

Long-Term Cardiovascular Outcomes of Obstructive Sleep Apnea by Adiposity Phenotype: A Korean Nationwide Cohort Study

Se-Jung Yoon

drpuoohsjy@gmail.com

Ilsan hospital, National Health Insurance Service <https://orcid.org/0000-0002-2398-0393>

Hyeongsoo Kim

Hancheol Lee

Taemi Youk

Jung Hyun Chang

Moojun Kim

Geunhee Park

Joongmin Kim

Ji-Yong Jang

Ilsan hospital, National Health Insurance Service

Jong-Kwan Park

Ilsan hospital, National Health Insurance Service

Seung-Jin Oh

Ilsan hospital, National Health Insurance Service

Article

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Abstract

Background

Obstructive sleep apnea (OSA) has been identified as an independent determinant of cardiovascular risk and the prevalence rate is steadily increasing. We aimed to assess differences in clinical characteristics and long-term cardiovascular outcomes between the clinically suspected OSA and control groups across detailed eight subgroups according to body mass index (BMI) and waist circumference (WC) with a propensity score–matched analysis using the entire Korean National Health Insurance (KNHI) database.

Methods

A total of 184,879 subjects with OSA were enrolled from the KNHI database between 2010 and 2021. Participants were classified into eight subgroups according to baseline BMI and WC. They were compared with control by 1:2 Propensity Score matching and followed up until 2023 for the occurrence of cardiovascular events.

Results

Among the total study population, the mean age of the OSA group was 47.2 years and several metabolic risk factors were significantly more prevalent in the OSA group. OSA HR / Control HR was significantly higher for angina in the severe obesity with high waist circumference (HWC) group. For atrial fibrillation, this ratio was significantly higher in both the underweight with low waist circumference (LWC) group and the severe obesity with HWC group. It was significantly higher for composite end point in the underweight with LWC group.

Conclusions

Higher cardiovascular outcomes were observed in subgroups at both extreme phenotypes of adiposity. Both the underweight with LWC group and the severe obesity with HWC group were significantly associated with higher incidence of atrial fibrillation.

Introduction

Obstructive sleep apnea (OSA) is a common disorder characterized by repeated collapse of the upper airway during sleep, leading to intermittent hypoxemia and disrupted sleep architecture. In the general population, the prevalence of OSA is estimated at 25–38% in men and 9–17% in women, with substantially higher rates observed among individuals with cardiovascular disease (CVD) or related risk factors such as obesity and hypertension [1–7].

Recent epidemiological projections indicate that the global burden of OSA will increase considerably over the next few decades. The prevalence is expected to rise to approximately 34.7 to 46.2% by 2050, corresponding to nearly 76.6 million affected individuals [8, 9]. OSA imposes a considerable burden on both healthcare systems and society due to its associated morbidity and economic impact [4]. Nevertheless, despite its widespread occurrence, OSA remains markedly underrecognized, which is particularly important given its role as a modifiable contributor to cardiovascular risk [1–3, 5–7].

As is well known, OSA has been identified as an independent determinant of cardiovascular risk. A large body of evidence indicates that OSA is associated with an increased likelihood of hypertension, coronary artery disease, stroke, heart failure, cardiac arrhythmias (including atrial fibrillation and ventricular arrhythmias), and sudden cardiac death. In particular, severe untreated OSA has been linked to a 2- to 3-fold higher risk of fatal cardiovascular events and all-cause mortality compared with individuals without OSA [1–3, 10–13]. Findings from large-scale cohort studies and observational analyses consistently support an elevated risk of cardiovascular events and mortality among patients with OSA, even after adjustment for

conventional risk factors [2, 3, 11]. Accordingly, the American Heart Association recommends screening for OSA in high-risk populations, including those with resistant hypertension, pulmonary hypertension, recurrent atrial fibrillation, heart failure (New York Heart Association class II–IV), and symptoms suggestive of sleep-disordered breathing [2, 3]. Considering its strong association with adverse cardiovascular outcomes, OSA should be viewed not merely as a condition affecting sleep quality but as a clinically significant and modifiable cardiovascular risk factor. From a broader perspective, it represents an important public health concern that requires greater attention within healthcare systems [12, 14].

The relationship between OSA and cardiovascular disease is explained by multiple interconnected pathophysiological processes. Recurrent episodes of apnea and hypopnea result in cyclical hypoxemia and reoxygenation, heightened sympathetic activation, marked intrathoracic pressure fluctuations, and frequent arousals from sleep. These repeated insults promote a cascade of chronic effects, including oxidative stress, systemic inflammation, endothelial dysfunction, metabolic abnormalities such as insulin resistance, and a prothrombotic state. Together, these alterations facilitate the initiation and progression of cardiovascular disease [2, 15–17].

However, despite its high prevalence and frequent clinical suspicion, OSA remains substantially underdiagnosed in clinical practice, which may be attributed to several factors as described below. Polysomnography (PSG), the current gold standard for diagnosing sleep disorders, is not readily accessible in many real-world settings. From the patient's standpoint, barriers such as limited time, restricted access to specialized centers, and financial burden are particularly relevant. As a result, the availability of testing is often limited, leading to delays in diagnosis [18]. Therefore, in the present study, we sought to evaluate cardiovascular outcomes in the individuals with clinically suspected OSA compared with a control group.

Furthermore, given the well-established association between OSA and obesity, obesity is the most important modifiable risk factor for OSA; increased body mass index (BMI), waist circumference (WC), and neck circumference are strongly associated with OSA prevalence and severity. A previous report has shown that even modest increases in body weight elevate the risk of OSA, whereas weight reduction may lower its incidence [3]. OSA and obesity share overlapping pathophysiological mechanisms, including systemic inflammation, sympathetic activation, endothelial dysfunction, and dyslipidemia, which together amplify cardiovascular risk [19–21]. Therefore, in designing a study on OSA, rather than simply comparing outcomes with a control group, a more precise and accurate analysis can be achieved by assessing and comparing risk according to detailed stratification based on BMI and WC. In this study, we aimed to assess differences in clinical characteristics and long-term cardiovascular outcomes between the clinically suspected OSA and control groups across detailed eight subgroups according to BMI and WC with a propensity score–matched analysis using the entire Korean National Health Insurance (KNHI) database.

