (2). Indeed, approximately 70% of people with clinician-diagnosed PTSD have co-occurring sleep problems (2, 3). Additional research has shown that disrupted sleep is also common among ACS patients (4), and disrupted sleep has been shown to predict

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cardiovascular outcomes (5). Understanding and modifying risk factors for disrupted sleep in patients after ACS may ultimately reduce morbidity and mortality. Although risk factors for disrupted sleep have been examined in patients with chronic diseases, including those with stable coronary heart disease (6), few studies have examined predictors of sleep in post-ACS patients.

Notwithstanding the wealth of data linking PTSD to sleep problems in the general populations and psychiatric samples, no studies to our knowledge have examined the specific association of *ACS-induced* PTSD symptoms with sleep in patients with an ACS. Given that medically-induced forms of trauma such as an ACS have been differentiated from other forms of trauma both conceptually and clinically (7), it is possible that the associations of PTSD symptoms and sleep differ based on type of trauma. In addition, little is known about the mechanisms linking PTSD to outcomes, and poor sleep may be a candidate mechanism by which PTSD confers increased risk of ACS recurrence and mortality. We thus examined the associations of *ACS-induced* PTSD symptoms with overall self-reported sleep in a cross-sectional study of 188 patients with ACS. Given the well-characterized association between PTSD and sleep reported in community samples and other patient populations, we hypothesized that greater ACS-induced PTSD symptoms would be associated with worse self-reported sleep. We secondarily tested whether pre-ACS history of PTSD was also associated with overall self-reported sleep in this sample. Finally, we examined whether specific components of self-reported sleep, including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medications, and daytime dysfunction, were associated with greater ACS-induced PTSD symptoms.

Method

Participants

Participants were consecutively hospitalized patients with ACS who were enrolled in the Prescription Usage Lifestyle and Stress (PULSE) study, an ongoing, single site, prospective, observational cohort study of the prognostic risk conferred by psychosocial factors at the time of an ACS. Patients with unstable angina pectoris or acute ST and non-ST segment elevation myocardial infarction were recruited from Columbia University Medical Center within one week of hospitalization for their ACS. Patients completed a structured psychiatric interview 3–7 days post-discharge and a follow-up interview 1 month later. The current analyses include 188 participants who completed self-report measures of ACS-induced PTSD symptoms and sleep approximately 1 month after their index ACS event. Excluded from analyses were 507 participants who were enrolled prior to initiation of PTSD data collection ($n = 362$), were missing data on sleep ($n = 4$), were missing data on both PTSD symptoms and sleep ($n = 94$), or whose PTSD and sleep data were obtained beyond the window of the 1-month follow-up visit ($n = 47$). Compared to the 188 participants included in this study, the 507 participants not included did not differ on any sociodemographic, behavioral, or clinical factors included in these analyses. Data collection occurred between February 2009 and June 2010. The Institutional Review Board of Columbia University approved this study, and all participants provided informed consent.

Measures

Self-Reported Sleep Problems—One-month following their discharge from the hospital, participants attended a follow-up visit during which they completed the Pittsburgh Sleep Quality Index (PSQI), a widely used self-report measure of sleep over the past month on which higher total scores indicate worse overall sleep (8). Probable sleep disorder was defined categorically as a global PSQI score > 5, a cutoff with diagnostic sensitivity of

89.6% and specificity of 86.5% in distinguishing groups of "good" and "poor" sleepers compared to gold standard clinical and laboratory measures (8).

In addition to a total sleep score, which has a possible range of 0 to 21, the PSQI also provides information regarding seven components of sleep. The first component, *subjective sleep quality*, is measured by a single item that asks participants to rate their overall sleep quality using a 4-point response scale ranging from 1 ("very good") to 4 ("very bad"). The second component, *sleep latency*, is assessed with two items, including how many minutes it usually takes the participant to fall asleep and how often participants are unable to fall asleep within 30 minutes. For the second item, participants use a 4-point response scale ranging from 1 ("not during the past month") to 4 ("three or more times per week"). This 4-point response scale is used for all other frequency items on the PSQI. The third component, *sleep duration*, is based on the number of hours of sleep during a typical night reported by participants. The fourth component, *habitual sleep efficiency*, is assessed using participants' reports of when they typically went to bed, when they typically awoke, and how many hours they typically slept. This information is used to compute the proportion of sleep time relative to the number of hours in bed. The fifth component, *sleep disturbance*, is assessed using nine items such as frequency of waking during the night or frequency of having pain. The sixth component, *use of sleep medications*, is assessed based on participants' reports of how often they took prescription or over-the-counter medication to help them sleep. The seventh component, *daytime dysfunction*, is assessed using two items that ask participants about the frequency by which they were not able to "keep up enthusiasm to get things done" and "having trouble staying awake while driving, eating meals, or engaging in social activity."

