Comparing Obstructive Versus Central Apneas in Obesity of Sleep—A Nationwide Population-Based Case Control Study

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Abstract

Objectives: This study used a case control Study to compare whether obstructive sleep apnea (OSA) or central sleep apnea (CSA) are related with an increased risk of obesity.

Materials and Methods: We used 2005 Longitudinal Health Insurance Database (LHID2005), which is part of the Taiwan National Health Insurance Research Database (NHIRD) to identified 24 363 obese patients; 97 452 patients without obesity were identified also, from the same database from January 1, 2000, to December 31, 2015. Age, sex, and index date were matched. Multiple logistic regression was used to analyze the previous exposure risk of OSA or CSA with obesity and. A *p* value of <.05 was considered significant.

Results: Obesity patients have higher risk to be exposed to OSA than did those with CSA (OSA adjusted OR [AOR] = 2.927, 95% CI = 1.878-4.194, *p* < .001; CSA adjusted OR [AOR] = 2.234, 95% CI = 1.483-4.380, *p* < .001). Furthermore, the closeness of the exposure period to the index time was positively associated with the severity of obesity, with a dose–response effect (OSA exposure <1 year, AOR = 3.895; OSA exposure ≥1 year and <5 years, AOR = 2.933; OSA exposure ≥5 years, AOR = 2.486 ; CSA exposure <1 year, AOR = 2.484; CSA exposure ≥1 year and <5 years, AOR = 2.105; CSA exposure ≥5 years, AOR = 1.862). The exposure duration of OSA in patients with obesity was 2.927 times than that of CSA was 2.234 times. Longer exposure durations were associated with more severe obesity with a dose–response effect (OSA exposure <1 year, AOR = 2.251; OSA exposure ≥1 year and <5 years, AOR = 2.986; OSA exposure ≥5 years, AOR = 3.452; CSA exposure <1 year, AOR = 2.101; CSA exposure ≥1 year and <5 years, AOR = 2.207; CSA exposure ≥5 years, AOR = 2.976).

Conclusions: OSA patients have significantly higher risk of Obesity than that of CSA in this study. Longer exposure to OSA or CSA was related with a higher likelihood of obesity, with a dose-response effect.

1. Introduction

Obese adults have an increased risk of death due to various acute and chronic diseases (including hypertension, dyslipidemia, coronary heart disease, diabetes, gallbladder disease, gout, arthritis, and respiratory diseases).[1] In the latest survey, the National Health Administration of the Ministry of Health and Welfare reported that the rate of obesity among adults (≥18 years of age) has increased from 38% in 2009 to 43.9% in 2018.[2] Obesity is an increasingly common problem because, for many people, modern life involves eating lots of cheap, high-calorie foods and spending a lot of time sitting at a desk, sofa or car.[2] In addition, it is now well established that obesity (depending on the degree, duration and distribution of excess weight/adipose tissue) progressively causes and/or exacerbates a wide range of comorbidities, including type 2 diabetes, hypertension, dyslipidemia, cardiovascular disease, non-alcoholic Fatty liver disease, reproductive dysfunction, respiratory abnormalities, mental illness, and even increase the risk of certain types of cancer.[2]

Obstructive sleep apnea (OSA) is classified as a sleep-related respiratory system disease and is divided into two categories (adult OSA and pediatric OSA) in the third edition of the International Classification of Sleep Disorders (ICSD-3).[3] Obstructive sleep apnea (OSA) is characterized by episodes of complete collapse of the airway or partial collapse with an associated decrease in oxygen saturation or arousal from sleep. This disturbance results in fragmented, nonrestorative sleep. OSA has significant implications for cardiovascular health, mental illness, quality of life, and driving safety. [4]

