Sleep-disordered breathing and heavy drinking: clinical features and polysomnographic findings

ABSTRACT
Objectives: The main objective of this study was to describe clinical features, particularly depressive symptoms, comorbidities, and polysomnographic characteristics of heavy drinkers with sleep disordered breathing (SDB).

Methods: This was a cross-sectional study of 140 cases (92 males; mean age 54.6±8.2 years) referred for overnight polysomnography with suspicion of obstructive sleep apnea (OSA). Patients were questioned about clinical and demographic data and evaluated for depressive symptoms by the 17-item Hamilton Rating Scale for Depression (HRSD), excessive daytime sleepiness by the Epworth Sleepiness Scale (ESS) and comorbidity severity by the Charlson Comorbidity Index (CCI).

Results: Fourteen patients were primary snorers [Apnea-Hypopnea Index (AHI)<5; 10%] and 126 had OSA (AHI>5): 86 (61.4%) moderate/severe type (AHI>15). Heavy drinking was more common in male (p<0.005) and younger (p=0.01) individuals. Among heavy drinkers, there was a correlation between depressive symptoms and body mass index (BMI) (r=0.33; p=0.01) and this was more evident in women (r=0.68; p<0.005) than in men (r=0.53; p=0.02). Excessive daytime sleepiness (ESS≥10) was present in 57 cases (40.7%). ESS scores were positively correlated with arousals (r=0.24; p=0.02) and negatively with SpO2 (r=-0.18; p=0.03).

Heavy drinkers with AHI<15 presented higher ESS scores (p=0.03) and a trend of association remained after controlling for age and gender (p=0.08).

Conclusions: In patients referred for polysomnography, heavy drinking is more common in younger male individuals. Depressive symptoms are related to BMI in heavy drinkers. More studies are warranted to clarify the influence of chronic heavy drinking on sleep abnormalities.

Keywords: Sleep apnea syndromes; Alcohol drinking; Alcohol beverages; Polysomnography; Comorbidity; Depression; Body mass index; Disorders of excessive somnolence

INTRODUCTION
Obstructive sleep apnea (OSA) is an important clinical condition affecting around 4% of men and 2% of women(1), with more recent surveys showing even higher prevalence rates(2). Sleep alterations, such as decreased sleep efficiency, increased number of arousals, nocturnal oxyhemoglobin desaturation, periodic leg movements, increased REM latency, and decreased amounts of slow wave sleep (stages 3 and 4 NREM sleep) and REM sleep have been reported in association with OSA(3-5). Clinical and polysomnographic abnor-
Sleep disordered breathing and heavy drinking

Malignancies in OSA are known to be influenced by gender and mood disorders. For instance, men have more severe apnea-hypopnea index (AHI) and women present with more depressive symptoms.

Previously, the clinical characteristics of long-term excessive use of alcohol in connection with OSA have not been sufficiently investigated. Alcohol consumption has been associated with both, a reduction of sleep latency and disruption of sleep and the latter can lead to daytime fatigue and sleepiness. As regards to the relation of heavy drinking and sleep disordered breathing, previous studies have been contradictory. Alcohol has been shown to aggravate OSA while heavy drinking has been found not to be predictive of sleep disordered breathing. Moreover, moderate alcohol ingestion is said not to influence continuous positive airway pressure (CPAP) therapy results. Recently, serum gamma-glutamyl transferase (GGT), a marker of alcohol consumption, has been associated with increased nocturnal arterial oxygen desaturations suggesting a deleterious effect of alcohol in OSA. Other effects of alcohol consumption on various clinical conditions may also be considered contradictory. For instance, mild to moderate alcohol consumption has been claimed to have beneficial effects in coronary heart disease, hypertension and cerebrovascular disease; on the other hand, heavy alcohol ingestion has been connected with increased depression and worsening of sleep parameters.

The relevance of the relationship between heavy drinking and OSA is reinforced by the fact that stroke has been recognized in association with OSA. Other conditions, scores ranging from 1 to 6. The scale deals with several aspects such as anxiety (psychological and somatic), depressed mood, insomnia subdiagnosis of severe OSA (AHI > 30). Cases were divided into two groups for statistical purpose: snorers/mild OSA (AHI ≤ 15) and moderate/severe OSA (AHI > 15). Cases were analyzed considering the presence/absence of heavy drinking. The protocol was approved by the local Research Ethics Committee and written informed consent was obtained in all cases.

