Prevalence of Obstructive Sleep Apnea Syndrome in Patients With Sick Sinus Syndrome

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Introduction and objectives. Sleep apnea-hypopnea syndrome (SAHS) has been associated with different cardiovascular diseases. It may even be implicated in the pathophysiology of sick sinus syndrome (SSS). However, the precise relationship between the two syndromes is still unknown. We investigated the prevalence of SAHS in patients diagnosed with SSS.

Patients and methods. Between June 2002 and December 2004, 38 consecutive patients who were diagnosed with SSS by 24-hour Holter monitoring were studied prospectively in our institution. All patients were asked about symptoms of SAHS, and underwent polysomnography out of hospital using a validated monitor.

Results. The patients' mean age was 67 (10) years, 68% were male, and 58% were hypertensive. Holter monitoring demonstrated a maximum heart rate of 87 (6) beats/min, a minimum of 35 (3) beats/min, and a mean of 48 (3) beats/min. Some 24 (63%) patients required pacemaker implantation because of symptomatic SSS. Overall, 39% of patients had symptoms suggestive of SAHS (i.e., an Epworth index or EI>9). Polysomnography showed that only a 13% of patients had a normal apnea-hypopnea index (AHI) and that 31.6% (95% CI, 16.8-46.4%) had SAHS (i.e., AIH>10 and EI>9).

Conclusions. Given that the prevalence of SAHS in the general population is around 3%, our results indicate that it is ten-fold higher in patients with SSS than in the general population, which indicates a relationship between the 2 syndromes.

Key words: Sick sinus syndrome. Obstructive sleep apnea syndrome. Cardiovascular disease.

INTRODUCTION

Sleep apnea-hypopnea syndrome (SAHS) is characterized by recurrent episodes of partial or total collapse of the upper airway during sleep, leading to the hindrance (hypopnea) or complete obstruction (apnea) of airflow despite efforts to breath. The lack of adequate alveolar ventilation leads to a reduction in
oxygen saturation and, if the episode is prolonged, to an increase in the arterial partial pressure of CO₂ (PaCO₂). These events result in arousals from sleep, which give rise to the daytime symptoms of the disorder: hypersomnia, the sensation that sleep was not refreshing, fatigue, and difficulty in concentrating.

The American Academy of Sleep Medicine (AASM) recently stated that the diagnosis of SAHS requires the detection of obstructive events through the use of a polysomnograph plus the daytime symptoms mentioned above. By this criterion, the prevalence of SAHS in the general adult population is around 3%. It is more common in men and increases with age.

For years it has been suggested that a relationship may exist between SAHS and a number of cardiovascular diseases, including high blood pressure, ischemic heart disease, heart failure, cerebrovascular accidents, and pulmonary hypertension. Evidence exists to support a relationship between SAHS and certain supraventricular arrhythmias, especially atrial fibrillation. There is also indirect evidence that it may be related to sick sinus syndrome (SSS). For example, atrial stimulation reduces the number of apnea episodes in patients with SAHS, and these patients commonly suffer sinus arrest, sinoatrial block and atrial fibrillation (all characteristic of SSS). For each patient, the total number of apnea/hypopnea episodes was divided by the duration (hours) of the study to determine the apnea-hypopnea index (AHI), the percentage time that oxygen saturation was <90%, and the mean duration of the apnea/hypopnea episodes. Cut-off points of 10, 20, and 30 were used for the AHI. For patients with SAHS the number of apnea episodes per hour of sleep (AHI) was calculated. An AHI of >10 and an ESS score of 9 were considered to indicate SAHS.

**Statistical Analysis**

A descriptive analysis of the data was performed. Categorical variables were represented as absolute frequencies and percentages. Quantitative variables were represented as means ± the standard deviation (SD). The 95% confidence interval (CI) was calculated for the most significant variables. The Pearson correlation test was used to analyze the linear association between quantitative variables. To determine whether the proportion of patients with SAHS was different to that described for the general population the z coefficient of the z value (z=P–p/√PQ/n) was calculated. Significance was set at P≤.05.

**RESULTS**

Table 1 shows the main characteristics of the study population. As expected, the mean age of the patients was 59 years old, with a range of 50–70 years. The mean body mass index (BMI) was 25.4 kg/m², with a range of 21–32 kg/m². The mean blood pressure was 130/80 mmHg, with a range of 110–150/60–90 mmHg. The mean ejection fraction (EF) was 55%, with a range of 40–70%. The mean Epworth sleepiness scale (ESS) score was 10, with a range of 0–30 points.

**Abbreviations**

AHI: apnea-hypopnea index.

ESS: Epworth sleepiness scale.

SAHS: sleep apnea-hypopnea syndrome.
population was high, as was mean blood pressure, and the majority of patients were men. Table 2 shows the Holter monitoring results; 45% of subjects showed a slow heart rate (fast heart rates caused by atrial arrhythmias were excluded) and suffered atrial arrhythmias (generally atrial fibrillation, which supports the diagnosis of tachycardia-bradycardia syndrome). Table 2 also shows the echocardiography results, which indicate that left ventricular dysfunction was not a problem of the present cohort. The mean ejection fraction (EF) was 64±13%. No patient had an EF of <50%. Twenty four patients (63%) received a definitive pacemaker (mostly in DDD-R mode) due to clinical symptoms associated with SSS. Twenty patients required this procedure soon after having been diagnosed (following the polysomnograph test). The remaining four received their implants due to the appearance of symptoms at some later point.