Central sleep apnea (CSA) is characterized by insufficient respiratory function during sleep leading to repeated hypoventilation and impaired gas exchange. [5] CSA is characterized by reduced or stopped breathing due to reduced effort rather than an upper airway obstruction.[5] It is necessary to evaluate respiration to correctly classify apnea as obstructive or central, considering the specificity of muscle activity in the absence of airflow and whether respiration continues or increases. [6-7] CSA occurs when the level of chemical drive is insufficient to initiate ventilation.[8-13] Consequently, any neurologic condition affecting the anatomic sites of chemo sensation and ventilatory control could manifest as CSA, and such events are almost always hypercapnic in nature.[14-15]

Longitudinal observational studies investigating the relationship between OSA, or CSA and obesity are limited. Therefore, we hypothesized that Obesity patients have higher risk to be exposed to OSA than CSA. We used the National Health Insurance Research Database (NHIRD) of the Ministry of Health and Welfare to investigate whether OSA or CSA increases the subsequent risk of obesity.

2. Materials And Methods

2.1 Data source

Taiwan's National Health Insurance launched the single-payer system on March 1, 1995. As of 2017, 99.9% of Taiwan's population is enrolled in this program. Data for this study were collected from the 2005 Longitudinal Health Insurance Database (LHID2005), which is part of the NHIRD, and 2 000 000 people were randomly selected from the entire population. The National Institutes of Health encrypts all personal information before releasing the LHID2005 to protect the privacy of patients. In the LHID2005, the disease diagnosis code is based on the "International Classification of Diseases, Ninth Revision, Clinical Modification" (ICD-9-CM) criteria.[16] The flowchart of study design (nested case-control study) from the National Health Insurance Research Database in Taiwan (Fig. 1). All methods were carried out following relevant guidelines and regulations. The Ethical Review Board of the Tri-Service General Hospital of the National Defense Medical Center (TSGHIRB No. B-109-39) approved this study.

2.2 Determining cases and controls

Patients diagnosed as having obesity (ICD-9-CM code 278) were defined as an obesity case group. The control group consisted of patients without obesity. Patients in both the case and control groups were

matched by the index date, sex, and age at a ratio of 1:4.

2.3 Identifying SDs, obesity, and comorbidities

The risk factor discussed in this study is SD, which is defined based on at least 3 outpatient diagnoses from 2000 to 2015, identified using the ICD-9 codes 780.5 (SDs); 780.50 (SDs, not specified); 780.52 (insomnia, not specified); 780.51, 780.53, and 780.57 (sleep apnea syndrome); 307.4 (specific SDs of nonorganic origin); 780.54 (insufficient sleep, unspecified); 780.55 (24-hour sleep-wake cycle interruption, unspecified); 780.56 (dysfunction related to the sleep phase or awakening from sleep); 780.58 (dyskinesia related to sleep, unspecified); and 780.59 (SDs, other).

The outcome of obesity was measured in patients diagnosed as having the following conditions: overweight, obesity, and other hyperalimentation (ICD-9-CM code 278); overweight and obesity (ICD-9 CM code 278.0); morbid obesity (ICD-9-CM code 278.01); overweight (ICD-9-CM code 278.02); and obesity hypoventilation syndrome (ICD-9-CM code 278.03).

The comorbidities evaluated in this study were DM (ICD-9-CM code 250), hypertension (ICD-9-CM code 401-405), hyperlipidemia (ICD-9-CM code 272.4), CAD (ICD-9-CM code 414.01), stroke (ICD-9-CM code 430-438), chronic heart failure (ICD-9-CM code 428.0), chronic obstructive pulmonary disease (ICD-9-CM code 490-496), CKD (ICD-9-CM code 585), liver cirrhosis (ICD-9-CM code 571.5), tumor (ICD-9-CM code 199), anxiety (ICD-9-CM code 300.00), and depression (ICD-9-CM codes 296.2-296.3, 300.4, and 311).

