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Prevalence of risk factors for atrial fibrillation and stroke among 1210 patients with sleep disordered breathing☆,☆☆

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ABSTRACT

Aims: This study sought to identify the prevalence of risk factors for atrial fibrillation and stroke in a sleep apnea population.
Methods: Study participants included 1210 consecutive adults who were referred with suspicion of sleep apnea. Statistical analysis was used to determine the relationship between sleep apnea syndrome and risk factors for atrial fibrillation and stroke.
Results: Among 1210 enrolled patients, 65.8% had severe sleep apnea (Apnea/hypopnea Index — AHI > 30), 25.2% had mild to moderate sleep apnea (AHI 5 to 30), and 8.8% had no sleep apnea (AHI < 5). At baseline, the mean apnea–hypopnea index in patients with sleep apnea syndrome was 35. Compared to patients with an AHI <5, those with an AHI >30 were older (47.3 ± 11.4 vs. 52.74 ± 12.4, $p < 0.001$) and had a higher body mass index (BMI) (30.7 ± 7.3 vs. 33.83 ± 10.1, $p < 0.001$), a higher prevalence of hypertension (38 vs. 16%, $p < 0.001$), and a higher $CHADS_2$ (congestive heart failure, hypertension, age, diabetes and prior stroke) score (0.59 ± 0.8 vs. 0.28 ± 0.64, $p < 0.001$).
Conclusions: Patients with severe sleep apnea have a higher prevalence of risk factors for atrial fibrillation and stroke when compared with subjects without sleep apnea.

1. Introduction

After insomnia, sleep apnea (SA) is the most common sleep disorder, affecting approximately 5% of the adult population [1]. Sleep apnea has been independently linked to adverse cardiovascular outcomes such as hypertension [2–4], stroke [2–4], myocardial ischemia [5–8], arrhythmias [9,10], and both cardiovascular [2,3] and all-cause mortality [3,11,12]. Patients with sleep apnea are frequently diagnosed with coexisting autonomic imbalance [13] and diastolic heart dysfunction [14]. It is considered that these two mechanisms may potentially promote the development of atrial fibrillation (AF), which is the most common arrhythmia encountered in clinical practice and an important cause of stroke [15,16].

Earlier studies have assessed the prevalence of sleep-disordered breathing among patients with cardiovascular disease in individuals undergoing cardiac assessment [17] or cardiac interventions [18], for example. The main objective of this study was to assess the prevalence of cardiovascular risk factors for AF and stroke in an unbiased sample of patients referred for the assessment of sleep apnea and to determine whether there is a correlation between the severity of sleep apnea and the prevalence of AF and stroke.

2. Methodology

We conducted a cross-sectional study that included consecutive patients referred to the Sleep Laboratory of San Cecilio Hospital specifically for the evaluation of sleep-disordered breathing. All patients underwent a baseline clinical evaluation and diagnostic polysomnography. The presence of clinical risk factors for AF, including: age, gender, hypertension, body mass index (BMI), congestive heart failure, prior stroke and $CHADS_2$ score; were compared between three patient groups: those

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without sleep apnea (AHI < 5), those with mild to moderate sleep apnea (AHI > 5 and <30) and those with severe sleep apnea (AHI > 30). The study was approved by the Human Investigation Committee of San Cecilio University Hospital and all subjects gave their written informed consent prior to their inclusion in the study.

3. Baseline assessment

Data on demographic characteristics, sleep, medical history, medication use, and habits were obtained with the use of a standardized questionnaire administered by a trained researcher. Patients with documented history of AF were excluded from the study. The interview was conducted in the presence of participants' most closely related person, preferably their spouse. After completion of this assessment, subjects underwent overnight polysomnography. Patients' height and weight were recorded at the time of the polysomnography and used to calculate their body mass index (BMI). Sleep-history data included the Berlin questionnaire [19], the Epworth Sleepiness Scale [20], and self-reported habitual snoring, which was defined as loud snoring occurring "frequently" or "constantly." Data regarding medication use included the daily use of beta-blockers, diuretics, other antihypertensive medications, antiplatelet therapy, and anticoagulants. Data on risk factors included a history of hypertension, diabetes mellitus and chronic obstructive pulmonary disease (COPD), either reported by the patient on the baseline medical questionnaire or noted by the referring physician.

In addition, the pulmonary function test, electrocardiography, and plethysmography performed during polysomnography were considered sufficient evidence to establish the diagnosis of sleep apnea. Patients were classified according to whether they were current or former smokers or had never smoked. If applicable, data were based on the number of pack-years of smoking.

4. Polysomnography

Participants attended overnight polysomnography using Grass data-acquisition systems (Astro-Med, West Warwick, RI) on the basis of a protocol described previously [21]. A single polysomnography study conducted during an entire night was used to establish the presence of sleep apnea [22]. Sleep stages were scored over 30-second intervals according to standard criteria [23]. Total cessation of airflow in the nose and mouth for at least 10 s was classified as apnea (obstructive apnea if respiratory efforts were present and central apnea if respiratory efforts were absent). Partial airway closure resulting in a reduction of more than 30% of airflow for at least 10 s and associated with 4% or more oxygen desaturation was considered hypopnea [24]. The polysomnographic variables assessed included the apnea–hypopnea index (AHI), that is, the number of apneas–hypopneas per hour of sleep.