Materials and Method

1. Statement of Ethics

This study was conducted in accordance with the principles of the Declaration of Helsinki and was reviewed and approved by the Institutional Review Board of the National Health Insurance Service Ilsan Hospital, Gyeonggi-do, Korea (NHIMC IRB 2024-11-004). The study utilized the entire Korean National Health Insurance Service (NHIS) database (NHIS-2025-02-1-003) as well as the National Health Insurance Service - Health Screening data provided by the NHIS. The requirement for written informed consent was waived, and all personal identifiers were anonymized during cohort construction in accordance with the regulations of the Korean Health Insurance Review and Assessment Service. The authors report no conflict of interest related to the NHIS.

2. Data Source

The National Health Insurance Database

The National Health Insurance Service (NHIS) operates a universal, single-insurer healthcare system in South Korea that mandatorily covers nearly all residents under a predominantly fee-for-service payment model. Insured individuals are classified into three categories: employee subscribers, self-employed subscribers, and recipients of the Medical Aid Program [22, 23]. Funded mainly through income-based insurance contributions, the NHIS currently provides coverage for about 97% of the Korean population, while the Medical Aid Program is supported by government funding for economically disadvantaged individuals [23].

The NHIS database contains extensive nationwide healthcare claims information, including diagnostic records, procedural data, prescription information, demographic variables, institutional characteristics, and medical costs from inpatient, outpatient, and dental services. Since all citizens are assigned a unique identification number at birth, healthcare utilization can be longitudinally monitored accurately without duplication or loss of records. Diseases in the NHIS database are coded using the Korean Classification of Diseases (KCD), which is adapted from and compatible with the International Classification of Diseases (ICD) system [22–25].

National health screening program

The national health screening program in Korea was first implemented in 1980 and was later broadened to include the general population. This program provides standardized medical evaluations composed of self-administered questionnaires regarding lifestyle and health-related behaviors, physical examinations including anthropometric measurements and blood pressure, and sensory assessments such as vision and hearing tests. Laboratory assessments include fasting glucose levels, lipid panels, liver function parameters, serum creatinine, urinalysis, and chest X-ray examinations. In addition, supplementary assessments such as bone densitometry and screening for cognitive dysfunction and depressive symptoms are included. The NHIS offers biennial health and cancer screening services at no cost to insured individuals aged 40 years or older and their eligible dependents [22, 23].

3. Study population

Individuals without prior medical claims related to OSA were identified from the NHIS database between 2010 and 2021. Nationwide data for newly diagnosed OSA cases were collected, including BMI, WC, blood pressure measurements, and laboratory findings obtained from the national health screening program. The study population was subsequently monitored through 2023 for the occurrence of cardiovascular events and mortality outcomes.

Individuals with a previous diagnosis of any cardiovascular disease (such as angina, coronary artery occlusive disease (CAOD), acute myocardial infarction (AMI), ischemic heart disease (IHD), peripheral artery occlusive disease (PAOD), atrial fibrillation, stroke, chronic kidney disease (CKD)), as well as diagnosis of any cancer within the two years prior to the index diagnosis were excluded (Fig. 1).

4. definition of OSA and cardiovascular disease.

The ICD codes for OSA were expressed as 'G47.3'. All subjects were followed for cardiovascular events, such as CAOD, AMI, IHD, PAOD, angina, atrial fibrillation, stroke, CKD, and cardiovascular mortality by searching KCD codes.

5. Classification according to BMI and waist circumference

Body mass index was derived from anthropometric measurements obtained during the national health examinations. Obesity was defined as a BMI ≥ 25.0 kg/m², calculated as body weight in kilograms divided by height in meters squared, based on the Asia-Pacific World Health Organization criteria and the guidelines of the Korean Society for the Study of Obesity [26–29].

For BMI-based analyses, participants were categorized into four groups: underweight ($< 18.5 \text{ kg/m}^2$), normal-to-overweight ($18.5\text{--}24.9 \text{ kg/m}^2$), obesity ($25.0\text{--}29.9 \text{ kg/m}^2$), and severe obesity ($\geq 30.0 \text{ kg/m}^2$) [28–29]. To avoid overly fragmented subgroup analyses, individuals with normal weight and overweight status were combined into a single category.

Waist circumference was dichotomized into lower and higher groups according to the sex-specific median (50th percentile) values. Using conventional waist circumference criteria [29] would have resulted in very small numbers of participants in some subgroups within our cohort. Therefore, in the present study, abdominal obesity was defined using the median waist circumference within each BMI group.

Based on the combination of baseline BMI and WC categories, participants were further classified into eight distinct groups as follows:

Group 1; Underweight ($\text{BMI} < 18.5 \text{ kg/m}^2$) with low waist circumference ($\text{LWC} < 50\%$)

Group 2; Underweight ($\text{BMI} < 18.5 \text{ kg/m}^2$) with high waist circumference ($\text{HWC} \geq 50\%$)

Group 3; Normal ($18.5 \text{ kg/m}^2 \leq \text{BMI} < 25 \text{ kg/m}^2$) with $\text{LWC} < 50\%$

Group 4; Normal ($18.5 \text{ kg/m}^2 \leq \text{BMI} < 25 \text{ kg/m}^2$) with $\text{HWC} \geq 50\%$

Group 5; Obesity ($25 \text{ kg/m}^2 \leq \text{BMI} < 30 \text{ kg/m}^2$) with $\text{LWC} < 50\%$

Group 6; Obesity ($25 \text{ kg/m}^2 \leq \text{BMI} < 30 \text{ kg/m}^2$) with $\text{HWC} \geq 50\%$

Group 7; Severe obesity ($30 \text{ kg/m}^2 \leq \text{BMI}$) with $\text{LWC} < 50\%$

Group 8; Severe obesity ($30 \text{ kg/m}^2 \leq \text{BMI}$) with $\text{HWC} \geq 50\%$

6. Statistical Analysis

After applying the predefined exclusion criteria, propensity score matching was performed at a 1:2 ratio between patients with OSA and controls based on age, sex, region of residence, and insurance premium quintile. Continuous variables are presented as mean $\pm$ standard deviation (SD), and categorical variables are expressed as frequencies and percentages.