Methods

Study design

This was a cross-sectional study of 150 consecutive patients referred for polysomnography with clinical suspicion of OSA. Cases with cancer, severe neurological, renal, hepatic, lung or cardiac diseases were excluded. Five individuals declined to participate in the study and five others were considered too ill to participate. Among the latter, three had dementia and two had suffered a recent ischemic stroke. Thus, the final sample consisted of 140 patients. Cases included in the study were not involved in shift work and did not have recent hospitalizations for the last three months. A structured face-to-face interview was conducted prior to sleep study. All cases were submitted to overnight polysomnography. Those with AHI ≤ 5 events per hour of sleep were considered primary snorers. Obstructive sleep apnea was diagnosed in subjects with an AHI > 5. These patients were further classified as having mild (5 < AHI < 15), moderate (15 < AHI < 30) or severe OSA (AHI > 30). Cases were divided into two groups for statistical purpose: snorers/mild OSA (AHI ≤ 15) and moderate/severe OSA (AHI > 15). Cases were analyzed considering the presence/absence of heavy drinking. The protocol was approved by the local Research Ethics Committee and written informed consent was obtained in all cases.

Procedures

Demographic and clinical data were recorded using a closed-question data collection instrument. Body mass index (BMI) was calculated as the ratio between weight (kg) and squared height (m²). Special emphasis was put on the history of heavy drinking and use of medication in the previous 30 days. Heavy alcohol drinking was considered present if, on a daily basis, more than 4 drinks for men and more than 3 drinks for women were reported, or more than 14 drinks for men and more than 7 drinks for women, on a weekly basis.

Hamilton Rating Scale for Depression

Depressive symptoms were evaluated by the Hamilton Rating Scale for Depression - 17 item (HRSD). This scale takes into consideration several aspects such as anxiety (psychological and somatic), depressed mood, insomnia subdivided in early, middle and late insomnia among other mood-related questionings.

Epworth sleepiness scale

Daytime somnolence was assessed by the Epworth sleepiness scale (ESS), a questionnaire containing eight items that ask for expectation of dozing in eight hypothetical situations. Dozing probability ratings range from zero (no probability) to three (high probability). An ESS score of 10 or more indicates excessive daytime sleepiness.

Comorbidity Index Severity

Comorbidities were investigated using the modified Charlson Comorbidity Index (CCI). The CCI has been validated and is a strong predictor of clinical outcome in this population. The CCI is a composite score of multiple comorbid conditions, scores ranging from 1 to 6. The scale deals with
questions regarding organ system pathology such as the cardiovascular, respiratory, gastrointestinal, genitourinary, muscle-skeletal and neuro-psychological system. For the purpose of this study, the comorbid conditions were evaluated both by interview and confirmed by chart review and scored accordingly. However, age was not included in the index in order to examine the influence of age, independent of comorbidities.

**Polysonmography**

Standard overnight polysomnography (PSG) was performed on a digital polygraph (ALICE III®, Respironics Inc.). Polysomnographic recordings were set to begin at 10 p.m. (lights-out) and end at 6 a.m. (lights-on). Monitored variables included: electroencephalogram (C3, C4, O1, O2 referenced to contralateral ear electrodes), bilateral electrooculograms, submental electromyogram (EMG), two-lead electrocardiogram, pulse oximetry, bilateral *tibialis* EMG and airflow, using a nasal/oral thermocouple. Body position and thoracic and abdominal movements (inductance plethysmography) were also recorded. Sleep staging was performed by 30-s epochs, according to standard procedures. Polysomnography-derived parameters evaluated were AHI, minimum oxygen saturation (SpO₂₉₉), sleep latency, sleep efficiency, REM sleep latency, amount of REM sleep (% of total sleep time), amount of non-rapid eye movement (NREM) sleep (% of total sleep time), number of arousals and periodic leg movements. Arousal analysis and scoring of respiratory events during sleep were performed according to published criteria. Apneas were defined as cessation of airflow for 10s or more and hypopneas as a reduction of inspiratory air flow of 50% or more associated with either oxygen desaturation of >3% or an arousal. Severity of sleep-disordered breathing was estimated by calculating the apnea index (AI; apneas per hour of sleep) and the apnea-hypopnea index (AHI; apneas plus hypopneas per hour of sleep).