2.4 Statistical analysis

Descriptive data are presented as percentages, means, and standard deviations. The chi-square test and *t* test were used to evaluate the distribution of categorical and continuous variables between cases and controls. Conditional logistic regression analyses were performed to evaluate the effect of OSA or CSA on the risk of obesity after adjusting for age, sex, education, insured premium, comorbidities, Charlson Comorbidity Index (CCI), season, location, urbanization level, and level of care. The effect of the first to last OSA or CSA exposure before obesity diagnosis on the factors of obesity was examined using conditional logistic regression. All analyses were performed using SPSS version 22 (IBM, Armonk, NY, USA). A p value of <.05 was considered significant.

3. Results

3.1 Demographic data

As presented in Table 1, in the average age of 121815 patients, cases was 44.25 ± 15.53 years and controls was 44.31 ± 15.67 years, among whom 42.77% were men and 57.23% were women. We recruited 24 363 patients with obesity (cases) and 97 452 patients without obesity (controls). Patients in the case

group had a higher prevalence of comorbidities than did those in the control group. In the case group, the CCI and season were significant.

Table 1

Demographic characteristics among patients

Obesity	Cases		Controls		*p Value*
Variables	**n**	**%**	**n**	**%**	
Total	24 363	20.00	97 452	80.00	
OSA					
Without	22 572	92.65	96 708	99.24	
With	1 791	7.35	74	0.76	
CSA					<0.001
Without	24 274	99.63	97 431	99.98	
With	89	0.37	21	0.02	
Sex					
Male	10421	42.77	41684	42.77	0.999
Female	13942	57.23	55768	57.23	
Age (years)	44.25 ± 15.53		44.31 ± 15.67		0.592
Age group (yrs)					
20-44	14827	47.48	59308	47.48	0.999
45-64	6866	21.99	27464	21.99	
≥65	9536	30.54	38144	30.54	
Comorbidities					<0.001
CCI_R					
OSA	0.07 ± 0.38		0.05 ± 0.26		<0.001
CSA	0.06 ± 0.35		0.05 ± 0.24		<0.001
Season					<0.001
OSA					<0.001
Spring (Mar-May)	6 110	25.08	22 516	23.10	
Summer(Jun-Aug)	6 646	27.28	24 372	25.01	
Autumn(Sep-Nov)	6201	25.45	27 386	28.10	
Winter (Dec-Feb)	5 406	22.19	23 178	23.78	
CSA					<0.001
Spring (Mar-May)	6 120	25.12	22,529	23.12	
Summer(Jun-Aug)	6 583	27.02	24,382	25.02	
Autumn(Sep-Nov)	6 211	25.49	27,367	28.08	
Winter (Dec-Feb)	5 449	22.37	23,174	23.78	

P: Chi-square/Fisher's exact test for categorical variables and *t* test for continuous variables.

3.2 Logistic regression of obesity variables

As shown in Fig. 2, a significantly higher risk of obesity was observed in the OSA group than in the control group (AOR = 2.927, 95% CI = 1.878-4.194), CSA group than in the control group (AOR = 2.234, 95% CI = 1.483-4.380).

3.3 Logistic regression to analyze obesity factors between different periods of sleep disorder exposure

As illustrated in Fig. 3, obese patients were more likely to have experienced OSA compared with nonobese patients (AOR = 2.927). Obese patients were more likely to have experienced CSA compared with nonobese patients (AOR = 2.234) Furthermore, the closeness of the exposure duration to the time of the study was positively associated with obesity severity in a dose–response manner (OSA exposure < 1 year, AOR = 3.895; OSA exposure ≥ 1 year and < 5 years, AOR = 2.933; OSA exposure ≥ 5 years, AOR = 2.486, CSA exposure < 1 year, AOR = 2.484; CSA exposure ≥ 1 year and < 5 years, AOR = 2.105; CSA exposure ≥ 5 years, AOR = 1.862).