Baseline characteristics among the eight subgroups defined by BMI and WC within both the OSA and control groups were compared separately using analysis of variance (ANOVA) for continuous variables and the chi-square test for categorical variables.

Cox proportional hazards regression models adjusted for diabetes mellitus, hypertension, dyslipidemia, CKD, age, sex, systolic blood pressure, diastolic blood pressure, fasting glucose level, smoking status and insurance premium quintile were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for cardiovascular event incidence and cardiovascular mortality within each cohort. The group with 'normal BMI and low waist circumference' was used as the reference category.

To evaluate the relative risk of cardiovascular outcomes in the OSA compared with the control group, ratios of hazard ratios were calculated by dividing the adjusted HR in the OSA by the corresponding adjusted HR in the control group. Confidence intervals for the ratios of HRs were estimated using Taylor's exact method.

All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant. All analyses were performed using SAS software (version 9.4; SAS Institute Inc., Cary, NC, USA).

Results

1. Demographic data of study population after 1:2 propensity score matching

A total of 184,879 people was enrolled and the median follow-up duration was 6.8 years. A control group of 369,758 individuals selected based on age, sex, region, and socioeconomic status using propensity score matching was used for comparison. The 40–54-year age group accounted for the largest proportion of the overall study population (42.1%) and males comprised 77.6% of the population (Table 1, Fig. 2).

Table 1
Demographic data of study population after 1:2 propensity score matching

		Before 1:2 PS Matching					After 1:2 PS Matching				
		OSA 184,879 (N/%)		Control 1,545,289 (N/%)		p-value	OSA 184,879 (N/%)		Control 369,758 (N/%)		p-value
Sex	M	143,383	77.6	1,170,455	75.7	< .001	143,383	77.6	286,766	77.6	1.000
	F	41,496	22.4	374,834	24.3		41,496	22.4	82,992	22.4	
Age	20–24	1,193	0.6	11,270	0.7	< .001	1,193	0.6	2,386	0.6	1.000
	25–29	8,612	4.7	71,007	4.6		8,612	4.7	17,229	4.7	
	30–34	21,303	11.5	165,413	10.7		21,303	11.5	42,606	11.5	
	35–39	23,350	12.6	184,841	12.0		23,350	12.6	46,700	12.6	
	40–44	25,809	14.0	208,640	13.5		25,809	14.0	51,618	14.0	
	45–49	26,020	14.1	211,698	13.7		26,020	14.1	52,040	14.1	
	50–54	25,844	14.0	217,152	14.1		25,844	14.0	51,688	14.0	
	55–59	22,996	12.4	201,214	13.0		22,996	12.4	45,992	12.4	
	60–64	15,214	8.2	137,916	8.9		15,214	8.2	30,428	8.2	
	65–69	8,503	4.6	79,824	5.2		8,503	4.6	17,006	4.6	
	70–74	3,874	2.1	38,247	2.5		3,874	2.1	7,748	2.1	
	75–79	1,640	0.9	14,526	0.9		1,640	0.9	3,280	0.9	
	80+	521	0.3	3,541	0.2		521	0.3	1,037	0.3	
Region of residence	Urban	97,607	52.8	778,574	50.4	< .001	97,607	52.8	195,209	52.8	0.992
	Non-urban	87,272	47.2	766,715	49.6		87,272	47.2	174,549	47.2	
Insurance premium quintile	Medical Aid	1,921	1.0	14,547	0.9	< .001	1,921	1.0	3,837	1.0	1.000
	Q1–Q5	22,080	11.9	230,639	14.9		22,080	11.9	44,160	11.9	
	Q6–Q10	29,271	15.8	308,887	20.0		29,271	15.8	58,542	15.8	
	Q11–Q15	52,084	28.2	460,279	29.8		52,084	28.2	104,168	28.2	
	Q16–Q20	79,523	43.0	530,937	34.4		79,523	43.0	159,051	43.0	
Q20 represents the highest insurance premium quantile.											

2. Clinical and laboratory data of 1:2 Propensity Score– matched groups

The mean age of the OSA group was 47.2 years, and the prevalence of diabetes, hypertension, dyslipidemia, and CKD, as well as height, body weight, waist circumference, waist-to-height ratio, BMI, and blood pressure, were significantly higher in the OSA group than in the 1:2 propensity score–matched control group (Table 2).

Table 2
Clinical and laboratory data of 1:2 Propensity Score– matched groups

		After 1:2 PS Matching				
		OSA (N = 184,879)		Control (N = 369,758)		p-value
Male (N/%)		143,383	77.6	286,766	77.6	1.000
Age (mean/sd)		47.2	11.8	47.2	11.8	0.826
Diabetes (N/%)		19,352	10.5%	32,778	8.9%	< .001
Hypertension (N/%)		44,566	24.1%	62,352	16.9%	< .001
Dyslipidemia (N/%)		48,095	26.0%	64,073	17.3%	< .001
CKD (N/%)		939	0.5%	1,166	0.3%	< .001
Smoking (N/%)	Non-smoker	82,061	44.4%	165,620	44.8%	< .001
	Ex-smoker	48,011	26.0%	79,900	21.6%	
	Current smoker	54,807	29.6%	124,238	33.6%	
Height (cm)		168.7	8.7	168.2	8.7	< .001
Weight (Kg)		73.9	14.0	69.3	12.4	< .001
Waist circumference (cm)		86.3	10.3	82.9	9.5	< .001
Waist circumference/height		0.51	0.05	0.49	0.05	< .001
BMI (kg/m ²)		25.9	7.0	24.4	3.3	< .001
SBP (mmHg)		124.3	13.9	123.1	13.9	< .001
DBP (mmHg)		78.3	10.2	77.1	9.8	< .001
Fasting glucose (mg/dL)		99.8	20.9	100.0	24.5	0.013
GGT (IU/L)		47.1	49.3	44.0	55.6	< .001
Hemoglobin (g/dL)		14.9	1.4	14.8	1.5	< .001
SGOT (IU/L)		28.3	21.8	26.9	25.6	< .001
SGPT (IU/L)		33.8	30.9	29.0	28.7	< .001
Abbreviations: OSA, obstructive sleep apnea; CKD, chronic kidney disease; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; GGT, gamma-glutamyl transferase; SGOT, aspartate aminotransferase; SGPT, alanine aminotransferase.						

