STUDENT CORNER
RESEARCH ARTICLE

The impact of sleep deprivation on the occurrence of acute coronary syndrome depending on obesity status

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Abstract

Objective: To determine how sleep insufficiency affects the occurrence of acute coronary syndrome in the presence of obesity.

Method: The observational study was conducted in South Korea on the basis of data collected by a cohort study conducted from January 2001 to December 2020, and comprised adults of either gender aged 40-69 years. During the 20-year period, 9 follow-up surveys were conducted at two-year intervals. Those with sleep <7 hours were in the insufficient sleep group A, and those with >7 hours of sleep were in the sufficient sleep group B. The main exposure variable was sleeping insufficiency, and the dependent variable was the occurrence of acute coronary syndrome. The hazard ratio between sleep insufficiency and obesity was identified, and the risk with respect to obesity was worked out. Finally, the joint effect of sleep insufficiency and obesity on the occurrence of acute coronary syndrome was determined. Data was analysed using SAS 9.4.

Results: Of the 3,698 subjects, 2,411(65.2%) were in group A; 1,234(51.2%) females and 1,177(48.8%) males with mean age 50.4+/-7.55 years, and 1,287(34.8%) subjects were in group B; 747(58%) males and 540(42%) females with mean age 50.4+/-7.41 years. There were 1,637(44.3%) obese subjects; in group A 1,097(45.5%) and in group B 540(42%). Overall, 127(3.4%) subjects developed acute coronary syndrome; in group A 85(3.5%) and in group B 42(3.3%). Incidence of acute coronary syndrome was not significantly associated with sleep sufficiency and obesity status (p>0.05). The risk of developing acute coronary syndrome was significantly higher in obese individuals having sleep insufficiency compared to non-obese individuals without sleep insufficiency (hazard ratio: 1.54, 95% confidence interval: 1.03-2.62).

Conclusion: There was a significant combined effect of obesity and sleep insufficiency on the occurrence of acute coronary syndrome.

Key Words: Acute coronary syndrome, Obesity, Sleep insufficiency.
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Introduction

Acute coronary syndrome (ACS) is a medical condition characterised by insufficient oxygen and nutrient supply to the heart muscle, primarily resulting from the constriction or blockage of coronary arteries. Sudden constriction or blockage of coronary arteries can lead to reduced blood flow in the coronary arteries, causing damage to cardiac muscle cells, and, potentially, cardiac arrest.[1]

ACS is a serious public health concern. According to the World Health Organisation (WHO) 2019 data, approximately 8.9 million people worldwide died from ACS, accounting for approximately 16% of the global mortality that year.[2] Data for 2020 released by the South Korean Statistical Office also indicated that cardiovascular diseases (CVDs) ranked second among the top 10 causes of death. The mortality rate due to ACS, including myocardial infarction and angina, accounted for 63% of the overall mortality rate, steadily increasing from 46.9% in 2010.[3] The incidence of ACS is expected to continue rising in the future. Moreover, the global economic burden attributed to ACS is projected to increase to $1.44 trillion by 2030; a 22% increase compared to the 2010 figure of $863 billion.[4]

Accurately assessing the risk factors is essential for the development of effective ACS prevention and control strategies.[5] The current known risk factors for ACS include age, gender, family history of heart disease, hypertension (HTN), diabetes mellitus (DM), dyslipidaemia, smoking,

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alcohol consumption, obesity, lack of physical activity, and sleep status.[6,7] Of these, sleep, a fundamental and critical lifestyle habit, has been associated with the development of ACS, along with various sleep disorders, such as insomnia, sleep apnoea and snoring.[8,9] Previous research has shown that insufficient or irregular sleep, as well as poor sleep quality, can lead to increased inflammation, elevated blood pressure (BP), disruptions in glucose metabolism, heightened sympathetic nervous system activity, circadian rhythm disturbances, and increased oxidative stress (OS), all of which are associated with an increased risk of developing CVDs.[10] Additionally, sleep insufficiency can result in increased feelings of fatigue, decreased energy levels, reduced concentration, and decreased motivation for physical activity.[11, 12]

Dietary intake may also add to the impact, as irregular eating habits lead to an imbalance of appetite-related hormones, such as leptin and ghrelin.[13] This increases cravings for unhealthy, high-calorie, high-carbohydrate foods, potentially exacerbating the risk of obesity, chronic diseases and ACS.[14]