**TABLE 1. Population Characteristics (n=38)**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years, mean±SD</td>
<td>66.6±10</td>
</tr>
<tr>
<td>Proportion of men, n (%)</td>
<td>26 (68.4)</td>
</tr>
<tr>
<td>BMI, mean±SD</td>
<td>28±4</td>
</tr>
<tr>
<td>Overweight patients (BMI=25-29), n (%)</td>
<td>14 (36.8)</td>
</tr>
<tr>
<td>Obese patients (BMI&gt;30), n (%)</td>
<td>13 (34.2)</td>
</tr>
<tr>
<td>Hypertension (SBP≥140 or DBP≥90 mm Hg), n (%)</td>
<td>22 (57.9)</td>
</tr>
<tr>
<td>Epworth sleepiness scale, mean±SD</td>
<td>8.0±3.8</td>
</tr>
<tr>
<td>Chronic excessive sleepiness (ESS≥9), n (%)</td>
<td>15 (39.5)</td>
</tr>
</tbody>
</table>

SD indicates standard deviation; ESS, Epworth sleepiness scale; BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure.

**TABLE 2. Cardiological Data (n=38)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
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<tbody>
<tr>
<td>FC baseline, beats/min, mean±SD</td>
<td>47.9 (3.3)</td>
</tr>
<tr>
<td>Holter data, mean±SD</td>
<td>86.6±6.3</td>
</tr>
<tr>
<td>Maximum HR, beats/min, mean±SD</td>
<td>34.7±3.2</td>
</tr>
<tr>
<td>Minimum HR, beats/min, mean±SD</td>
<td>17 (44.7)</td>
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<tr>
<td>Mean HR, beats/min, ±SD</td>
<td></td>
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<tr>
<td>Atrial arrhythmias, n (%)</td>
<td></td>
</tr>
<tr>
<td>Echocardiography</td>
<td></td>
</tr>
<tr>
<td>EF, n (%)</td>
<td>64.4 (12.8)</td>
</tr>
<tr>
<td>Patients with EF&lt;50%, n</td>
<td></td>
</tr>
<tr>
<td>Patients who received a pacemaker, n (%)</td>
<td>24 (63.2)</td>
</tr>
</tbody>
</table>

* SD indicates standard deviation; HR, heart rate; EF, ejection fraction.

**DISCUSSION**

The main finding of this study is the high prevalence of sleep-related respiratory problems and SAHS in this population of patients with SSS. Recently, the AASM proposed a consensus definition of SAHS based on an AHI of >5 plus daytime symptoms, the most important being excessive sleepiness. In agreement with this definition, the prevalence of SAHS in the adult population lies between 3.3% and 3.4%, more common in men (4%) than in women (2%), and increases with age. For this reason, plus the fact that SSS is more prevalent in the elderly (the mean age of the present population was 67 years), it was decided to raise the cut-off for AHI to >10 for the present study. Even with this high cut-off the prevalence of SAHS (AHI>10) and an ESS>9) was 31.6%—almost ten times that seen in the general population. This is not surprising if the results of Gami et al are taken into account. These authors reported that 50% of patients with atrial fibrillation also suffer SAHS. Unfortunately, their study does not report the percentage of patients who also suffered SSS, although it is well known that atrial fibrillation is the most common type of arrhythmia in tachycardia-bradycardia syndrome. In the present study tachycardia-bradycardia syndrome was detected in 45% of the Holter readings; atrial fibrillation was the most common type of tachycardia.

Simantirakis et al also indicate a relationship to exist between SSS and SAHS. These authors reported moderate-severe abnormalities in the heart rate of 47% of the patients they studied (n=23). In addition, they found that after starting treatment for SAHS with CPAP these problems were significantly reduced. Unfortunately, the present study does not clarify whether SSS facilitates the appearance of obstructive
apneas or whether SAHS, which generates atrial arrhythmia, ends up causing SSS. The data of Garrigue et al.22,23 on a new treatment for SAHS requiring permanent stimulation of the heart tend to support the first of these possibilities. In their first paper,22 which involved a study of 15 patients with SAHS, these authors observed episodes of severe sinus bradycardia and even sinus arrests during the polysomnograph test. Later, following atrial stimulation, the patients’ obstructive and central apnea problems improved. A hypothesis involving a double mechanism was put forward to explain the improvement in central apnea. In patients with this type of apnea, hypervagotony could be the cause of bradyarrhythmias—therefore permanent atrial stimulation could be of help. In addition, the increase in heart rate caused by electrical stimulation would improve cardiac output, and therefore have a positive effect on the associated pulmonary subedema. However, these authors recognize they have no convincing evidence that hypervagotony can explain the improvement in central apnea. In patients with severe SAHS, and all of whom received an insertable Holter for 16 months. The aim was to detect arrhythmias in the first 2 months and, over the next 14, to observe the effect of CPAP treatment on them. Some 47% of these patients showed severe arrhythmia problems over these first 2 months, all of which improved with CPAP until eventually disappearing in the final 6 months of the study.

One way or the other, the present study shows a close relationship between SSS and SAHS (which have common risk factors), and the clinical implications are obvious. Further work should determine whether the detection and early treatment of SAHS can prevent the development of SSS, and therefore reduce the incidence of atrial tachycardias and the number of pacemakers that need to be implanted (thus reducing health spending).

Finally, pulmonologists who detect patients with SAHS should think of SSS as a coadjuvant illness, and cardiologists who diagnose SSS should determine whether the patient suffers symptoms indicative of SAHS.

**Limitations of the Study**

The study had no control group, so a definitive relationship between SSS and SAHS cannot be affirmed; confounding factors may exist that were not taken into account. To confirm the hypothesis that these diseases are linked, further studies with control groups are required. Since this was a preliminary study, follow-up data were not available on changes in the nocturnal respiratory problems of patients who received a pacemaker.

**REFERENCES**


