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Research Article

Risk factor panels associated with hypertension in obstructive sleep apnea patients with different body mass indexes

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Abstract

Although hypertension (HTN), obstructive sleep apnea (OSA), and obesity frequently co–occur, the precise role of obesity in this interrelationship is not completely understood. A total of 727 OSA patients were assigned to body mass index (BMI) <25 (27.6%; $n = 201$), 25≤ BMI <29.99 (53.4%; $n = 388$), and BMI ≥30 (19%; $n = 138$). HTN risk factors in each group were evaluated. A total of 244 (33.6%) patients exhibited co–morbid HTN, of whom 20.5% (50/244), 52.9% (129/244), and 26.6% (65/244) were distributed between the BMI <25, 25≤ BMI <29.99, and BMI ≥30 groups, respectively. Multiple logistic regression indicated that age, male gender, triglycerides (TG), low–density lipoprotein cholesterol (LDL–C), and apnea–hypopnea index (AHI) scores were HTN risk factors for the BMI<25 group. In the 25≤ BMI <29.99 group, risk factors were age, BMI, diabetes, and AHI. Finally, in the BMI ≥30 group, risk factors were age, diabetes, TG, LDL–C and AHI. These results demonstrate that different risk factor panels were associated with HTN in OSA patients with different BMIs. J Am Soc Hypertens 2015;9(5):382–389.

Keywords: Body mass indexes; hypertension; obstructive sleep apnea; risk factor panels.

Introduction

Hypertension (HTN) frequently co–occurs with obesity and obstructive sleep apnea (OSA).[1–3] The prevalence of HTN, obesity, and OSA, all of which contribute to the pathophysiologic process of cardiovascular disease (CVD), continues to increase.[4] It is estimated that between 5% and 10% of the general population, and 50%–60% of HTN patients, suffer from OSA.[3,4] In the Wisconsin Sleep Cohort Study, a dose–response association between OSA and HTN was observed following adjustment for baseline HTN status, body mass index (BMI), neck and waist circumference, age, gender, and weekly use of alcohol and cigarettes.[5] Furthermore, epidemiological data have consistently indicated a correlation between obesity and hypertension. In the Framingham Heart Study, 65%–78% of the risk for hypertension, in females and males, respectively, was related to obesity.[6] In certain populations, there is an almost linear relation between BMI and systolic/diastolic blood pressure.[4] Upper–body obesity, especially in the presence of increased visceral fat, is more strongly associated with hypertension compared with lower–body obesity.[1] Meanwhile, obesity is also a dominant risk factor for OSA, with significant sleep apnea present in up to 40% of obese individuals; furthermore, approximately 70% of OSA patients are obese.[7] In a population–based prospective study, weight gain of 10% was associated with a 6–fold increase in the odds of developing sleep apnea.[8]

Although evidence supports correlations between obesity, HTN, and OSA, conjecture regarding the precise nature of these correlations remains. In a recent, prospective longitudinal cohort study (1180 normotensive subjects recruited over a 7.5–year mean follow–up period), OSA was not associated with incident systolic HTN after adjusting for confounders (age, gender, BMI, neck circumference, fitness level, and alcohol, tobacco, and caffeine

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consumption).[9] OSA and obesity are characterized by several similar pathophysiological mechanisms and consequences.[1] OSA may contribute, at least in part, to some of the pathological processes traditionally ascribed to obesity alone, most notably sympathetic overactivity and humoral, metabolic, and neuroendocrine abnormalities.[1,3] Because the relationship between obesity and hypertension is complex, and probably represents an interaction between gender, racial, demographic, neurohormonal, genetic, and other factors, the key challenge in deciphering the OSA–HTN connection lies in accounting for confounding variables, particularly obesity.[1–4]

Current representative cross–sectional studies exploring the relationship between OSA and HTN are almost completely reliant on adjustments applied during multiple logistic regression modeling.[5–10] In these studies, in which BMI is treated as a covariate, the true role of obesity might be distorted. Obesity should be considered the principal confounding factor when evaluating the risk factors for HTN in OSA patients. We herein recruited 727 OSA patients exhibiting various degrees of apnea severity (apnea–hypopnea index [AHI] scores between 5 and 107), assigned to the following three groups: BMI <25; $25\leq$ BMI <29.99; and BMI ≥ 30. Different independent risk factors and risk factor panels for HTN, for each patient group, were revealed by multiple logistic regression analysis.

Methods

Patients

A total of 801 OSA patients with HTN, who visited our Department of Cardiology or Department of Respiratory, were recruited sequentially from July 2009 to December 2013. An OSA patient referred for diagnosis was screened first by a single physician, and then subjected to whole–of–night polysomnography (PSG).

Subjects who met the following exclusion criteria were excluded from the study: treatment with continuous positive airway pressure or other substitution therapies; presence of sleep disorders other than OSA; and chronic kidney disease or receiving hormone treatment. Ultimately, 727 adult patients were included.

Written informed consent regarding the procedures to be undertaken and use of medical data was obtained from all patients according to the guidelines of the Chinese National Ethics Regulation Committee. The Review Board of the Center Hospital of Minhang District approved this protocol in accordance with the amended Declaration of Helsinki (reference number: SHMHCH 2009–0009).

Data Acquisition

Anthropometric and demographics data: Age and gender data were collected before PSG. Neck circumference was measured at the level of the cricothyroid membrane while the subject was standing; waist circumference was measured midway between the lower costal margin and iliac crest, and hip circumference was measured as the maximum girth at the greater trochanters.[11] The mean of two measurements was used to calculate the subject's BMI and waist/hip circumference (WHR). Patients with a BMI >30 kg/m^2, or WHR >0.90 for males and >0.86 for females, were defined as obese.[12]

Epworth Sleepiness Scale (ESS): The ESS is a self–administered questionnaire that provides a measure of subjective daytime sleepiness. The ESS comprises questions on subjective sleepiness in eight situations. We translated the original version of the ESS; our version was subsequently validated. Respondents use a four–point scale (0–3) to respond to eight different questions, with scores summed to derive an overall score between 0 and 24.[13]

Polysomnography

Patients arrived at our sleep laboratory in the early evening. Multiple channel PSG (Somnostar 4100; SensorMedics Corp, Yorba Linda, CA) was conducted by full–time technicians. Electroencephalogram, electromyogram, electrooculogram, electrocardiogram, heart rhythm, pressure transducer, oxygen saturation, and chest and upper abdominal wall movement parameters were recorded. AHI was calculated by dividing the number of sleep events by the number of hours of sleep. Subjects with an AHI value >5 were defined as OSA.

Serum Lipids

Serum lipid profiles were measured in the hospital laboratory, including total cholesterol (TC), triglyceride (TG), high–density lipoprotein cholesterol (HDL–C), low–density lipoprotein cholesterol (LDL–C), apolipoprotein A–I (apoA–I), B (apoB), and E (apoE), Lipoprotein(a) (Lp(a)), and serum glucose, using routine procedures and a H–7600 Autoanalyzer (Hitachi, Tokyo, Japan).

Diabetes

Fasting blood samples were also collected during the morning after PSG monitoring. Serum glucose concentrations were also measured using the H–7600 Autoanalyzer. Diabetes was indicated by a fasting plasma glucose of ≥ 7.0 mmol/L, previous diabetes diagnosis, or treatment with an anti–diabetic medication 1 week before measurement.

Hypertension

Waking blood pressure was measured during the morning after overnight PSG monitoring using a mercury sphygmomanometer. Following a 5–minute rest period, seated patients underwent three measurements at 5–minute intervals, with mean values subsequently calculated. All measurements were performed by experienced physicians