Obesity is closely associated with physiological and metabolic changes that negatively impact cardiovascular health, making it a significant risk factor for ACS. Individuals with obesity often have elevated levels of low-density lipoprotein (LDL) cholesterol and triglycerides (TG), which contribute to the formation of blood clots in arteries. Obesity can also induce a chronic inflammatory state within blood vessels, promoting endothelial dysfunction and further promoting blood clot formation. Furthermore, obesity indirectly increases the risk of ACS by serving as a risk factor for conditions, such as HTN and DM, which are known risk factors of ACS.[15]

Evidence suggests an interactive effect between sleep insufficiency and obesity on the onset and exacerbation of various diseases, including HTN, DM and metabolic syndrome.[16] However, while both sleep insufficiency and obesity have been identified as individual risk factors for ACS, their combined effects on ACS have not been widely studied.[17,18]

The current study was planned to fill the gap in literature by determining how sleep insufficiency affects the occurrence of ACS in the presence of obesity. It was hypothesised that sleep insufficiency and obesity may interact, and the combination may contribute to ACS risk.

Materials and Methods

The observational study was conducted in South Korea, and was based on data from the Korean Genome and Epidemiology Study (KoGES), a community-based prospective cohort that was conducted from January 2001 to December 2020. The KoGES, conducted by the Korea Centres for Disease Control and Prevention (KCDC) and the Korean National Institute of Health (KNIH), aimed at investigating the major genetic and environmental risk factors for chronic diseases. To achieve this, a population-based cohort was established, comprising participants from the general population. Epidemiological data, as well as human biological samples, such as deoxyribonucleic acid (DNA), serum, plasma and urine, were collected through health surveys and examinations. Specifically, the study targetted adults aged 40-69 years, residing in two urban communities, Ansan and Anseong. The baseline survey commenced in 2001-02, and nine follow-up surveys were conducted at two-year intervals until 2020.[19, 20]

The current study selected individuals from the KoGES Ansan cohort who had participated in a sleep duration investigation during the first follow-up survey in 2003-04. Those with pre-existing ACS were excluded, and so were those who gave no response to the sleep-related questions, and those who did not provide information regarding ACS throughout the second (2005-06) to the ninth (2019-20) follow-up surveys in the KoGES study. For the participants who died during the follow-up period, the follow-up time period was defined as the time from the first follow-up survey until death.

Responses from the first follow-up survey, variables related to general characteristics, health-related characteristics, and blood test results associated with ACS were extracted from the KoGES database.[19,20]

The general characteristics included age, gender and educational period. Educational period was categorised based on the mandatory education period in South Korea, which is 9 years. The health-related characteristics included medical history, history of depression, obesity status, alcohol consumption, smoking status, physical activity, sleep duration, and serum cholesterol levels. Medical history, such as HTN, DM and hyperlipidaemia, was based on responses to questions about medical diagnoses (yes/no). Obesity status was defined as per global standards on the basis of body mass index (BMI). Alcohol consumption was based on responses to questions about alcohol use at the time of the survey (yes/no). Smoking status was categorised as current smoker, former smoker, or non-smoker. Physical activity was defined based on whether the participants engaged in vigorous physical activity at least once a week (yes/no).[21] Sleep duration, based on previous health studies on sleep duration, was defined as the number of hours of sleep during the weeknights, with <7 hours

considered insufficient sleep.[22] Data of those with insufficient sleep was placed in group A, and data of those with sufficient sleep was placed in group B. Depression was categorised as absent (0-13), mild (14-19), moderate (20-28), or severe depression (29-63) based on Beck Depression Inventory (BDI) scores conducted in the KoGES cohort.[19,20] Serum cholesterol levels were measured from the blood samples collected from the participants. Finally, the occurrence of ACS was indicated.

For the current study, the sample size was calculated using the WHO calculator with 90% confidence level and an 10% absolute precision based on literature.[23] The sample size was inflated by approximately 10% to ensure robust analysis.

Data was analysed using SAS 9.4. General and health-related characteristics were treated as categorical variables and presented as frequencies and percentages. Considering sleep duration, the characteristics were compared using independent samples t-tests for continuous variables, and chi-square tests were used for categorical variables. Means +/- standard deviations (SDs) were presented for continuous variables. Chi-square test was used to compare characteristics on the basis of sleep duration.