3. Demographic and laboratory data of eight subgroups (1:2 Propensity Score– Matched)

The prevalence of diabetes, hypertension, and dyslipidemia, as well as anthropometric and metabolic parameters—including height, body weight, waist circumference, waist-to-height ratio, BMI, blood pressure, fasting glucose, gamma-glutamyl

transferase (GGT), aspartate aminotransferase (AST), and alanine aminotransferase (ALT)—showed a gradual increasing trend with increasing BMI and waist circumference. Similar trends were also observed in the control group (Table 3, 4).

Table 3

Demographic and laboratory data of eight subgroups (1:2 Propensity Score–Matched) – obstructive sleep apnea

OSA (N = 184,879)									
	UW & LWC	UW & HWC	Normal weight & LWC	Normal weight & HWC	Obesity & LWC	Obesity & HWC	Severe obesity & LWC	Severe obesity & HWC	p- value
	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	
Total	850 (0.5)	1,090 (0.6)	32,722 (17.7)	46,512 (25.2)	32,434 (17.5)	48,045 (26.0)	9,446 (5.1)	13,780 (7.5)	< .001
Diabetes (N/%)	47 (5.5)	84 (7.7)	1,892 (5.8)	4,600 (9.9)	2,786 (8.6)	6,220 (12.9)	1,171 (12.4)	2,552 (18.5)	< .001
Hypertension (N/%)	55 (6.5)	99 (9.1)	3,804 (11.6)	9,618 (20.7)	7,318 (22.6)	14,941 (31.1)	3,153 (33.4)	5,578 (40.5)	< .001
Dyslipidemia (N/%)	105 (12.4)	156 (14.3)	5,761 (17.6)	12,114 (26.0)	8,038 (24.8)	14,525 (30.2)	2,804 (29.7)	4,592 (33.3)	< .001
CKD (N/%)	9 (1.1)	7 (0.6)	135 (0.4)	251 (0.5)	130 (0.4)	267 (0.6)	50 (0.5)	90 (0.7)	< .001
Height (cm)	162.8 (7.5)	165.0 (8.3)	166.6 (8.5)	168.5 (9.0)	167.9 (8.0)	170.5 (8.4)	168.4 (8.6)	171.6 (8.7)	< .001
Weight (Kg)	46.2 (5.0)	48.5 (5.2)	60.9 (7.9)	66.8 (8.1)	74.4 (7.5)	80.1 (8.6)	89.1 (9.1)	99.9 (13.3)	< .001
WC (cm)	61.7 (3.4)	69.4 (5.1)	74.0 (4.9)	83.4 (6.2)	84.0 (3.6)	92.8 (4.1)	93.9 (4.5)	106.2 (10.3)	< .001
WC/height	0.38 (0.02)	0.42 (0.03)	0.44 (0.03)	0.50 (0.04)	0.50 (0.02)	0.55 (0.03)	0.56 (0.06)	0.63 (0.07)	< .001
BMI (kg/m ²)	17.4 (0.9)	17.8 (0.7)	21.9 (1.6)	23.4 (1.2)	26.3 (1.1)	27.5 (1.3)	31.6 (2.6)	33.9 (3.3)	< .001
SBP (mmHg)	110.7 (13.0)	112.9 (13.8)	117.9 (13.0)	122.0 (13.1)	125.0 (12.9)	127.3 (13.1)	130.4 (13.7)	133.1 (14.6)	< .001
DBP (mmHg)	70.1 (9.0)	71.0 (9.3)	73.9 (9.4)	76.4 (9.4)	78.8 (9.6)	80.3 (9.8)	82.7 (10.5)	84.5 (11.2)	< .001
Fasting glucose (mg/dL)	89.9 (14.7)	91.4 (15.9)	94.0 (16.2)	98.0 (19.1)	99.5 (19.7)	102.9 (22.0)	104.1 (22.5)	108.3 (28.3)	< .001
GGT (IU/L)	21.8 (28.7)	26.5 (58.1)	29.7 (36.4)	39.4 (44.3)	48.2 (46.7)	57.2 (53.8)	63.4 (53.6)	69.2 (57.1)	< .001
Hemoglobin (g/dL)	13.5 (1.4)	13.6 (1.4)	14.4 (1.5)	14.6 (1.4)	15.0 (1.3)	15.2 (1.3)	15.3 (1.4)	15.4 (1.4)	< .001
OSA, obstructive sleep apnea; UW, Underweight; LWC, low waist circumference; HWC, high waist circumference; CKD, chronic kidney disease; WC, Waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; GGT, gamma-glutamyl transferase; SGOT, aspartate aminotransferase; SGPT, alanine aminotransferase.									

OSA (N = 184,879)									
	UW & LWC	UW & HWC	Normal weight & LWC	Normal weight & HWC	Obesity & LWC	Obesity & HWC	Severe obesity & LWC	Severe obesity & HWC	p- value
SGOT (IU/L)	23.3 (14.2)	23.8 (19.3)	24.1 (19.4)	25.7 (17.9)	27.9 (24.4)	30.2 (23.5)	33.9 (19.5)	38.2 (24.6)	< .001
SGPT (IU/L)	17.2 (23.9)	18.5 (22.2)	22.3 (21.8)	27.0 (19.9)	33.6 (33.3)	39.1 (32.2)	49.1 (35.9)	57.3 (42.6)	< .001
OSA, obstructive sleep apnea; UW, Underweight; LWC, low waist circumference; HWC, high waist circumference; CKD, chronic kidney disease; WC, Waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; GGT, gamma-glutamyl transferase; SGOT, aspartate aminotransferase; SGPT, alanine aminotransferase.									