Furthermore, Fig. 4 reveals that the mean exposure duration of OSA in patients with obesity was 2.927 times that in patients without obesity (AOR = 2.927). The mean exposure duration of CSA in patients with obesity was 2.234 times that in patients without obesity (AOR = 2.234). Third, a longer exposure duration was associated with more severe obesity, with a dose–response effect (OSA exposure < 1 year, AOR = 2.251; OSA exposure ≥ 1 year to < 5 years, AOR = 2.986; OSA exposure ≥ 5 years, AOR = 3.452. CSA exposure < 1 year, AOR = 2.101; CSA exposure ≥ 1 year to < 5 years, AOR = 2.207; CSA exposure ≥ 5 years, AOR = 2.976).

4. Discussion

This study showed that OSA or CSA is related with an increased risk of obesity. Obesity patients have higher risk to be exposed to OSA than CSA. This study may be the first study to compare OSA and CSA is related with an increased risk of obesity. The results of this study found that the risk of OSA with obesity was significantly greater than CSA with obesity (AOR = 2.927 versus 2.234), especially if obese patients were previously exposed to the two types of sleep disorders is 2.927 and 2.234 times that of non-obese patients, and the closer the exposure duration is to the current time, the more serious the obesity situation. The relationship between obesity and the two types of sleep disorders shows a dose-response; besides, the probability of obesity exposure duration of the two types of sleep disorders is 2.927 and 2.234 times that of non-obese patients, and the longer the exposure duration, the more serious the obesity situation, and the relationship between obesity and the exposure duration of the two types of sleep disorders also show a dose-effect.

Losing 10-15% of body weight in moderately obese patients can reduce the severity of OSA by 50%. Unfortunately, while weight loss can significantly improve OSA, it usually does not lead to a complete cure, and many people with sleep apnea require additional treatment. [17] Sleep disorders appear to be a risk factor for obesity, split sleep, overall sleep loss, and daytime sleepiness associated with OSA may also contribute to obesity, thereby further worsening OSA.[18] Sleep loss is not only due to habitual behavior, but also pathological conditions related to sleep disorders, such as OSA. The increase in the prevalence and severity of obesity has led to an increase in the prevalence of obesity-related comorbidities, including OSA.[19] According to this new paradigm, OSA will lead to a complex interaction of behavioral changes, leptin resistance and increased ghrelin levels, leading to reduced physical activity and/or increased unhealthy eating habits.[20] OSA adversely affects multiple organs and systems and is particularly associated with cardiovascular disease. Several diseases associated with OSA, such as hypertension, insulin resistance, systemic inflammation, visceral fat deposition, and dyslipidemia, are also present in other diseases closely associated with OSA, such as obesity and shortened sleep duration. Weight loss was accompanied by improvements in not only obesity-related traits but also OSA-related traits, suggesting that weight loss may be the cornerstone of treatment for both disorders. [20] Our research shows that OSA is associated with an increased risk of obesity.

The mechanism of CSA affecting overall health is unclear, but some studies have shown that sleep changes may affect the levels of various inflammatory markers, such as tumor necrosis factor (TNF), C-reactive protein (CRP)) and regulate the intermittent response of inflammation. [21-23] Severe obesity seems to be related to obvious sleep disturbances, even in individuals with CSA.[24-25] This kind of sleep disorder may also cause severely obese people to accumulate sleep debts and may lead to an appetite unregulated, restrict physical activity, and further impair weight maintenance.[26-27] Patients with CSA had a significantly increased risk of obesity. Long-term exposure to CSA and obesity is more likely and has a dose-response effect. [28] These pathophysiological factors may explain the association between CSA and obesity demonstrated in this study. Our research shows that CSA is associated with an increased risk of obesity.

Our results show that the OSA risk of obesity is significantly higher than that of obese CSA, and the closer the exposure duration is to the present time, the more serious the obesity situation; the probability of exposure duration of the two sleep disorders in obese patients is 2.927 and that of non-obese patients 2.234 times, and the longer the exposure time, the more serious the obesity situation. Therefore, the relationship between the occurrence and duration of OSA or CSA and obesity warrants consideration.