Consistent with the PSQI scoring instructions (7) response to items for each PSQI component were recoded such that scores ranged from 0 to 3, with higher scores indicating poorer sleep on that component. Correlations (Spearman's *rho*) among the individual sleep components ranged from = 0.11 (sleep duration and use of sleep medications) to = 0.65 (sleep duration and habitual sleep efficiency). All correlations—except the associations of use of sleep medications with sleep duration, daytime dysfunction, and habitual sleep efficiency—were significant (all *p's* 0.008). The mean correlation was = 0.31.

Scores across the seven PSQI component scores were aggregated into a global score of self-reported sleep with a range from 0 to 21. As global scores above 5 are considered indicative of probable sleep disorder (8), any participant with a global score above 5 was given a score of 1 on probable sleep disorder, and all other respondents were coded 0.

ACS-Induced PTSD Symptoms—ACS-induced PTSD symptoms were assessed at the 1-month follow-up visit with the Impact of Events Scale-Revised (IES-R) (9). Participants used a 5-point scale ranging from 0 ("not at all") to 4 ("extremely") to report the extent to which they had been bothered by each of 22 PTSD symptoms in the past week with reference to their hospitalization for ACS. Specifically, they were told, "Think about when you were hospitalized on [DATE], and we enrolled you in this study. In the past seven days have you been bothered by any of the following in reference to that hospitalization?" The IES-R yields a total score with a range from 0 to 88, and the internal validity of this score in the current study was acceptable (Cronbach's = 0.94).

Pre-ACS History of PTSD—Within 3–7 days of their discharge from the hospital, participants were screened for PTSD in the 3 months preceding hospitalization for their index ACS event using the Structured Clinical Interview for DSM-IV (10). They reported whether they had (1) experienced a traumatic event ("such as a major disaster, very serious accident or fire, being physically assaulted or raped, seeing another person killed or dead, or badly hurt, or hearing about something horrible that has just happened to someone close to

you"), (2) whether the event recurred "in nightmares, flashbacks, or thoughts you cannot get rid of," and (3) whether the experience of "thoughts or feelings coming back" had happened in the past 3 months. Participants who responded "yes" to all three items were categorized as having a pre-ACS history of PTSD. Responses to these questions correctly identified 97% of PTSD cases in previous studies (11). All analyses that included pre-ACS history of PTSD were limited to the 158 participants who were not missing data on this variable.

Sociodemographic and Clinical Covariates—During their hospitalization, participants identified their ethnicity (Hispanic versus non-Hispanic), race (Black versus non-Black), high school education (yes/no), and partner status (has a partner or spouse/does not have a partner or spouse). Medical chart review was used to ascertain body mass index (BMI), prior cardiovascular disease and cardiac procedures (unstable angina, myocardial infarction, percutaneous coronary intervention, coronary artery bypass grafting, New York Heart Association heart failure class), prior cerebrovascular disease (stroke, transient ischemic attack), and other chronic conditions (e.g., respiratory diseases, liver diseases, and rheumatologic diseases). Age and sex were recorded for each participant. Measures of disease severity included the Global Registry of Acute Coronary Events risk score as an estimate of 6-month post-ACS mortality risk (12), and the Charlson comorbidity index as a measure of medical comorbidities (13).