Table 4
Demographic and laboratory data of eight subgroups (1:2 Propensity Score–Matched) – control

Control (N = 369,758)									
	UW & LWC	UW & HWC	Normal weight & LWC	Normal weight & HWC	Obesity & LWC	Obesity & HWC	Severe obesity & LWC	Severe obesity & HWC	p- value
	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	N (%), Mean (sd)	
Total	3,716 (1.0)	4,335 (1.2)	107,340 (29.0)	106,786 (28.9)	59,721 (16.2)	67,836 (18.3)	9,580 (2.6)	10,444 (2.8)	< .001
Diabetes (N/%)	125 (3.4)	250 (5.8)	5,274 (4.9)	10,429 (9.8)	4,877 (8.2)	8,921 (13.2)	1,077 (11.4)	1,814 (17.4)	< .001
Hypertension (N/%)	148 (4.0)	315 (7.3)	9,103 (8.5)	18,520 (17.3)	10,680 (17.9)	17,691 (26.1)	2,508 (26.2)	3,387 (32.4)	< .001
Dyslipidemia (N/%)	238 (6.4)	393 (9.1)	11,936 (11.1)	2,253 (19.0)	10,920 (18.0)	15,645 (23.1)	2,083 (21.7)	2,605 (24.9)	< .001
CKD (N/%)	9 (0.2)	17 (0.4)	271 (0.3)	376 (0.4)	153 (0.3)	272 (0.4)	23 (0.2)	45 (0.4)	< .001
Height (cm)	164.9 (8.2)	167.2 (8.9)	167.0 (8.2)	168.3 (9.0)	167.8 (8.1)	170.1 (8.7)	168.5 (8.8)	171.5 (9.1)	< .001
Weight (Kg)	47.2 (5.2)	49.7 (5.6)	60.6 (7.5)	66.2 (8.0)	74.0 (7.4)	79.2 (8.8)	88.8 (9.4)	97.8 (12.8)	< .001
WC (cm)	63.1 (3.4)	70.7 (4.5)	74.1 (4.7)	83.1 (7.1)	83.7 (3.6)	92.4 (4.0)	93.6 (4.6)	104.9 (5.9)	< .001
WC/height	0.38 (0.02)	0.42 (0.03)	0.44 (0.03)	0.50 (0.04)	0.50 (0.02)	0.54 (0.03)	0.56 (0.03)	0.61 (0.04)	< .001
BMI (kg/m ²)	17.3 (0.9)	17.7 (0.7)	21.6 (1.6)	23.3 (1.2)	26.2 (1.0)	27.3 (1.3)	31.2 (1.3)	33.2 (2.8)	< .001
SBP (mmHg)	112.8 (13.3)	115.6 (14.1)	118.8 (13.1)	122.5 (13.5)	125.1 (13.0)	127.4 (13.4)	130.3 (14.0)	132.7 (14.4)	< .001
DBP (mmHg)	71.3 (9.1)	72.6 (9.7)	74.4 (9.3)	76.5 (9.4)	78.5 (9.4)	79.9 (9.7)	82.1 (10.2)	83.7 (10.7)	< .001
Fasting glucose (mg/dL)	92.1 (20.7)	94.3 (23.0)	95.4 (20.5)	100.2 (24.8)	100.5 (22.7)	104.9 (27.8)	105.5 (27.9)	110.0 (32.9)	< .001
GGT (IU/L)	26.5 (47.4)	36.4 (75.9)	33.1 (48.9)	42.3 (56.4)	47.8 (53.2)	56.2 (59.2)	60.8 (54.1)	67.3 (64.5)	< .001
OSA, obstructive sleep apnea; UW, Underweight; LWC, low waist circumference; HWC, high waist circumference; CKD, chronic kidney disease; WC, Waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; GGT, gamma-glutamyl transferase; SGOT, aspartate aminotransferase; SGPT, alanine aminotransferase.									

Control (N = 369,758)									
	UW & LWC	UW & HWC	Normal weight & LWC	Normal weight & HWC	Obesity & LWC	Obesity & HWC	Severe obesity & LWC	Severe obesity & HWC	p- value
Hemoglobin (g/dL)	13.9 (1.5)	14.0 (1.6)	14.5 (1.5)	14.6 (1.5)	15.0 (1.4)	15.1 (1.4)	15.2 (1.4)	15.4 (1.4)	< .001
SGOT (IU/L)	23.5 (21.4)	25.9 (25.9)	24.5 (23.1)	26.0 (28.4)	27.4 (30.0)	29.5 (19.7)	32.9 (23.9)	36.6 (25.1)	< .001
SGPT (IU/L)	17.4 (14.3)	19.4 (18.6)	22.1 (25.9)	26.5 (23.4)	31.7 (33.9)	36.6 (27.5)	46.1 (38.0)	53.2 (41.6)	< .001
OSA, obstructive sleep apnea; UW, Underweight; LWC, low waist circumference; HWC, high waist circumference; CKD, chronic kidney disease; WC, Waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; GGT, gamma-glutamyl transferase; SGOT, aspartate aminotransferase; SGPT, alanine aminotransferase.									

4. Comparison of Hazard ratio of cardiovascular event in two groups

OSA HR / Control HR was significantly higher for angina in the severe obesity with HWC group (HR, 1.17; 95% CI, 1.02–1.36). For atrial fibrillation, this ratio was significantly higher in both the underweight with LWC group (HR, 2.95; 95% CI, 1.32–6.57) and the severe obesity with HWC group (HR, 1.48; 95% CI, 1.07–2.06).

It was significantly higher for composite end point in the underweight with LWC group (HR, 1.37; 95% CI, 1.00–1.86) (Fig. 3 and supplementary Tables).

Discussion

To our knowledge, this is the first large-scale cohort study to investigate the clinical characteristics and long-term cardiovascular outcomes of patients with clinically suspected OSA according to BMI and WC-specific phenotypes compared with a 1:2 propensity score-matched control population. The present study included a large proportion of patients who were clinically diagnosed with OSA based on symptoms in routine clinical practice without undergoing PSG, the gold standard diagnostic modality for OSA, owing to the high cost and limited accessibility of PSG. In Korea, national health insurance coverage for PSG and positive airway pressure therapy was introduced in July 2018 and has gradually expanded thereafter; however, the present study included patients enrolled between 2010 and 2021. Therefore, this study is characterized by the inclusion of a large proportion of patients who were enrolled based on clinical diagnosis during a period when many patients were unable to undergo PSG.

Interestingly, among the various cardiovascular outcomes evaluated, atrial fibrillation demonstrated a distinct pattern in which the incidence was significantly higher in the OSA group than in the matched control group specifically at the two extremes of adiposity phenotypes - the extremely underweight with LWC group and the severely obese with HWC group.