**Statistical analysis**

Statistical analysis was carried out using SPSS for Windows, version 16.0. For univariate analysis, we used the Fisher Exact Test for categorical variables, Student’s *t*-test, or Mann-Whitney U test, as appropriate. Pearson correlation test was used between variables. Multivariate analysis was performed to estimate the independent contributions of variables. Variables with a p value ≤ 0.1 by univariate analysis were selected for entry into multivariate analysis. A two-tailed p-value <0.05 was considered to indicate a significant difference.

**RESULTS**

Patients of both genders (n=144; 65% of male gender), aged 19 to 81 years (mean age 54.6 ± 8.2 years) were included in the study. Fifty-three individuals (37.8%) were identified as heavy drinkers. Table 1 depicts clinical and demographic data of cases according to the presence or absence of heavy drinking. Patients who were heavy drinkers were younger (*p=0.01*) and predominantly of male gender (*p=0.003*). Table 2 summarizes clinical and polysomnographic characteristics of patients according to OSA severity and to the presence/absence of heavy drinking.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Heavy drinking n=53</th>
<th>Non-heavy drinking n=87</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) mean (SD)</td>
<td>41.1 (14.1)</td>
<td>47.4 (14.2)</td>
<td>* 0.01*</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>43/10</td>
<td>49/38</td>
<td><em>b 0.003</em></td>
</tr>
<tr>
<td>BMI mean (SD)</td>
<td>29.4 (5.9)</td>
<td>28.6 (5.9)</td>
<td>* 0.41</td>
</tr>
<tr>
<td>HDRS scores mean (SD)</td>
<td>6.4 (4.2)</td>
<td>7.4 (5.6)</td>
<td>* 0.26</td>
</tr>
<tr>
<td>ESS scores mean (SD)</td>
<td>10.6 (4.3)</td>
<td>9.6 (4.7)</td>
<td>* 0.24</td>
</tr>
<tr>
<td>CCI mean (SD)</td>
<td>3.3 (1.6)</td>
<td>3.6 (2.1)</td>
<td>* 0.28</td>
</tr>
<tr>
<td>Hypertension (Yes/No)</td>
<td>27/59</td>
<td>24/30</td>
<td>* 0.14</td>
</tr>
<tr>
<td>Sleep latency (min) mean (SD)</td>
<td>11.8 (14.9)</td>
<td>13.2 (9.8)</td>
<td>* 0.50</td>
</tr>
<tr>
<td>REM latency (min) mean (SD)</td>
<td>102.7 (58.6)</td>
<td>119.8 (69.3)</td>
<td>* 0.13</td>
</tr>
<tr>
<td>Arousal index (events/h) mean (SD)</td>
<td>32.1 (17.9)</td>
<td>27.8 (16.2)</td>
<td>* 0.25</td>
</tr>
<tr>
<td>Sleep efficiency (%) mean (SD)</td>
<td>86.9 (10.5)</td>
<td>84.8 (11.0)</td>
<td>* 0.26</td>
</tr>
<tr>
<td>AHI (events/h) mean (SD)</td>
<td>28.9 (23.3)</td>
<td>26.1 (22.9)</td>
<td>* 0.57</td>
</tr>
<tr>
<td>Mean SpO₂ (%) mean (SD)</td>
<td>92.4 (3.1)</td>
<td>92.8 (3.0)</td>
<td>* 0.53</td>
</tr>
<tr>
<td>Minimum SpO₂ (%) mean (SD)</td>
<td>79.8 (10.0)</td>
<td>81.4 (8.7)</td>
<td>* 0.32</td>
</tr>
<tr>
<td>SpO₂ &lt;90% (%TST) mean (SD)</td>
<td>16.2 (22.3)</td>
<td>13.6 (21.3)</td>
<td>* 0.65</td>
</tr>
</tbody>
</table>

BMI: body mass index; HDRS: Hamilton Depressive Rating Scale; ESS: Epworth Sleepiness Scale; CCI: Charlson Comorbidity Index; SpO₂: peripheral oxygen saturation; REM: rapid eye movement; TST: Total Sleep Time.

* Student’s *t*-Test, *b* Fisher Exact Test, *c* Mann–Whitney Test.