Psychological and Behavioral Covariates—During the 1-month follow-up, participants also completed the 21-item Beck Depression Inventory (14) and the Paffenbarger Physical Activity Questionnaire (15). The Beck Depression Inventory is a self-report instrument that assesses severity of depressive symptoms experienced during the past week, with possible scores for each question ranging from 0 to 3 and a possible total score ranging from 0 to 63. It has excellent psychometric properties, as shown with multiple community and medical samples (16). In the current study, the internal consistency of the Beck Depression Inventory as measured by Cronbach's was 0.88. The Paffenbarger Physical Activity Questionnaire was used to calculate weekly energy expended through leisure time physical activity using the scoring system derived for this questionnaire, as described by Paffenbarger and colleagues (17).

Analytic Strategy

Baseline sociodemographic, behavioral, and clinical characteristics were calculated separately for those with and without probable sleep disorder, and participants in these two groups were compared using independent-samples t-tests and Pearson's chi-square tests for continuous and categorical variables, respectively. The association between ACS-induced PTSD symptoms and self-reported sleep was examined in an unadjusted and an adjusted linear regression model with total IES-R score as the primary predictor and total continuous PSQI score as the primary dependent variable. Given that the total PSQI score was positively skewed, a square-root transformation was applied to this variable. The adjusted linear regression model included sociodemographic, behavioral, and clinical predictors of self-reported sleep (18) as covariates, including age, sex, partner status, high school education, BMI, Paffenbarger physical activity score, GRACE risk score, Charlson comorbidity index, and Beck Depression Inventory score. All covariates were entered with the primary predictor variable in a single block. Similar unadjusted and adjusted binary logistic regression models were conducted with probable sleep disorder (i.e., global PSQI score > 5) as the outcome measure. To secondarily examine possible differential associations between self-reported sleep and pre-ACS history of PTSD versus ACS-induced PTSD symptoms, all aforementioned linear and logistic regression models were repeated substituting pre-ACS history of PTSD for IES-R scores.

To test whether specific components of self-reported sleep were associated with ACS-induced PTSD symptoms, we conducted exploratory unadjusted and adjusted ordered logistic regression models for each of the seven PSQI sleep components. The assumption of proportional odds, which specifies homogeneity of the effect of each predictor variable across all levels of the outcome, was evaluated for each model. This assumption was violated for models with sleep duration, sleep efficiency, and sleep disturbances. Because these three components of the PSQI are calculated using a continuous, interval scale before being recoded to the continual, ordinal scale used by the other PSQI components, we conducted a median split of these variables and analyzed them using binary logistic regression.

We additionally performed exploratory analyses to characterize the unique and common variance contributions of ACS-induced PTSD symptoms and depressive symptoms with overall self-reported sleep. To do so, we conducted a hierarchical linear regression model with three blocks of variables. Block 1 included age, sex, and all demographic and clinical covariates except the Beck Depression Inventory; block 2 added the Beck Depression Inventory; and block 3 added the IES-R. Using this approach, we report the change in the variance explained in overall self-reported sleep associated with the addition of ACS-induced PTSD symptoms to a model already including depressive symptoms. We additionally report the partial correlations of ACS-induced PTSD symptoms with overall self-reported sleep first controlling for all demographic and clinical covariates except depressive symptoms and then additionally controlling for depressive symptoms.

The percentage of missing data on covariates ranged from 1.0% (for partner status) to 1.6% (for Paffenbarger physical activity score). We applied multiple imputation generating five imputed datasets. All variables except the primary independent variables (i.e., ACS-induced PTSD symptoms and pre-ACS history of PTSD) and dependent variables (i.e., all PSQI scores) were included. All analyses were conducted using SPSS 20.0 (IBM, Armonk, NY).

Results

Participants in this sample were predominantly male (65.4%) and non-Hispanic (63.8%) with a mean age of 63.2 ± 11.3 years. Other descriptive characteristics of this sample are shown in Table 1.

