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Original article

Association between obstructive sleep apnea and premature supraventricular contractions

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ABSTRACT

Objective: The exact association between obstructive sleep apnea (OSA) and premature supraventricular contractions (PSVCs) has not been established.
Methods: We prospectively performed polysomnography together with 24-hour Holter electrocardiography in 431 patients who were clinically suspected of having OSA and examined the association between OSA severity and PSVCs during wakefulness and sleep. The patients were classified into 4 groups according to the apnea–hypopnea index (AHI) quartiles (Q1 = patients with AHI < 13.8, Q2 = those with 13.8 ≤ AHI < 28.8, Q3 = those with 28.8 ≤ AHI < 48.1, Q4 = those with AHI ≥ 48.1).
Results: The number of PSVCs/hour during sleep differed significantly among the 4 groups, but the number of PSVCs/hour during wakefulness did not. The prevalence of PSVC ≥ 5/hour during sleep was significantly higher in Q4 (21.0%) than the other 3 groups (Q1, 9.0%; Q2, 8.0%; Q3, 6.0%; all $p < 0.05$ for Q4), but the prevalence of PSVC ≥ 5/hour during wakefulness did not differ among the 4 groups. A multivariate logistic regression analysis showed that the highest AHI quartile was significantly associated with PSVC ≥ 5/hour during sleep (odds ratio 3.04, 95% confidence interval 1.44–6.42, $p = 0.004$).
Conclusions: Severe OSA can cause PSVCs during sleep, but its effect appears not to be strong. Further studies are needed to clarify the clinical significance of this small but significant increase in PSVCs during sleep in severe OSA patients.

Introduction

Much attention has been recently paid to the association between sleep-disordered breathing and cardiovascular diseases including hypertension, arrhythmias, coronary artery disease, aortic dissection, congestive heart failure, pulmonary hypertension, and strokes [1–3]. Obstructive sleep apnea (OSA), which is a chronic condition characterized by repetitive episodes of upper airway collapse, apnea, and arousal during sleep, is the most frequent sleep-disordered breathing. The association between OSA and premature ventricular contractions has been repeatedly investigated [4–13]. On the other hand, only several investigations have been undertaken about the association between OSA and premature supraventricular contractions (PSVCs) [8–10,12], and the results of these studies are conflicting. It has been demonstrated that an isolated PSVC can trigger atrial fibrillation and supraventricular tachycardia [14,15] and that frequent PSVCs are associated with increased risks for stroke or death as well as atrial fibrillation [16,17]. Therefore, it is clinically important to clarify whether OSA can contribute to the occurrence of PSVCs. In this study, we prospectively examined the association between OSA severity and PSVCs during wakefulness as well as sleep in patients who were clinically suspected of having OSA.

Methods

Subjects

A total of 431 patients (336 males and 95 females, median age 56 years) who were clinically suspected of having OSA (severe snoring, daytime sleepiness, and witnessed apnea) and who met the inclusion criteria were prospectively enrolled into this study. All patients underwent full polysomnography together with 24-hour Holter electrocardiography. The inclusion criteria were aged ≥ 20 years, absence of chronic atrial fibrillation/flutter, no use of antiarrhythmic agents, no signs or symptoms of congestive heart failure, no pacemaker implantation and no treatment of OSA with oral

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appliance, continuous positive airway pressure, bi-level positive airway pressure, or adaptive servo-ventilation. Patients with dominantly central sleep apnea ($n = 2$) and the occurrence of atrial fibrillation on the 24-hour Holter electrocardiogram ($n = 2$) were excluded. The study protocol was approved by the ethics committee of Oita University Hospital, and informed consent was obtained from each patient before the study.