5. Statistical analysis

We used SPSS statistical software, version 16.0 for descriptive statistics and data analysis. Subjects were distributed into 3 groups according to their polysomnography results: subjects with severe SA (AHI > 30), subjects with mild to moderate SA (AHI: 5–30), and subjects with no SA (AHI < 5). All continuous data were examined for normality using the Shapiro–Wilk W statistic. An ANOVA was used to compare numerical variables among the three groups. The χ^2 test was used to compare categorical variables among three groups. A p value <0.05 was considered statistically significant.

6. Results

The study population consisted of 1210 consecutive subjects referred for polysomnography. Severe SA (AHI > 30) was present in 65.8% of participants, mild to moderate SA (AHI: 5–30) was found in 25.2% of them, and no SA (AHI < 5) was found in 8.8% of the sample. The age, BMI, gender, and medical history characteristics of subjects according to their AHI results are reported in Table 1.

Patients with severe SA were older, more likely to be male, and had a higher BMI (Table 1). Patients with severe SA were also more likely to have a history of hypertension and a higher CHADS2 score (Table 1). Smoking history and daily alcohol consumption were frequent in this population, but no significant differences were found among patients with severe SA, mild to moderate SA, and no SA. No significant differences were found either in medication use between the SA groups.

Compared to patients without SA, those with severe SA had a higher systolic blood pressure and more abnormal Berlin questionnaire results. However, no differences were found in any pulmonary function test parameters or the Epworth Sleepiness Scale (Table 2).

7. Discussion

This large, single-center study demonstrates that risk factors for AF and stroke are highly prevalent among patients referred for the assessment of SA, particularly those with evidence of severe SA at the time of polysomnography. Nearly 50% of patients with severe SA had a CHADS2

Table 1
Recognized risk factors for the development of Stroke and AF, distribution of medication intake distribution in the sample by AHI score. Statistical significance were shown if p < 0.001 between: group AHI < 5 and group 5-AHI-30: a; group AHI < 5 and group AHI > 30: b; and group 5-AHI-30 and group AHI > 30: c.

Variables	AHI < 5 (n = 107)	AHI 5–30 (n = 306)	AHI > 30 (n = 797)	p-Value
Age (years) Mean ± SD	47.3 ± 11.4	49.3 ± 12.3	52.74 ± 12.4	0.001[b]
Sex (females) n (%)	97 (59.5)	77 (33.3)	170 (21)	<0.001[b,c]
BMI (Kg/m^2) Mean ± SD	30.7 ± 7.3	30.8 ± 4.7	33.83 ± 10.1	<0.001[b,c]
Smoking history:				
Never	77 (43.9)	113 (48.9)	262 (32.5)	
Current	51 (36.4)	61 (26.9)	289 (35.7)	<0.001
Ex-smoker	35 (19.6)	56 (24.3)	258 (32.0)	
Alcohol (daily consumers) n (%)	46 (43.0)	154 (50.3)	430 (54.0)	0.081
Percent SaO_2 < 90% Mean ± SD	3.7 ± 11.3	7.7 ± 12.8	23.2 ± 25.1	<0.001[b,c]
HTN n (%)	19 (17.8)	83 (27.1)	308 (38.6)	<0.001
COPD n (%)	3 (2.8)	11 (3.6)	38 (4.8)	0.502
Diabetes n (%)	4 (3.7)	24 (7.8)	78 (9.8)	0.093
Prior stroke or TIA n (%)	3 (2.8)	7 (2.3)	18 (2.3)	0.939
Heart failure n (%)	1 (0.9)	6 (2)	18 (3.4)	0.206
$CHADS_2$ score Mean ± SD	0.28 ± 0.64	0.43 ± 0.8	0.59 ± 0.8	<0.001[b]
$CHADS_2$ score 0 n (%)	82 (79.6)	207 (70.2)	423 (56.3)	
$CHADS_2$ score 1 n (%)	16 (15.5)	64 (21.7)	248 (33)	<0.001
$CHADS_2$ Score ≥ 2 n (%)	5 (4.9)	24 (8.1)	81 (10.8)	
Medications				
Digoxin, %	1.7	1.9	2.5	0.918
ACE inhibitors, %	16.9	12.3	10.9	0.517
Diuretics, %	10.2	5.7	14.3	0.103
B-blockers, %	10.2	6.6	10.9	0.509
Amiodarone, %	0.5	5.7	3.4	0.166
ASA, %	8.5	5.7	5	0.651
Anticoagulant, %	13.6	12.3	11.8	0.943
Benzodiazepines, %	1.7	6.6	9.2	0.165

AHI: Apnea/hypopnea Index; SD: Standard deviation; BMI: Body mass index; SaO_2: Oxygen saturation; HTN: Hypertension; COPD: Chronic obstructive pulmonary disease; TIA: Transient ischemic attack; ACE: Angiotensin converting enzyme; ASA: Acetylsalicylic acid.