Association of ACS-induced PTSD symptoms with overall self-reported sleep

The mean IES-R score for the sample of 188 participants was 6.3 ± 11.0 (range: 0 – 55.0), and the mean Global Registry of Acute Coronary Events total score was 5.2 ± 4.0 (range: 0 – 17.0). Greater ACS-induced PTSD symptoms were independently associated with worse overall self-reported sleep in unadjusted and adjusted linear regression models. In the adjusted model, which included as covariates age, sex, ethnicity, partner status, high school education, BMI, Paffenbarger physical activity score, GRACE risk score, Charlson comorbidity index, and depressive symptom score, each standard deviation increase on the IES-R measure of ACS-induced PTSD symptoms was associated with a 0.22 standard deviation worsening in overall self-reported sleep ($ = 0.22$, $p = 0.003$; Table 2). Hispanic ethnicity, greater BMI, and depressive symptoms were also independent, significant correlates of overall self-reported sleep in this adjusted linear regression model.

Of note, the unadjusted association of ACS-induced PTSD symptoms with overall self-reported sleep was attenuated when depressive symptoms were added to the regression model but not when other demographic and clinical characteristics were added. Specifically, the zero-order correlation of ACS-induced PTSD symptoms with overall self-reported sleep was 0.38; the partial correlation of ACS-induced PTSD symptoms with overall self-reported

sleep independent of all covariates except depressive symptoms was 0.39; and the partial correlation of ACS-induced PTSD symptoms with overall self-reported sleep independent of all covariates including depressive symptoms was 0.26 (all p's < 0.001). Notwithstanding this attenuated effect, the addition of ACS-induced PTSD symptoms to a fully-adjusted model that included depressive symptoms contributed to a significant increase of 5% in the variance explained in overall self-reported sleep (F_{change} (1, 172) = 12.5, p = 0.001).

Association of ACS-induced PTSD symptoms with probable sleep disorder

Of the 188 participants, 76 (40.4%) had a PSQI total score > 5, which is indicative of probable sleep disorder. Compared to participants who did not have probable sleep disorder, those who did have a probable sleep disorder had lower scores on the total PSQI score and all seven sleep components, which is indicative of poorer self-reported sleep. They were also significantly more likely to be female and Hispanic, had a significantly higher BMI, and reported significantly more depressive symptoms and ACS-induced PTSD symptoms. In the unadjusted logistic regression model, greater ACS-induced PTSD symptoms were significantly associated with elevated odds of having probable sleep disorder (i.e., PSQI total score > 5). Each point increase on the IES-R measure of ACS-induced PTSD symptoms was significantly associated with approximately 6% increased odds of having probable sleep disorder. ACS-induced PTSD symptoms were *not* significantly associated with odds of probable sleep disorder in the adjusted model, although greater BMI and greater depressive symptoms were significantly associated with increased odds of probable sleep disorder in this model. Hispanic ethnicity was associated with significantly *lower* odds of probable sleep disorder.

Association of pre-ACS history of PTSD with overall self-reported sleep and probable sleep disorder

Pre-ACS history of PTSD was not associated with either overall self-reported sleep or probable sleep disorder in unadjusted or adjusted regression models (all p's > 0.11).

Association of ACS-induced PTSD symptoms with individual components of self-reported sleep

Greater ACS-induced PTSD symptoms were significantly associated with six of seven PSQI sleep components in unadjusted and adjusted logistic regression models (Table 3). The association of ACS-induced PTSD symptoms with sleep latency was only significant in an unadjusted model. The magnitude of the significantly increased odds of poor self-reported sleep associated with each 1-point increase on the IES-R measure of ACS-induced PTSD symptoms ranged from 2% for sleep quality and daytime dysfunction to 9% for sleep disturbances. Female sex, Hispanic ethnicity, BMI, partner status, Charlson comorbidity index, and depressive symptoms were also significantly associated with various self-reported sleep components.

Discussion

We found that greater ACS-induced symptoms of PTSD were associated with worse overall self-reported sleep and poorer scores on six of seven individual component of sleep as measured by the PSQI in 188 patients with recent hospitalization for ACS. These associations remained significant after adjusting for sociodemographic covariates, medical comorbidities (Charlson), cardiac disease severity (Global Registry of Acute Coronary Events risk score), and depressive symptoms (Beck Depression Inventory). We also observed a trend for an association between ACS-induced PTSD symptoms and probable sleep disorder, although this association did not remain significant after multivariable

adjustment. In contrast, pre-ACS history of PTSD was not associated with overall self-reported sleep or probable sleep disorder in either unadjusted or adjusted models.