OSA is well known as an independent and modifiable risk factor for atrial fibrillation, with increasing severity of sleep-disordered breathing associated with a higher incidence and recurrence of atrial fibrillation. People with OSA had an approximately two- to four-fold higher risk of developing new-onset atrial fibrillation than those without OSA [30–34]. The relationship between OSA and atrial fibrillation is complex and involves several metabolic, inflammatory, autonomic, and neurohumoral factors [35]. However, despite the well-established association between OSA and atrial fibrillation [36, 37], it was unexpected that the OSA group did not demonstrate a significantly higher incidence of atrial fibrillation across all adiposity phenotypes compared with the control group without OSA in this study.

The association between underweight status and atrial fibrillation has also been previously reported. In a nationwide population-based study including 132,063 individuals from the Korean NHIS database, a U-shaped relationship was observed between BMI and incident atrial fibrillation, with an increased risk of atrial fibrillation in underweight individuals with a BMI < 18.5 kg/m². Furthermore, the risk of atrial fibrillation in the underweight group was reported to be approximately 23% higher than that in individuals with normal body weight [38]. Another Korean study involving 196,136 patients with new-onset atrial fibrillation also demonstrated a U-shaped association between BMI and atrial fibrillation risk and reported that the risk was further increased when diabetes mellitus coexisted with underweight [39].

Moreover, a cohort study from Taiwan including 64,339 patients with type 2 diabetes mellitus demonstrated that the underweight group confers the highest risk of developing atrial fibrillation in Asian patients with type 2 diabetes mellitus. In this study, underweight individuals showed a 52% higher risk of incident atrial fibrillation compared with those with normal BMI (HR 1.52), and the relationship between BMI and atrial fibrillation risk demonstrated a J-shaped or L-shaped pattern [40].

In our study, the incidence of atrial fibrillation did not increase progressively with increasing BMI compared with the control group. Rather, both the extremely underweight with LWC group and the severely obese with HWC group demonstrated higher atrial fibrillation incidence rates than control group without OSA. These findings may reflect the combined effects of adverse risk factors associated with underweight status, including frailty, sarcopenia, chronic inflammation, autonomic imbalance, and latent systemic disease.

The diagnosis of OSA was primarily based on medical records and ICD codes, without universal confirmation by PSG in this study. Although PSG is considered the gold standard for the diagnosis of OSA, many patients in real-world settings are initially diagnosed based on clinical features and surveys. Polysomnography differs from routine diagnostic approaches in that it requires overnight monitoring in a specialized laboratory setting with dedicated equipment and trained personnel, and accurate interpretation depends on experienced sleep specialists. In addition, the hospital-based environment and attachment of multiple sensors may cause discomfort and alter habitual sleep patterns. Consequently, the limited use of PSG in routine clinical practice is influenced by accessibility, cost, and patient tolerability, leaving many individuals with suspected OSA undiagnosed or without confirmatory testing despite appropriate clinical indications [18].

Although the diagnosis of OSA based exclusively on diagnostic codes in the absence of PSG confirmation can be susceptible to underdiagnosis and misclassification bias, however given the limited accessibility of PSG in many countries, numerous studies have already been conducted to evaluate the accuracy of clinical diagnosis for OSA. A study of United States validated the performance of a simple electronic health record (EHR)-based case-identification algorithm across multiple institutions and reported excellent predictive characteristics compared with PSG-confirmed OSA [41]. Similarly, a comparable study evaluated the accuracy of OSA diagnosis derived from a coded database against actual specialist diagnosis, reporting a positive predictive value (PPV) of approximately 75–85% for the OSA identification algorithm in UK [42]. Another research investigating the agreement between clinical assessment and polysomnographic findings in adult patients with OSA has consistently shown that nocturnal dyspnea and choking or gasping sensations constitute the most diagnostically reliable predictors of OSA [43].

The American Academy of Sleep Medicine (AASM) guideline recommends that clinical suspicion alone has limited specificity for OSA diagnosis when compared with PSG, the current diagnostic standard. However, validated EHR-based algorithms using repeated diagnostic codes have shown high positive predictive values for identifying clinically diagnosed OSA [18]. Therefore, evaluating and analyzing cardiovascular outcomes in large-scale epidemiologic studies of OSA diagnosed in real-world clinical practice may be of considerable practical importance and the impact of this limitation on major outcomes can be less pronounced.

As a comparison of atrial fibrillation incidence according to heterogeneous BMI phenotypes, a study published in 2021 demonstrated that both overall obesity and abdominal obesity were associated with an increased risk of atrial fibrillation, and that BMI and body weight themselves were strong predictors of atrial fibrillation development [44]. However, to date, no study

has ever specifically evaluated differences in atrial fibrillation incidence according to combined BMI and WC phenotypes in patients with OSA.

We reported that the relationship between adiposity phenotype and atrial fibrillation risk in patients with OSA may not be linear, and both extremes of body habitus may confer increased arrhythmic vulnerability in this study.

Study Limitations

The major limitation of this present study is that OSA was not confirmed by PSG. Although previous validation studies have demonstrated moderate-to-high positive predictive values for coded OSA diagnoses compared with specialist or PSG-based diagnoses [18, 32, 42, 43], it is unavoidable that the diagnostic specificity may be lower in patients enrolled based on clinical diagnosis—derived from subjective reports by patients and their family together with physician assessment—rather than PSG, which remains the current gold standard for the diagnosis of OSA.

Another important limitation is the potential inaccuracy of diagnoses based solely on administrative diagnostic codes compared with diagnoses confirmed through detailed medical record review. Because this dataset relies on computerized diagnostic codes entered by a person during routine clinical practice and insurance claim processes, errors such as incomplete coding, underestimation, or misclassification of medical conditions may occur.

In the BMI-based classification used in the present study, the normal-weight and overweight groups were combined to avoid excessive subgroup stratification; however, future studies with more finely subdivided BMI categories may provide additional insights. In addition, the substantial imbalance in sample sizes among the eight subgroups may have affected the study findings.