Polysomnography

Polysomnography was performed using a computerized recording system (E-series, Compumedics, Abbotsford, Australia). This investigation consisted of monitoring of the electro-encephalogram, electrooculogram, submental and limb electromyograms, electrocardiogram, thoraco-abdominal excursions by a piezo belt, oronasal airflow by a nasal pressure transducer and an oronasal thermistor, and the arterial oxyhemoglobin saturation by transcutaneous pulse oximetry. An obstructive apnea event was defined as the absence of oronasal airflow for ≥10 seconds associated with continued or increased inspiratory effort. A central apnea was defined as the absence of oronasal airflow for ≥10 seconds associated with an absent inspiratory effort. A hypopnea was defined as a ≥50% reduction in oronasal airflow for ≥10 seconds, associated with a ≥3% fall in arterial oxyhemoglobin saturation or an arousal. The apnea–hypopnea index (AHI) was calculated as the mean number of apneas and hypopneas per hour of sleep.

24-Hour Holter electrocardiography

Together with overnight polysomnography, 24-hour Holter electrocardiography was performed using a digital recorder with 3 channels (RAC-3103, Nihon Koden, Tokyo, Japan). Polysomnography and 24-hour Holter electrocardiography were time-synchronized. The presence or absence of PSVC ≥ 5/hour during wakefulness as well as sleep was assessed. This PSVC cut-off value has been used in previous studies investigating the association between OSA and PSVCs [8,10,12]. The mean numbers of PSVCs/hour during wakefulness as well as sleep was also calculated. Based on the electro-encephalogram, the sleep period was defined as the period from the starting of sleep to the end of sleep.

Statistical analysis

Continuous data are expressed as the median (first-third quartiles). The Kruskal–Wallis test with the Scheffe's post hoc test was used for comparisons of continuous data among 4 groups. Comparisons of categorical variables were analyzed by the Fisher's exact test or chi-square test. The univariate and multivariate logistic regression analyses were performed to determine variables associated with PSVC ≥ 5/hour during sleep. A value of $p < 0.05$ was considered to be statistically significant. Statistical analyses were performed using IBM SPSS Statistics 20 (International Business Machines, New York, NY, USA).

Results

Of 431 patients, 385 (89.3%) had an AHI ≥ 5. The 431 patients were classified into 4 groups according to the AHI quartiles (Q1 = 107 patients with AHI < 13.8; Q2 = 108 with 13.8 ≤ AHI < 28.8; Q3 = 108 with 28.8 ≤ AHI < 48.1; Q4 = 108 with AHI ≥ 48.1). The patient characteristics are shown in Table 1. Age, body mass index, and the prevalences of male gender, hypertension, diabetes mellitus, calcium antagonist use, and angiotensin-converting enzyme inhibitor or angiotensin receptor blocker use differed significantly among the 4 groups.

The number of PSVCs/hour during sleep differed significantly among the 4 groups (Q1, 0.4 [0.0–1.2]; Q2, 0.6 [0.2–1.5]; Q3, 0.5 [0.1–1.2]; Q4, 0.9 [0.2–3.1]; $p = 0.01$), but the number of PSVCs/hour during wakefulness did not (Q1, 0.6 [0.1–1.3]; Q2, 0.6 [0.2–1.6]; Q3, 0.5 [0.2–1.3]; Q4, 0.6 [0.2–2.4]; $p = 0.75$). The prevalence of PSVC ≥ 5/hour during sleep was significantly higher in Q4 (21.0%) than in the other 3 groups (Q1, 9.0%; Q2, 8.0%; Q3, 6.0%; all $p < 0.05$ for Q4), but the prevalence of PSVC ≥ 5/hour during wakefulness did not differ significantly among the 4 groups ($p = 0.20$) (Fig. 1).