Our findings are consistent with those from studies of PTSD and sleep among individuals with PTSD induced by non-medical events. Indeed, previous studies have indicated that as many as 70% of those with PTSD report sleep disturbances (3). Furthermore, polysomnography assessments have shown that compared to those without exposure to childhood trauma, those with exposure have greater trouble initiating and maintaining sleep, poorer sleep quality, greater sleep disturbances and nighttime awakenings, lower sleep efficiency, shorter duration of rapid eye movement sleep, and longer latency to enter rapid eye movement sleep (2). Another study of outpatients with a primary diagnosis of PTSD found positive correlations between severity of PTSD, the PSQI global score and five of the seven PSQI components (sleep efficiency and use of sleeping medications did not vary with PTSD severity) (19). The strong association between ACS-induced PTSD and sleep may be due to the fact that disturbed sleep is embedded in the PTSD phenotype and its diagnosis. However, results of recent treatment studies for PTSD and sleep disturbance suggest that the two conditions may be most usefully conceptualized as comorbid, rather than one being merely a symptom of the other (20, 21). In addition, autonomic dysregulation, which is associated with both PTSD (22) and disrupted sleep (23), may represent a common mechanism underlying their association.

Of note, the adjusted association of ACS-induced PTSD symptoms with probable sleep disorder was not significant, although the magnitude of the unadjusted and adjusted associations was comparable. Although it is possible that ACS-induced PTSD symptoms are indeed not associated with probable sleep disorder, there are other explanations for the observed null finding. First, we may have had limited statistical power to detect this association given that our total sample size was small relative to the number of covariates in our adjusted regression model. Second, it is possible that the cutpoint used to define probable sleep disorder (i.e., PSQI total score > 5) is not optimal for participants with PTSD or ACS-induced PTSD. Although the diagnostic accuracy of the PSQI has not been investigated exclusively for individuals with PTSD to our knowledge, studies of other measures have found different estimates of diagnostic accuracy for subgroups of patients with different clinical characteristics (24). Interestingly, in *post hoc* analyses of this study, we found that ACS-induced PTSD symptoms were significantly associated with an 8% increased odds of probable sleep disorder in an adjusted model with probable sleep disorder defined as having a PSQI total score greater than the median (> 4).

Our analyses identified several additional independent correlates of self-reported sleep and sleep components including female sex, Hispanic ethnicity, partner status, BMI, Charlson comorbidity index, and depressive symptoms. Consistent with several other studies, we found that depressive symptoms were significantly associated with overall self-reported sleep quality, probable sleep disorder, and some components of self-reported sleep (25–26). Although the association of ACS-induced PTSD symptoms with overall self-reported sleep was attenuated when controlling for depressive symptoms but not when controlling for other demographic and clinical covariates, the addition of ACS-induced PTSD symptoms to a fully-adjusted model with depressive symptoms contributed to a significant increase in the variance explained in overall self-reported sleep. Future studies should further consider reasons for the differential associations of depressive symptoms with various components of self-reported sleep and disentangle the possibly complex associations among depression, PTSD, and self-reported sleep in patients with ACS. We additionally observed that female sex and greater BMI were associated with worse overall self-reported sleep, while Hispanic ethnicity was associated with protective against various components of poor sleep. Although the BMI finding is consistent with previous findings (27), other studies have reported no

gender differences in objective and subjective sleep measures (28) and have suggested that Hispanic ethnicity might confer increased risk for sleep problems (29). Future studies may pursue a more detailed investigation of the association between sociodemographic factors and sleep specifically in patients with ACS and other cardiovascular diseases.