In addition, the substantial imbalance in sample sizes among the eight subgroups may have affected the study findings. Furthermore, evidence regarding the association between underweight and atrial fibrillation has been derived mainly from Asian populations, and comparable data from Western countries are still limited. Further investigations across diverse ethnic groups are therefore warranted to clarify whether the relationship between underweight and atrial fibrillation is consistent beyond Asian populations. Another limitation can be the possibility of reverse causation, it should also be carefully considered when interpreting the poor cardiovascular outcomes observed in the underweight with LWC phenotype. Underlying systemic diseases or chronic health conditions that contribute to low body weight may themselves have contributed to the adverse cardiovascular outcomes, rather than these outcomes being attributable to the OSA–underweight/LWC phenotype. To mitigate this concern, we excluded individuals with a history of cardiovascular disease or cancer identified during health screening and those without available health screening or baseline clinical data during the approximately 2 years preceding enrollment. However, residual confounding by unrecognized or subclinical conditions cannot be completely excluded.

Lastly, although efforts were made to obtain reliable and clinically relevant information from the claims database, including BMI and WC data, further prospective clinical studies, even in smaller populations, are warranted to validate the overall findings of the present study.

Conclusion

Our study demonstrated a distinct pattern of cardiovascular outcome in patients with OSA, in which the risk of atrial fibrillation and angina was significantly increased at both extremes of adiposity phenotypes, namely the extremely underweight with LWC group and severely obese with HWC groups, compared with matched controls. These findings also suggest that the relationship between adiposity phenotype and atrial fibrillation risk in OSA may not be linear, and that both extremes of body habitus may confer increased arrhythmic vulnerability.

Abbreviations

OSA Obstructive sleep apnea

BMI Body mass index

WC Waist circumference

KNHI Korean National Health Insurance

HWC High waist circumference

LWC Low waist circumference

PSG Polysomnography

NHIS National Health Insurance Service

KCD Korean Classification of Diseases

ICD International Classification of Diseases

CAOD Coronary artery occlusive disease

AMI Acute myocardial infarction

IHD Ischemic heart disease

PAOD Peripheral artery occlusive disease

CKD Chronic kidney disease

ANOVA Analysis of variance

HR Hazard ratio

CI Confidence interval

GGT Gamma-glutamyl transferase

AST Aspartate aminotransferase

ALT Alanine aminotransferase

EHR Electronic health record

PPV Positive predictive value

AASM American Academy of Sleep Medicine

Declarations

Conflicts of Interest: The authors declare no conflicts of interest.

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Data Availability Statement: Datasets supporting the findings of this article cannot be shared openly due to the data management policy of the NHIS of Korea but are available from the corresponding author upon reasonable request.

Author contributions: Study concept and design: SJY, JYJ; acquisition of data: MK, JHH and HL; analysis and interpretation of data: SJY, JKP, and GP; drafting of the manuscript: SJY, JK and MK; critical revision of the manuscript: HK, HL; statistical analysis: JHH, SO and JKP; obtained funding: SJY; administrative, technical, or material support: JHJ, JYJ and JK; and study supervision: SO, GP and HK.

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Figures

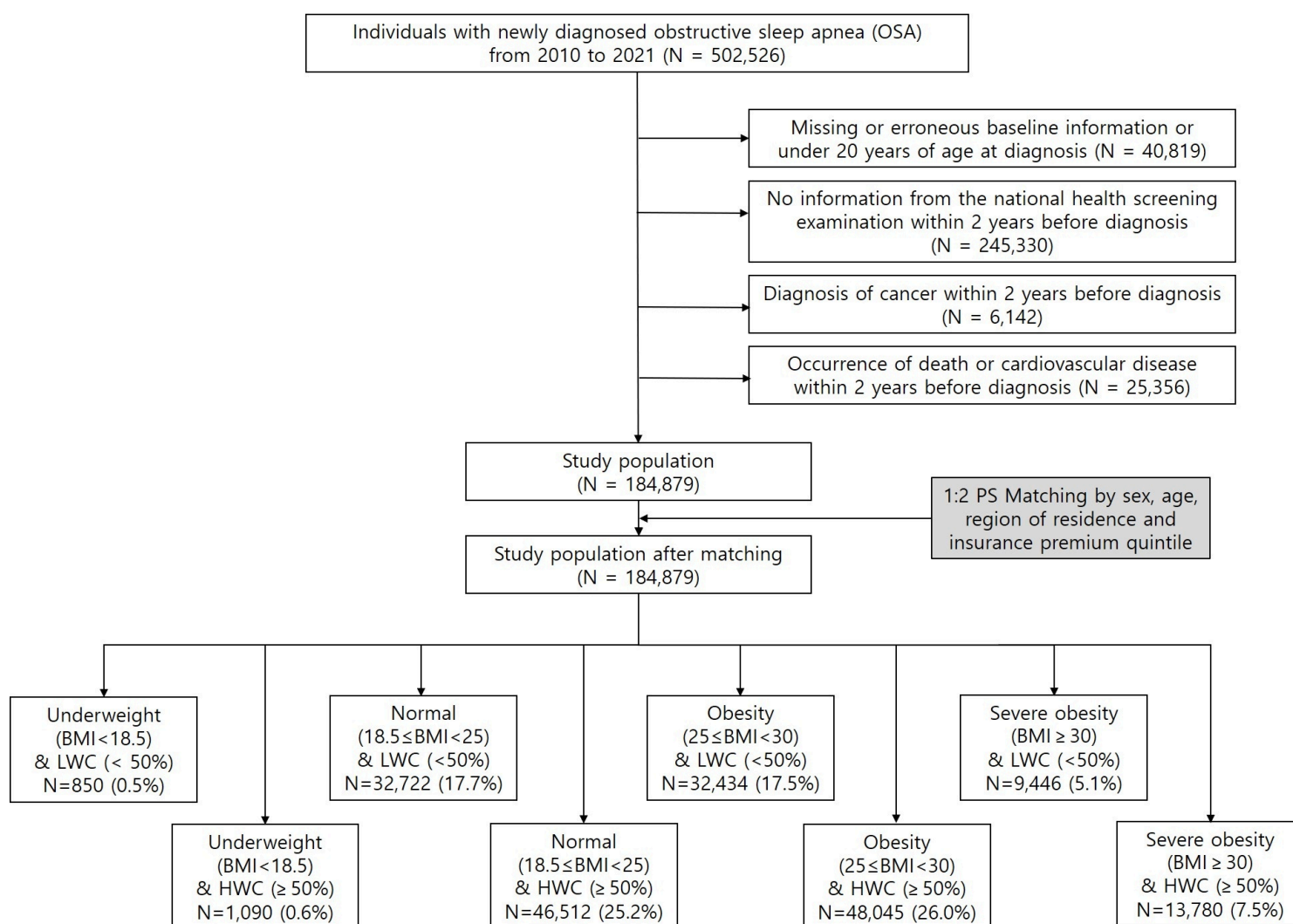
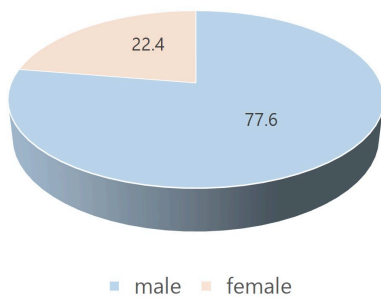


