

Prevalence of Obstructive Sleep Apnea (OSA) in Patients with Coronary Artery Disease

Shinichi Kadota*

Department of Cardiovascular Medicine, Saiseikai Futsukaichi Hospital, Japan

Corresponding author: Shinichi Kadota, Department of Cardiovascular Medicine, Saiseikai Futsukaichi Hospital, Japan, Email: shinichikadota67@yahoo.com

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Introduction

Obstructive Sleep Apnea (OSA) is a chronic condition characterized by repetitive episodes of upper airway collapse, apneas, and arousal during sleep. Community-based studies have shown that OSA [apnea-hypopnea index is observed in 17–26% of adult men and in 9–28% of adult women. It has been demonstrated that OSA is observed in about one-third of patients with Coronary Artery Disease (CAD) and that OSA is associated with adverse outcomes in patients with CAD. Furthermore, a prospective study showed that OSA is a risk factor for developing CAD. However, no information is so far available concerning the association between Coronary Spastic Angina pectoris (CSA) and OSA. Accordingly, we sought to clarify this association. Vasodilators were withheld for at least 48 h before coronary angiography. The presence or absence of coronary spasm was evaluated by an intracoronary acetylcholine or intravenous ergonovine provocation test after control coronary angiography. Heart rate, arterial blood pressure, and the 12-lead electrocardiogram were continuously monitored. Under support of right ventricular pacing set at a rate of 40 beats/min using a bipolar electrode catheter, acetylcholine was injected in incremental doses of 20, 50, and 100 µg into the left coronary artery and then in incremental doses of 20 and 50 µg into the right coronary artery, with 5-min intervals to the maximum tolerated dose. Coronary angiography was performed immediately after occurrence of ischemic ST-T changes with chest pain or 1 min after each injection. After that, 200 µg of intracoronary nitroglycerin was injected, and coronary angiography was performed. Methylergonovine maleate was intravenously administered in a dose of 0.2 mg.

Prevalence of Moderate to Severe OSA

Coronary angiography was performed immediately after occurrence of ischemic ST-T changes with chest pain or 4 min after the injection. If coronary spasm was not induced, an additional dose of 0.2 mg was intravenously administered. Coronary angiography was performed as the above-mentioned protocol. Subsequently, 200 µg of intracoronary nitroglycerin was injected, and coronary angiography was performed. Coronary spasm was defined as a total or subtotal obstruction or severe diffuse constriction of an epicardial coronary artery

associated with transient myocardial ischemia as evidenced by ischemic ST-segment changes. Overnight polysomnography was performed using a computerized system E-series, Compumedics Limited, Abbotsford, Australia. This investigation consisted of monitoring of the electro-encephalogram, electro-oculogram, submental electromyogram, electrocardiogram, thoraco-abdominal excursions using a piezo belt, oronasal airflow by a nasal pressure transducer and an oronasal thermistor, and arterial oxygen saturation by a pulse oximetry. An obstructive apnea was defined as the absence of oronasal airflow for ≥ 10 s associated with continued or increased inspiratory effort. A central apnea was defined as the absence of oronasal airflow for ≥ 10 s associated with an absent inspiratory effort. AHI was calculated as the mean number of apneas and hypopneas per hour of sleep. As obstructive hypopneas cannot be definitely distinguished from central hypopneas, we did not calculate the central or obstructive hypopnea index. OSA was classified into the following three groups based on AHI: mild OSA; moderate OSA; severe OSA. There were no significant differences in age, gender, body mass index, prevalences of coronary risk factors, lipid profiles, and fasting blood sugars between patients with CSA and control subjects. Patients with CSA had a higher prevalence of calcium antagonist use and a lower prevalence of beta blocker use than control subjects. Patients with CSA had a greater AHI than control subjects. This is the first study that has investigated the association between CSA and OSA. In the present study, patients with CSA had a greater AHI than control subjects, and the prevalence of moderate-to-severe OSA was significantly higher in patients with CSA than in control subjects.

Cause Effect Association between CSA and OSA

Furthermore, a multivariate logistic regression analysis selected moderate-to-severe OSA as an independent variable associated with CSA. These results suggest an intimate association between CSA and OSA. Because the present study is cross-sectional, a cause-effect association between CSA and OSA cannot be naturally determined. However, several potential mechanisms linking CSA to OSA are assumed as follows. First, repetitive hypoxia/reoxygenation increases reactive oxygen species and reduces the availability of endothelial nitric oxide,

resulting in endothelial dysfunction, which plays an important role in the pathogenesis of coronary spasm. Second, repetitive hypoxia/reoxygenation yields peroxynitrite. Subsequently, peroxynitrite nitrates and inactivates prostaglandin I₂ synthase, leaving unmetabolized prostaglandin H₂, which can cause vasospasm. Third, repetitive hypoxia/reoxygenation activates nuclear factor kappa B-dependent inflammatory pathways, resulting in the production of various proinflammatory cytokines. Proinflammatory cytokines including tumor necrosis factor- α , which has been shown to be elevated in the peripheral blood of patients with OSA, enhance Ca²⁺ sensitization in the vascular smooth muscle through activation of the RhoA/Rho-kinase pathway and thereby cause hypercontraction of the

vascular smooth muscle. Fourth, parasympathetic nervous activation followed by sympathetic nervous activation during one episode of OSA can enhance vascular tone of the epicardial coronary arteries with endothelial dysfunction. Thus, OSA could potentially predispose to coronary spasm through several mechanisms. In conclusion, the prevalence of moderate-to-severe OSA was significantly higher in patients with CSA than in control subjects, and moderate-to-severe OSA was an independent factor associated with CSA, suggesting that OSA may be one predisposing factor for coronary spasm. Further studies are needed to clarify the exact association between CSA and OSA.