Notwithstanding the strong and consistent associations observed between ACS-induced PTSD symptoms and both overall self-reported sleep and specific sleep components, our study is limited by several factors that deserve mention. First, it is cross-sectional, precluding us from considering the directionality of the association between ACS-induced PTSD and sleep. Although research in other populations suggests that exposure to a traumatic event can interfere with sleep and that PTSD is related to the development of sleep problems (2), other research has shown that poor sleep may contribute to the development of PTSD (29). Future research with repeat assessments of sleep and PTSD are needed to disentangle the possibly complex associations among these two factors in patients with ACS. Secondly, our assessment of sleep was obtained by self-report at a single point in time. Although the PSQI has been widely used in other studies, additional studies are needed with assessments of objectively measured sleep via gold-standard methods such as polysomnography. Future research should additionally consider whether sleep problems that precede an ACS event or sleep problems at other time points following an ACS contribute to the associations observed in the current study. Third, our assessment of ACS-induced PTSD symptoms did not include a diagnostic interview. Although the IES-R has been widely featured in studies of PTSD, has favorable psychometric properties (30), and has been linked to prognosis in ACS patients (31), additional studies of the association between clinician-diagnosed PTSD and sleep are needed. Fourth, we did not control for participants' overall medication use or a general measure of anxiety, which may confound the observed associations of ACS-induced PTSD symptoms and self-reported sleep. Finally, very few participants in our sample had a pre-ACS history of PTSD, which may have limited our power to detect an association of this variable with self-reported sleep. Nonetheless, the association of pre-ACS history of PTSD with each of the self-reported sleep outcomes was uniformly non-significant, and we did not observe any signals of an association.

Conclusion

Our study is the first to examine the association of *ACS-induced* symptoms of PTSD with self-reported sleep. The implications of the PTSD-sleep association in the context of the post-ACS recovery period is thus important to consider with respect to gaining a better understanding of the associations between PTSD, sleep, and cardiac prognosis as well as treatment strategies for patients recovering from an ACS. Both poor sleep (5) and PTSD symptoms (1) have been linked to adverse cardiovascular outcomes, and our findings suggest that either sleep or PTSD may mediate or modify the impact of the other on cardiac prognosis in ACS patients. If future research supports this mediation or moderation hypothesis, then additional research might consider whether interventions that address either PTSD symptoms or sleep among ACS patients offset the risk of adverse cardiovascular events in this population. Still other research might consider the feasibility and efficacy of routine screening for PTSD and sleep in patients with ACS to improve cardiovascular outcomes—a policy that has recently been adopted with respect to other psychosocial risk factors such depression in ACS (32).

Acknowledgments

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Table 1

Clinical and sociodemographic characteristics of 188 participants with acute coronary syndrome, by probable sleep disorder

Study variable	Full sample ($N = 188$)	Probable sleep disorder ($n = 76$)	No probable sleep disorder ($n = 112$)	P
Overall self-reported sleep (PSQI total score)	5.2 (4.0)	9.3 (2.9)	2.5 (1.6)	<0.001
Sleep quality	0.9 (0.8)	1.5 (0.8)	0.5 (0.6)	<0.001
Sleep latency	0.9 (1.0)	1.6 (1.0)	0.4 (0.6)	<0.001
Sleep duration	0.9 (1.1)	1.6 (1.1)	0.4 (0.7)	<0.001
Sleep efficiency	0.7 (1.0)	1.4 (1.2)	0.3 (0.6)	<0.001
Sleep disturbances	0.9 (0.6)	1.3 (0.6)	0.6 (0.5)	<0.001
Use of sleep medications	0.5 (1.0)	1.1 (1.3)	0.1 (0.4)	<0.001
Daytime dysfunction	0.5 (0.7)	0.9 (0.8)	0.2 (0.4)	<0.001
Sociodemographic factors				
Age, years	63.2 (11.3)	63.9 (10.4)	62.8 (12.0)	0.50
Female, n (%)	65 (34.6)	34 (44.7)	31 (27.7)	0.016
Hispanic ethnicity, n (%)	68 (36.2)	20 (26.3)	48 (42.9)	0.021
Has a partner or spouse, n (%)	111 (59.0)	44 (58.7)	67 (59.8)	0.88
Has a high school education, n (%)	137 (72.9)	56 (73.7)	81 (72.3)	0.84
Clinical and psychosocial factors				
Body mass index, kg/m^2	29.3 (6.1)	30.7 (6.5)	28.4 (5.7)	0.013
Paffenbarger physical activity score	974.5 (1,372.8)	846.1 (1,325.8)	1,061.6 (1401.8)	0.29
GRACE risk score	89.2 (27.7)	92.7 (29.2)	86.8 (26.4)	0.15
Charlson comorbidity index	1.6 (1.6)	1.8 (1.5)	1.5 (1.6)	0.14
BDI depressive symptom score	6.5 (7.1)	10.6 (8.5)	3.7 (4.2)	<0.001
ACS-induced PTSD symptoms	6.3 (11.0)	10.0 (14.4)	3.8 (6.8)	<0.001
Pre-ACS history of PTSD, n (%)	9 (4.8)	4 (6.2)	5 (5.3)	0.80