Figure 1

Flow chart for inclusion and exclusion of study participants

A

Prevalence and distribution by sex (%)

**B**

Prevalence and distribution by age (%)

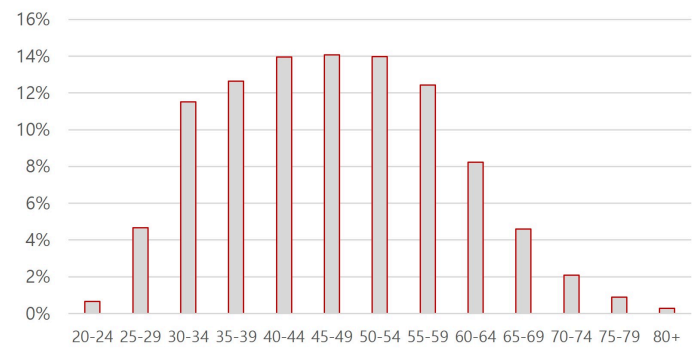


Figure 2

Demographic data of obstructive sleep apnea group

OSA HR / Control HR (reference : Normal BMI & low waist circumference)

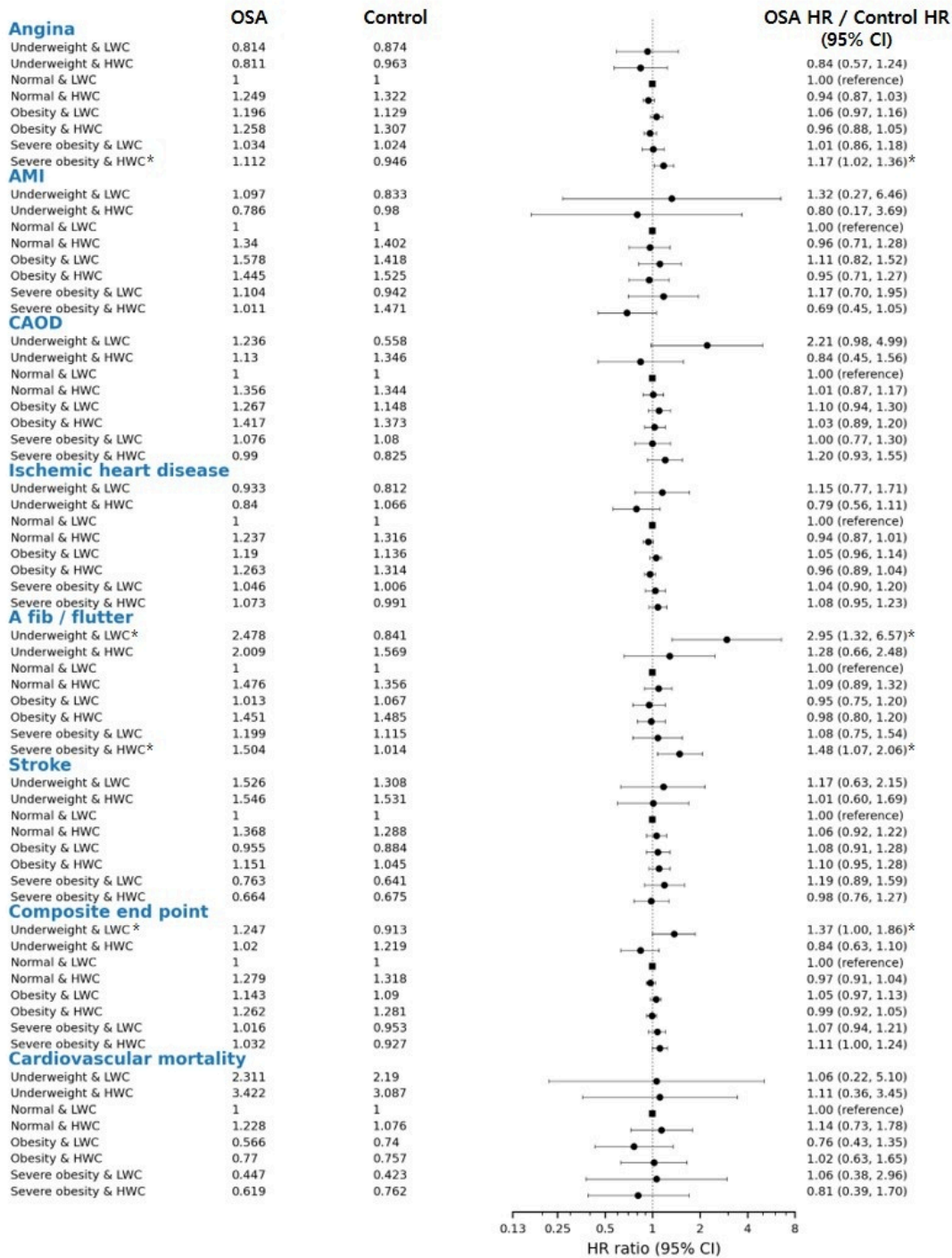


Figure 3

OSA HR / Control HR of cardiovascular event in two groups

OSA, obstructive sleep apnea; BMI, body mass index; LWC, low waist circumference; HWC, high waist circumference; AMI, acute myocardial infarction; CAOD, coronary artery occlusive disease; A fib, atrial fibrillation. Asterisk means p-value < 0.05.

Supplementary Files

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