This study has several limitations. First, the NHIRD does not provide detailed information, such as that related to alcohol consumption, smoking, eating, and physical activity behaviors, which may affect our findings. Second, the Body Mass Index (BMI) was not a variable in our study. Third, although this study was carefully designed and controlled for confounding factors, biases may still exist because of unmeasured or unknown confounding factors (eg, the onset of anxiety, the stage of obesity at the time of

diagnosis, and drugs that may affect the outcome). A prospective cohort study is recommended to evaluate the relationship between OSA or CSA and obesity.

5. Conclusions

Obesity patients have higher risk to be exposed to OSA than CSA.Furthermore, the closeness to the time of the study and the exposure duration were both positively related to the severity of obesity, with a dose-response effect. OSA or CSA may be a risk factor for obesity. Health care providers should pay close attention to the association between OSA or CSA and the risk of obesity.

Declarations

Acknowledgments

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Data Availability

Data are available from the National Health Insurance Research Database (NHIRD) published by the Taiwan National Health Insurance (NHI) Administration. Due to legal restrictions imposed by the government of Taiwan concerning the “Personal Information Protection Act,” data cannot be made publicly available. Requests for data can be sent as a formal proposal to the NHIRD (http://www.mohw.gov.tw).

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of Tri-Service General Hospital at the National Defense Medical Center in Taipei, Taiwan (TSGH IRB No.B-109-39). All methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

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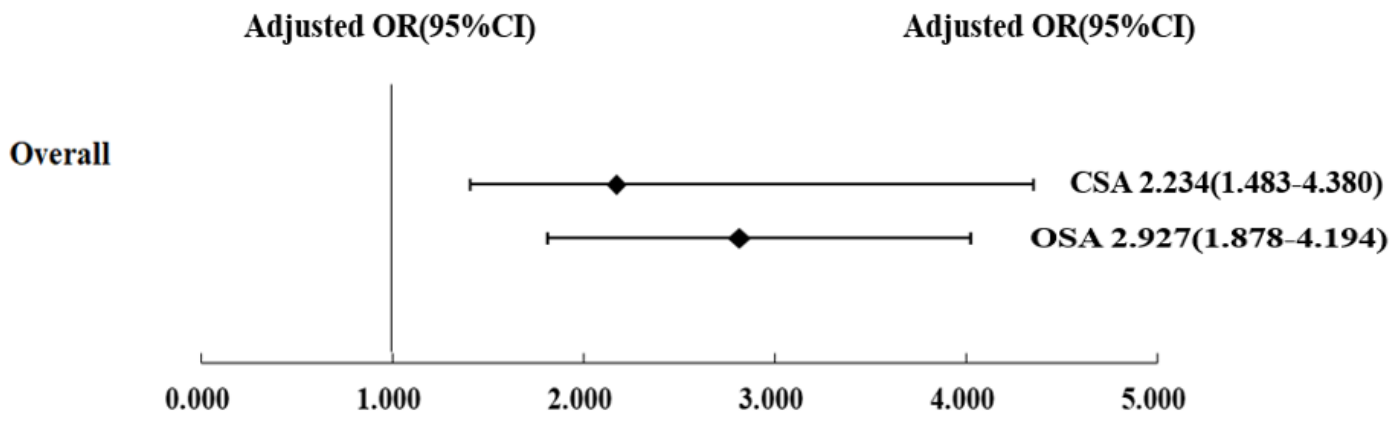
Figures

Figure 1

The flowchart of study design from the National Health Insurance Research Database in Taiwan.