To assess the individual risks of sleep insufficiency and obesity in the occurrence of ACS, Cox proportional hazard model analysis was performed. The analysis was adjusted for potential confounding variables, including general characteristics (age, gender, and educational period) and health-related characteristics (HTN, DM, hyperlipidaemia, alcohol intake, smoking, and physical activity). Adjusted hazard ratios (aHRs) and their corresponding 95% confidence intervals (Cis) were calculated. Additionally, the proportional hazards assumption was tested and confirmed to meet all variables included in the model. Furthermore, an interaction analysis was conducted to investigate the risk of ACS associated with sleep insufficiency according to obesity status. To examine the combined effect of sleep insufficiency and obesity, HRs and 95% CIs were calculated as each risk factor was added to the reference group comprising participants without sleep insufficiency and obesity. P<0.05 was considered statistically significant.

Results

Of the 4,023 cases, data was included for 3,698(91.92%) subjects was included (Figure). Of them, 2,411(65.2%) were in group A; 1,234(51.2%) females and 1,177(48.8%) males with mean age 50.4+/-7.55 years. There were 1,287(34.8%) subjects in group B; 747(58%) males and 540(42%) females with mean age 50.4+/-7.41 years. There were 1,637(44.3%) obese

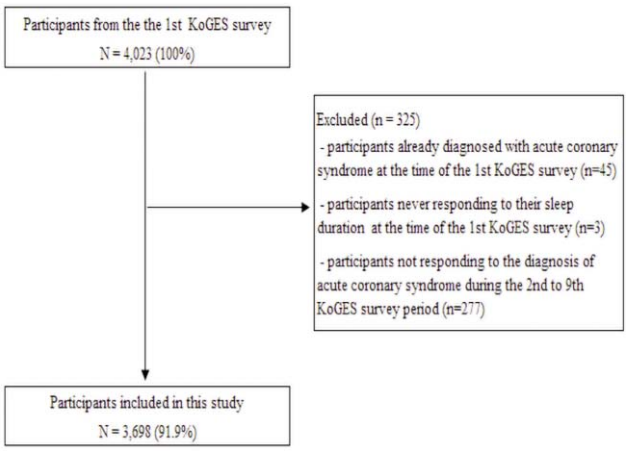

Figure: Incusion and exclusion criteria.
KoGES: Korean Genome and Epidemiology Study.

Table-1: Baseline characteristics according to sleep duration (n=3,698).

Variable	All	Sleep duration		P-value
	N (%)	Insufficient (< 7 hours)	Sufficient	
All	3,698 (100.0)	2,411 (100.0)	1,287 (100.0)	
General characteristics				
Age, year, mean (SD)	50.4 (7.50)	50.4 (7.55)	50.4 (7.41)	0.88
Gender, female	1,774 (48.0)	1,234 (51.2)	540 (42.0)	< 0.05
Educational period > 9 years	2,151 (58.2)	1,423 (59.0)	728 (56.6)	0.15
Health-related characteristics				
Disease history				
Hypertension	423 (11.4)	285 (11.8)	138 (10.7)	0.32
Diabetes mellitus	189 (5.1)	112 (4.6)	77 (6.0)	0.08
Dyslipidaemia	127 (3.4)	91 (3.8)	36 (2.8)	0.12
Obesity, yes (BMI>25)	1,637 (44.3)	1,097 (45.5)	540 (42.0)	< 0.05
Alcohol intake, yes	2,066 (55.9)	1,305 (54.1)	761 (59.1)	< 0.05
Smoking				
Current smoker	797 (21.6)	472 (19.6)	325 (25.3)	< 0.05
Former smoker	773 (20.9)	477 (19.8)	296 (23.0)	
Never smoker	2,128 (57.5)	1,462 (60.6)	666 (51.7)	
Physical activity, vigorous	2,082 (56.3)	1,381 (57.3)	701 (54.5)	0.1
Daily sleep hour, mean (SD)	6.03 (1.28)	5.30 (0.88)	7.39 (0.62)	< 0.05
Depression				0.84
None	3,044 (82.3)	1,976 (82.0)	1,068 (83.0)	
Mild	342 (9.2)	230 (9.5)	112 (8.7)	
Moderate	163 (4.4)	108 (4.5)	55 (4.3)	
Severe	149 (4.0)	97 (4.0)	52 (4.0)	
Serum cholesterol levels				
Total cholesterol (mg/dl), mean (SD)	201.3 (34.4)	201.6 (34.4)	200.8 (34.7)	0.82
Triglyceride (mg/dl), mean (SD)	140.0 (93.4)	139.4 (96.4)	141.1 (87.5)	0.14
Acute Coronary Syndrome	127 (3.4)	85 (3.5)	42 (3.3)	0.68

SD: Standard deviation, BMI: Body mass index.