*p<0.05; **p<0.01
ence/absence of heavy drinking. Eighty-six cases (61.4%) were diagnosed as moderate/severe OSA and 54 as primary snorers/mild OSA. Heavy drinkers from the group of snorers/mild OSA were younger (Student’s t test, p=0.001), predominantly of male gender (Fisher Exact Test, p=0.04) and presented more daytime sleepiness (Mann-Whitney, p=0.03) than non-heavy drinkers from the same group. In this same group, after controlling for age and gender, ESS scores tended to be higher in heavy drinkers (p=0.08). In the moderate/severe OSA group, heavy drinkers were more predominantly of male gender (Fisher Exact Test, p=0.02; Table 2). Also, in the moderate/severe OSA group, non-heavy drinkers had more depressive symptoms (p=0.01) and higher comorbidity severity (p=0.04) than heavy drinkers. However, these differences did not remain significant after adjusting for age (p=0.54 and p=0.33, respectively). Among heavy drinkers, there was a correlation between HDRS scores and BMI (r=0.53; p=0.02). These findings remained after controlling for age (men: r=0.22; p=0.04 and women: r=0.36; p=0.008). Among all cases, scores of the HDRS were higher in female (r=0.48) than in men (r=0.38; p=0.008). Age was correlated with comorbidity severity (r=0.24; p=0.003), but not with depressive symptoms, in both groups (Table 3).

Overall, 17 individuals (12.1%) used sedatives, mostly benzodiazepines, and eight cases (5.7%) were on antidepressants (selective inhibitors of serotonin reuptake) without any differences between heavy drinkers and non-heavy drinkers (Fisher Exact Test, p=0.81).

### DISCUSSION
This data show that, among individuals with clinical suspicion of OSA, heavy drinkers are most predominantly young men. Also, among snorers and mild OSA subjects who are heavy drinkers, a trend for more daytime somnolence was found. We hypothesize that increased daytime sleepiness observed in snorers/mild OSA that are heavy drinkers may be secondary to more disrupted sleep in these individuals compared to non-
heavy drinkers. However, we were not able to demonstrate any differences in objective sleep parameters between these two groups. There are limitations to this study that need to be acknowledged: this is a cross-sectional evaluation of patients referred for sleep study and it is possible that these findings may not be representative for the general population. Other important issue is the potential influence of circadian rhythm abnormalities such as delayed sleep phase syndrome, a potential contributor to daytime sleepiness that was not evaluated in these cases. Although prospective studies are more suited for the investigation of chronic effects of heavy-drinking, they are costly, present difficulties with adherence to protocol and involve some ethical issues.

Using a different study design, Aldrich et al. looked for the prevalence of OSA in heavy drinkers and reported increased disease severity with ageing(25). In the present study, cases that were older and had more severe OSA were not those identified as heavy drinkers. A possible explanation for this finding could be a greater awareness about the deleterious effects of heavy drinking among older individuals. After counseling, a follow-up of these patients including OSA severity and depressive symptoms evaluation may further clarify this issue. In this study, BMI was positively correlated with depressive symptoms in young heavy drinkers. This is in agreement with a previous report that points out the importance of body image on mood, particularly in young individuals(24). Others have reported an association between the BMI and depressive symptoms(25,26). In this study population, depressive symptoms as evaluated by the HDRS were frequent, affected more women than men, and in most cases, remained untreated. This is also in agreement with a previous report showing that women with OSA present significantly higher levels of depression and anxiety than men(27). Recently, it has been shown that women with OSA complain significantly more of insomnia, restless legs, depression, nightmares, palpitations at night, and hallucinations than men(25). It has also been suggested that gender-related differences in symptom profile may be one explanation for the clinical under recognition of sleep disordered breathing in women(26,28). Physicians involved in the care of patients with sleep disordered breathing should take notice of the necessity to investigate and diagnose more accurately depression in the obese and young cases with clinical suspicion of OSA.

In this study, AHI severity was not correlated with depressive symptoms. The relationship between depression and severity of OSA is controversial. Millman et al. reported that severity of OSA was associated with increased symptoms of depression(29). On the other hand, Pillar and Lavie did not observe any relationship between anxiety and depression and the severity of OSA in male patients, in agreement with our findings(27).

Our data do not support previous reports of an association between excessive daytime sleepiness and depressive symptoms in OSA(30,31). Oxygen desaturation and high number of arousals, but not the AHI, were correlated to daytime somnolence, as assessed by the ESS. Previously, excessive daytime sleepiness has been associated with hypoxia(26) as well as snoring, poor sleep efficiency and increased total number of arousals(32).

In summary, this study shows that heavy drinking is common in young snorers/mild OSA male patients presenting for sleep laboratory studies and snorers/mild OSA that are heavy drinkers tend to have more pronounced daytime sleepiness. Among these young individuals, HDRS scores were correlated with the BMI and depressive symptoms were more severe in women.

CONFLICT OF INTEREST
The authors have nothing to disclose.

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