OR = odds ratio; CI = confidence interval; Adjusted OR: Adjusted odds ratio

Figure 2

Logistic regression of obesity variables

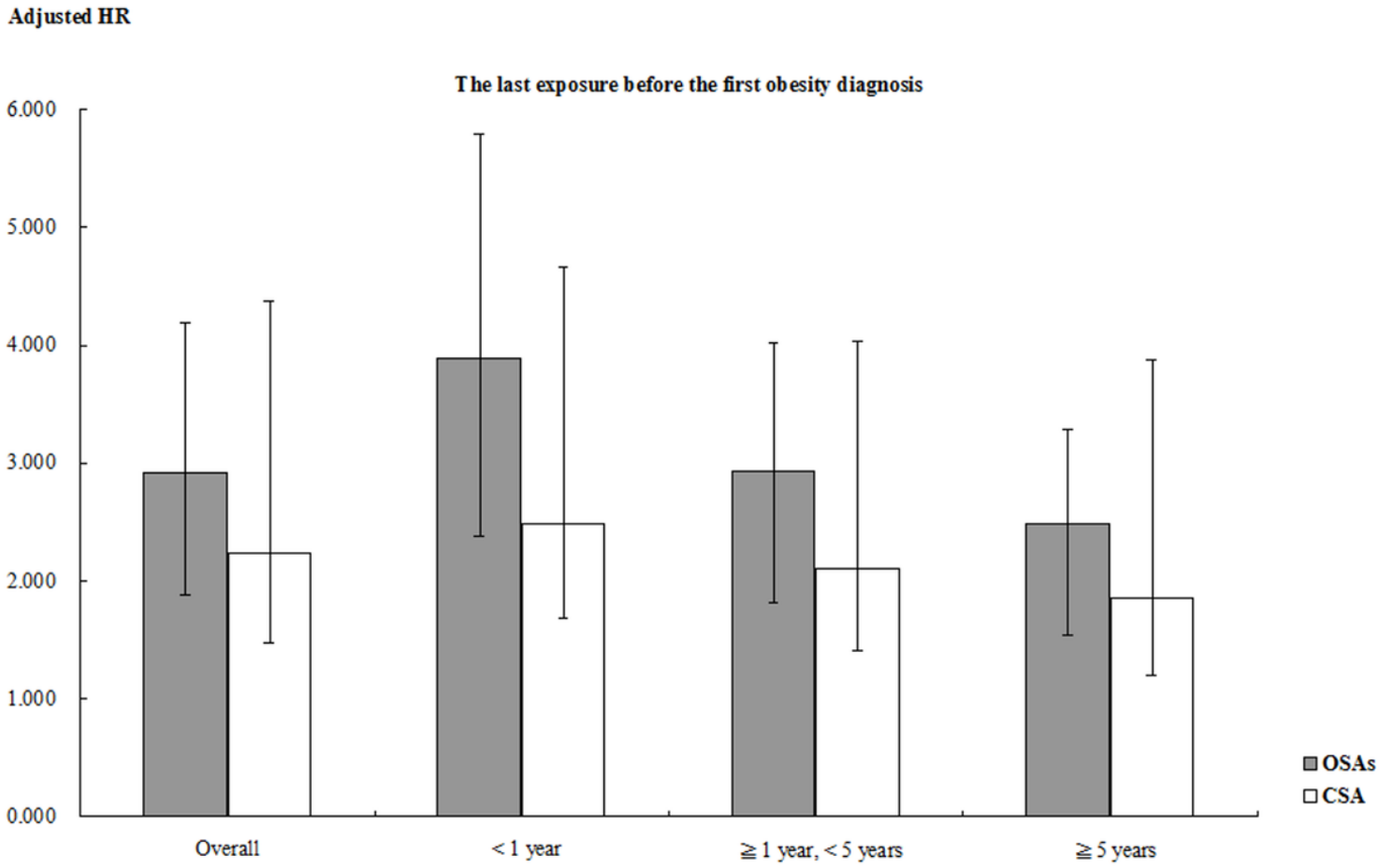


Figure 3

Factors of obesity among OSA and CSA exposure periods by using conditional logistic regression

Figure 4

Factors of obesity from different durations of OSA and CSA exposure by using conditional logistic regression