Table-2: ACS risk in relation to sleep duration and obesity.

Potential risk factor	Numbers at risk	NumbersMale ACS events	Person years	Incidence rate per 1000 PYS	Model 1 aHR (95% CI)	Model 2 aHR (95% CI)	Model 3 aHR (95% CI)
Sleep duration							
Sufficient	1287	42	17406.8	2.41	reference	reference	reference
Insufficient	2411	85	32961.6	2.58	1.11 (0.77-1.61)	1.14 (0.78-1.65)	1.14 (0.78-1.65)
p-value					0.14	0.11	0.1
Obesity							
No	2061	60	28322.2	2.12	reference	reference	reference
Yes	1637	67	22046.1	3.04	1.4 (0.98-1.98)	1.3 (0.92-1.86)	1.32 (0.93-1.88)
p-value					0.09	0.13	0.11

ACS: Acute coronary syndrome, PYS: Person-years, aHR: Adjusted hazard ratio, CI: Confidence interval.
Model 1: Adjusted age, gender, educational period, sleep duration, and obesity.
Model 2: Adjusted variables of model 1 and hypertension, diabetes and dyslipidaemia.
Model 3: Adjusted variables of model 2 and alcohol drinking, smoking status and physical activity.

Table-3: Multivariate analysis of sleep duration and ACS stratified by obesity.

Obesity status	Sleep duration		*p-value*
	Sufficient	Insufficient	
Non-obesity population			
No. of person-years	7284.97	14761.13	
No. of cases	22	45	
Multivariate HR (95% CI)	Reference	1.27 (0.74-2.18)	0.22
Obesity population			
No. of person-years	10121.78	18200.44	
No. of cases	20	40	
Multivariate HR (95% CI)	Reference	1.05 (0.63-1.75)	0.17

HR: Hazard ratio, CI: Confidence interval.

Table-4: ACS hazard ratios for the combined risk of obesity and sleep duration.

Obesity and sleep duration	No. of person-years	No. of cases	aHR (95% CI)	p-value
Non-obesity and sufficient sleep duration	10121.78	20	Reference	
Non-obesity and insufficient sleep duration	18200.44	40	1.24 (0.73-2.13)	0.21
Obesity and sufficient sleep duration	7284.97	22	1.47 (0.96-2.70)	0.07
Obesity and insufficient sleep duration	14761.13	45	1.54 (1.03-2.62)	< 0.05

aHR: Adjusted hazard ratio, CI: Confidence interval.

subjects; in group A 1,097(45.5%) and in group B 540(42%). Mean duration of follow-up was 13.7 years (SD=3.3 years). The mean sleep duration in group A was 5.30+/-0.88 hours, and it was 7.39+/-0.62 hours in group B (p<0.05) (Table 1).

Overall, 127(3.4%) subjects developed ACS; in group A 85(3.5%) and in group B 42(3.3%). The overall ACS incidence rate was 2.52 per 1000 person-years (PYS). Compared to group B (2.41), ACS incidence per 1000 PYS was higher in group A (2.58). However, after adjusting for all confounding variables, there was no significant increase in the ACS incidence rate in group A (aHR: 1.14; 95%CI: 0.78-1.65; p = 0.10). Obesity also showed no significant increase in the ACS incidence after adjusting for confounding variables (aHR: 1.32; 95%CI: 0.93-1.88; p=0.11) (Table 2).

In both obese and non-obese subjects, sleep insufficiency did not significantly increase the risk of ACS (p=0.17) (Table 3).

The risk of developing ACS was significantly higher in obese individuals having sleep insufficiency compared to non-obese individuals without sleep insufficiency (aHR: 1.54; 95%CI: 1.03-2.62; p<0.05) (Table 4).