Values are mean (standard deviation) unless otherwise specified. Probable sleep disorder was defined as a PSQI total score > 5.

Abbreviations. PSQI, Pittsburgh Sleep Quality Index; GRACE, Global Registry of Acute Coronary Events; ACS, acute coronary syndrome; BDI, Beck Depression Inventory; PTSD; posttraumatic stress disorder

Table 2

Associations of current ACS-induced PTSD symptoms with overall self-reported sleep and probable sleep disorder

	Global sleep score (continuous PSQI)				Probable sleep disorder (PSQI total score > 5)			
		95% CI	P	Adjusted R²	Odds Ratio	95% CI	P	Pseudo R²
Unadjusted model								
ACS-induced PTSD symptoms	**0.38**	**0.25 – 0.51**	**< 0.001**	0.14	**1.06**	**1.02 – 1.10**	**0.001**	0.10
Pre-ACS PTSD history [a]	0.11	−0.05 – 0.27	0.17	0.006	1.19	0.31 – 4.60	0.80	0.001
Adjusted model								
ACS-induced PTSD symptoms	**0.22**	**0.08 – 0.36**	**0.003**		1.02	0.98 – 1.07	0.31	
Pre-ACS PTSD history [a]	0.03	−0.11 – 0.17	0.65		0.49	0.10 – 2.41	0.42	
Age	−0.07	−0.28 – 0.15	0.53		0.99	0.93 – 1.05	0.69	
Female sex	0.13	−0.01 – 0.27	0.06		2.03	0.89 – 4.61	0.11	
Hispanic ethnicity	**0.16**	**0.01 – 0.31**	**0.03**	0.29	**0.33**	**0.12 – 0.87**	**0.03**	0.31
Partner status [b]	0.13	−0.02 – 0.27	0.08		0.43	0.17 – 1.06	0.07	
High school education [c]	−0.07	−0.22 – 0.08	0.38		1.39	0.49 – 3.94	0.54	
Body mass index	**0.16**	**0.03 – 0.29**	**0.03**		**1.07**	**1.01 – 1.14**	**0.03**	
Paffenbarger physical activity score	0.002	−0.13 – 0.13	0.98		1.00	1.00 – 1.00	0.87	
GRACE risk score	0.13	−0.10 – 0.34	0.26		1.02	1.00 – 1.05	0.12	
Charlson comorbidity index	0.06	−0.08 – 0.20	0.38		1.05	0.93 – 1.19	0.71	
BDI depressive symptom score	**0.34**	**0.19 – 0.48**	**< 0.001**		**1.20**	**1.11 – 1.29**	**< 0.001**	

[a] Pre-ACS PTSD history was not tested in the same model as ACS-induced PTSD symptoms;

[b] Referent group is "has a partner or spouse;"

[c] Referent group is "has a high school education."

Note. Bold font indicates significant associations at $p < 0.05$.

Abbreviations. ACS, acute coronary syndrome; PTSD, posttraumatic stress disorder; PSQI, Pittsburgh Sleep Quality Index; GRACE, Global Registry of Acute Coronary Events; BDI, Beck Depression Inventory.