The number of PSVCs/hour during sleep and that during wakefulness differed significantly among the 4 groups classified by the arousal index (ArI) quartiles (A-Q1 = 107 patients with ArI < 21.2: 0.4 [0.1–0.9]; A-Q2 = 107 with 21.2 ≤ ArI < 31.9: 0.5 [0.1–1.1]; A-Q3 = 109 with 31.9 ≤ ArI < 48.5: 0.6 [0.2–1.6]; A-Q4 = 108 with ArI ≥ 48.5: 1.0 [0.2–2.6]; $p = 0.001$, and A-Q1: 0.4 [0.1–1.2]; A-Q2: 0.6 [0.2–1.3]; A-Q3: 0.7 [0.3–2.0]; A-Q4: 0.7 [0.2–2.1]; $p = 0.048$, respectively). The prevalence of PSVC ≥ 5/hour during sleep differed significantly among the 4 groups (A-Q1, 6.5%; A-Q2, 4.7%; A-Q3, 11.9%; A-Q4, 17.6%; $p < 0.01$), and the prevalence of PSVC ≥ 5/hour during wakefulness tended to differ among the 4 groups (A-Q1, 7.5%; A-Q2, 3.7%; A-Q3, 11.0%; A-Q4, 13.9%; $p = 0.05$). The number of PSVCs/hour during sleep and that during wakefulness did not differ significantly among the 4 groups classified by the lowest SpO_2 (L-SpO_2) quartiles (L-Q1 = 100 with L-SpO_2 < 77%: 0.5 [0.1–2.1]; L-Q2 = 105 with 77% ≤ L-SpO2 < 83%: 0.5 [0.3–1.5]; L-Q3 = 110 with 83% ≤ L-SpO_2 < 88%: 0.6 [0.2–1.6]; L-Q4 = 116 with L-SpO_2 ≥ 88%: 0.4 [0.1–1.5]; $p = 0.11$, and L-Q1: 0.4 [0.1–1.8]; L-Q2: 0.6 [0.2–1.6]; L-Q3: 0.7 [0.3–1.7]; L-Q4: 0.6 [0.1–1.3]; $p = 0.30$, respectively). The prevalence of PSVC ≥ 5/hour during sleep tended to differ among the 4 groups (L-Q1, 16.0%; L-Q2, 11.4%; L-Q3, 5.5%; L-Q4, 8.6%, $p = 0.08$), but the prevalence of PSVC ≥ 5/hour during

Table 1
Patient characteristics and polysomnographic data.

Variables	Q1 ($n = 107$)	Q2 ($n = 108$)	Q3 ($n = 108$)	Q4 ($n = 108$)	p-Value
Age (years)	56.0 (41.0–64.0)	62.0 (51.0–70.0)	62.0 (50.3–70.0)	54.0 (44.0–66.8)	<0.001
Male gender	64 (59.8%)	84 (77.8%)	92 (85.2%)	96 (88.9%)	<0.001
Body mass index (kg/m^2)	23.3 (21.5–25.8)	24.4 (22.7–26.7)	25.5 (23.6–28.7)	28.4 (25.3–32.7)	<0.001
Hypertension	53 (49.5%)	73 (67.6%)	76 (70.4%)	74 (68.5%)	0.004
Dyslipidemia	64 (59.8%)	70 (64.8%)	78 (72.2%)	79 (73.1%)	0.11
Diabetes	14 (13.1%)	25 (23.1%)	33 (30.6%)	33 (30.6%)	0.01
Current smoker	22 (20.6%)	24 (22.2%)	17 (15.7%)	23 (21.3%)	0.65
Coronary artery disease	19 (17.8%)	22 (20.4%)	16 (14.8%)	8 (7.4%)	0.05
AHI	6.0 (2.8–9.3)	20.9 (17.5–23.9)	36.5 (31.6–42.1)	65.3 (58.4–81.9)	<0.001
Calcium antagonists	30 (28.0%)	49 (45.4%)	49 (45.4%)	52 (48.1%)	0.01
ACEIs/ARBs	27 (25.2%)	43 (38.9%)	50 (46.3%)	43 (39.8%)	0.01
Beta-blockers	14 (13.1%)	14 (13.0%)	14 (13.0%)	16 (14.8%)	0.97

Values are presented as the median (first–third quartiles).
Q1 = patients with AHI < 13.8; Q2 = those with 13.8 ≤ AHI < 28.8; Q3: those with 28.8 ≤ AHI < 48.1; Q4: those with AHI ≥ 48.1.
AHI, apnea–hypopnea index; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.