Discussion

The current study utilising KoGES data determined the combined influence of insufficient sleep and obesity on ACS occurrence. Independently, neither insufficient sleep (defined as <7 hours) nor obesity (defined as BMI >25kg/m2) escalated the risk of ACS. However, in the presence of both insufficient sleep and obesity, the ACS risk increased substantially, indicating a synergistic interaction between insufficient sleep and obesity, leading to a significant increase in ACS risk.

Previous studies have consistently linked chronic sleep insufficiency with obesity-related health issues, including metabolic syndrome, type 2 DM (T2DM), CVDs and mental health disorders.[24,25] Moreover, a comprehensive study

published in the Journal of the American College of Cardiology highlighted that both short sleep duration (<6 hours) and prolonged sleep duration (>9 hours) increased the risk of acute myocardial infarction (AMI).[26] Contrary to previous studies, the current study did not find a significant increase in ACS risk with insufficient sleep alone. These conflicting results suggest that the relationship between insufficient sleep and ACS occurrence may be influenced by interactions with other health factors.

Obesity is a well-established risk factor for various CVDs, including ACS. The suggested underlying mechanisms include promoting low-grade inflammation, enhancing lipid accumulation, and impairing vascular function.[27] According to a 2020 meta-analysis, obesity (defined by BMI) was strongly associated with a significant increase in ACS risk in the general population. Additionally, individuals who did not have a normal BMI exhibited an elevated risk of ACS-related mortality.[15] In this study, obesity alone did not significantly increase the ACS risk. This inconsistency can be attributed to several factors, including the distinctive participant characteristics, limitations associated with defining obesity solely based on BMI, potential confounding effects stemming from comorbid conditions and lifestyle factors, and the presence of the 'obesity paradox', which refers to situations in which individuals with obesity sometimes exhibit a better outcome or survival rate than what might be expected, particularly for certain medical conditions, such as CVDs, HTN and T2DM.[28] This further highlights the complex relationship between obesity and ACS, suggesting that various intricate factors may influence this association. A more nuanced understanding and comprehensive evaluation of obesity-related health risks are warranted in future research.

Previous studies have demonstrated that sleep deprivation and obesity serve as risk factors for high BP, T2DM, metabolic syndrome and ACS. The intricate interplay involves various mechanisms, including inflammation, immune response, hormonal alterations and autonomic nervous system activation, contributing to disease development.[29,30] In the joint effect analysis, the current study demonstrated a statistically significant increase in ACS risk when sleep insufficiency and obesity coexisted, compared to their independent presence. These findings supported the hypothesis that a combined effect of sleep insufficiency and obesity can influence ACS development, which may be mediated by several potential mechanisms. First, sleep insufficiency can influence health-related behaviours, such as dietary choices and physical activity levels. When combined with obesity, this could lead to a negative feedback loop that exacerbates cardiovascular health.[31] Second, obesity is known to worsen physiological stress responses, and sleep insufficiency may further intensify these responses, thus, further exacerbating inflammation and metabolic disturbances.[32] Lastly, sleep insufficiency and obesity may increase the risk of sleep-related disorders, such as sleep apnoea, which are associated with a higher CVD risk.[12] These potential mechanisms underpin the complex interplay among sleep, obesity and ACS, emphasising the need for a comprehensive approach to further enhance the current understanding of how these factors interact to influence health outcomes.

The current results have highlighted the importance of considering the individual risks of various factors, as well as potential synergistic effects when several risks coexist when exploring the complex relationship between lifestyle factors and disease risk. To prevent ACS, healthy sleep patterns and weight management should be promoted and recognised as crucial objectives in research and public health policies.

The current study has several limitations. First, it was based on self-reported data, which could have potentially introduced inaccuracies Owing to recall bias. Second, the study adjusted for some confounding variables, but dietary habits and stress levels, which could influence both sleep duration and BMI, were not considered. Third, due to specific demographic factors and geographic characteristics of the study population, the generalisability of the findings may be limited. Fourth, the study utilised data from the 9th follow-up survey, but some key variables, such as sleep duration and obesity, were initially collected during the baseline survey. Finally, there are potential limitations in the KoGES dataset, including selection bias, cohort effects, measurement errors, and data omissions.

Conclusion

A combined impact of insufficient sleep and obesity on ACS occurrence was noted, underscoring the importance of adequate sleep duration and weight management in the prevention of ACS.

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Authors' Contribution:
JYK, EJ, SL: Concept, design, data acquisition, analysis, interpretation, drafting, revision, final approval and agreement to be accountable for all aspects of the work.