Table 3

Associations of current ACS-induced PTSD symptoms with specific components of self-reported sleep

	Sleep component						
	Sleep quality	Sleep latency	Sleep duration [a]	Sleep efficiency [a]	Sleep disturbances [a]	Sleep med use	Daytime dysfunction
	Unadjusted model						
ACS-induced PTSD symptoms	**1.05 (1.03 – 1.08)**	**1.04 (1.02 – 1.07)**	**1.06 (1.02 – 1.10)**	**1.03 (1.00 – 1.06)**	**1.10 (1.05 – 1.16)**	**1.04 (1.01 – 1.07)**	**1.07 (1.04 – 1.10)**
	Adjusted model						
ACS-induced PTSD symptoms	**1.03 (1.00 – 1.06)**	1.02 (0.99 – 1.05)	**1.05 (1.01 – 1.09)**	**1.04 (1.00 – 1.08)**	**1.09 (1.03 – 1.15)**	**1.05 (1.01 – 1.09)**	**1.03 (1.00 – 1.07)**
Age	1.00 (0.96 – 1.04)	0.99 (0.94 – 1.03)	1.00 (0.96 – 1.05)	1.03 (0.98 – 1.09)	1.01 (0.96 – 1.06)	1.00 (0.95 – 1.05)	1.00 (0.95 – 1.05)
Sex	1.62 (0.83 – 3.16)	**2.11 (1.09 – 4.10)**	0.80 (0.38 – 1.67)	1.66 (0.77 – 3.60)	1.32 (0.68 – 2.54)	**2.61 (1.03 – 6.61)**	0.99 (0.46 – 2.15)
Hispanic ethnicity	0.65 (0.32 – 1.31)	0.85 (0.42 – 1.69)	0.56 (0.26 – 1.22)	**0.31 (0.14 – 0.71)**	1.41 (0.65 – 3.04)	**0.28 (0.10 – 0.82)**	0.78 (0.34 – 1.73)
Partner status [b]	**0.45 (0.23 – 0.88)**	0.68 (0.35 – 1.32)	1.15 (0.56 – 2.38)	0.51 (0.23 – 1.09)	0.61 (0.28 – 1.35)	**0.34 (0.12 – 0.96)**	1.84 (0.86 – 3.96)
High school education [c]	1.87 (0.87 – 4.04)	1.14 (0.53 – 2.43)	0.91 (0.38 – 2.15)	1.64 (0.67 – 4.01)	0.85 (0.35 – 2.09)	0.88 (0.28 – 2.73)	0.51 (0.50 – 1.29)
Body mass index	1.02 (0.97 – 1.07)	1.03 (0.98 – 1.08)	1.04 (0.98 – 1.10)	**1.07 (1.01 – 1.13)**	**1.09 (1.03 – 1.16)**	1.04 (0.97 – 1.10)	**1.07 (1.01 – 1.13)**
Paffenbarger physical activity score	1.00 (1.00 – 1.00)	1.00 (1.00 – 1.00)	1.00 (1.00 – 1.00)	1.00 (1.00 – 1.00)	1.00 (1.00 – 1.00)	1.00 (1.00 – 1.00)	1.00 (1.00 – 1.00)
GRACE risk score	1.00 (0.98 – 1.02)	1.01 (0.99 – 1.03)	1.00 (0.98 – 1.02)	0.99 (0.97 – 1.01)	1.00 (0.98 – 1.02)	1.01 (0.98 – 1.03)	1.01 (0.99 – 1.03)
Charlson comorbidity index	1.07 (0.88 – 1.30)	1.08 (0.89 – 1.31)	1.00 (0.81 – 1.23)	0.99 (0.79 – 1.23)	1.04 (0.84 – 1.30)	**1.37 (1.06 – 1.76)**	0.98 (0.77 – 1.22)
BDI depressive symptom score	**1.11 (1.05 – 1.16)**	**1.08 (1.03 – 1.13)**	1.04 (0.99 – 1.10)	1.00 (0.95 – 1.06)	**1.07 (1.01 – 1.14)**	0.97 (0.91 – 1.04)	**1.15 (1.09 – 1.22)**

[a] Results are derived from binary logistic regression models; all other results are derived from ordinal logistic regression.

[b] Referent group is "has a partner or spouse;"

[c] Referent group is "has a high school education."

Note. Bold font indicates significant associations at *p* < 0.05.

Abbreviations. ACS, acute coronary syndrome; PTSD; posttraumatic stress disorder; PSQI, Pittsburgh Sleep Quality Index; GRACE, Global Registry of Acute Coronary Events; BDI, Beck Depression Inventory.