



The Egyptian Society of Chest Diseases and Tuberculosis
Egyptian Journal of Chest Diseases and Tuberculosis

www.elsevier.com/locate/ejcdt
www.sciencedirect.com



ORIGINAL ARTICLE

Overlap of obstructive sleep apnea and bronchial asthma: Effect on asthma control



Mohamed Zidan, Rasha Daabis *, Heba Gharraf

Dept. of Chest Diseases, Faculty of Medicine, Alexandria University, Egypt

Received 17 December 2014; accepted 18 January 2015

Available online 18 February 2015

KEYWORDS

Obstructive sleep apnea;
Bronchial asthma;
Asthma control;
Severity;
GERD;
Obesity

Abstract Obstructive sleep apnea (OSA) and asthma are highly prevalent respiratory disorders that share several risk factors and are frequently comorbid. Multiple mechanisms have been postulated to explain this frequent coexistence, which is recently referred to as the “alternative overlap syndrome”. Moreover, OSA is generally linked to worse asthma outcomes.

Objectives: First, to assess the prevalence of OSA in a group of asthmatics. Second, to evaluate the potential risk factors underlying the development of OSA in these patients. Third, to determine the effect of this overlap on asthma control.

Methods: Polysomnography was done for 30 asthmatics and 12 controls. Demographics, spirometry, comorbidities and clinical data were collected. Asthma control was assessed according to the latest GINA guidelines.

Results: OSA defined by an AHI of ≥ 5 events/h was present in 18 (60%) asthmatics and 2 (17%) controls. Regression analysis revealed that high body mass index (BMI), coexistent gastroesophageal reflux disease (GERD) and asthma severity (FEV1%) are significant independent predictors for the development of OSA in asthmatics ($p = 0.03$, 0.034 , and < 0.001 respectively). Moreover, the presence of OSA in asthmatic patients was significantly associated with worse asthma control ($p < 0.001$).

Conclusion: A high index of suspicion is warranted for the overlap of OSA and asthma, particularly in the presence of obesity, GERD, and in patients with severe asthma. Individualized therapy addressing these moderating factors is warranted for optimal health outcomes. Recognition and treatment of OSA in asthmatics is an important element in improving asthma control.

© 2015 Production and hosting by Elsevier B.V. on behalf of The Egyptian Society of Chest Diseases and Tuberculosis. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Obstructive sleep apnea (OSA) and asthma are highly prevalent chronic respiratory disorders that share several risk factors and are frequently comorbid [1]. Both symptoms of

OSA and diagnosed OSA are more frequent in clinical populations with asthma compared to other populations [2–4].

In addition to overlapping risk factors, multiple evidence-based and hypothetical mechanisms have been postulated to explain the frequent coexistence of OSA and asthma, also referred to as the “alternative overlap syndrome” [4–7].

* Corresponding author.

Peer review under responsibility of The Egyptian Society of Chest Diseases and Tuberculosis.

Asthma is an inflammatory disease of the lower respiratory tract, manifesting as intermittent constriction of the bronchial airways. Obstructive sleep apnea (OSA), on the other hand, is a state-dependent condition that is characterized by intermittent obstruction of the upper airway during sleep leading to hypoxemia and sleep fragmentation [8]. The pathophysiology of these two conditions seems to overlap significantly, as airway obstruction, inflammation, obesity, and several other factors are implicated in the development of both diseases [5]. On the other hand it has been suggested that asthma comorbidities, such as GERD, and medications may also contribute to the development of OSA [9]. In this setting, a more specific understanding of what increases predisposition for OSA in asthma could be useful.

Moreover, OSA is generally linked to worse asthma outcomes. The effects of the direct pathophysiologic consequences of OSA (e.g., chronic intermittent hypoxemia, circadian alteration of autonomic functions, and increased intrathoracic pressure swings related to the occluded upper airway) on the clinical severity of asthma are poorly understood [1]. Moreover, the National Asthma Education and Prevention Program Expert Panel Report recommends evaluating for OSA as a potential contributor to poor asthma control [10]. Thus, clarifying the nature of the relationship between OSAS and asthma is a critical area with important therapeutic implications.

Therefore, the aim of this study was: First, to assess the prevalence of OSA in a group of asthmatics. Second, to evaluate the potential risk factors underlying the development of OSA in these patients. Third, to determine the effect of this overlap on asthma control.

Patients and methods

30 patients with bronchial asthma were enrolled in the study. Participants were recruited from consecutive patients presenting to asthma clinics of Alexandria. We included adults (age ≥ 18 years) with a diagnosis of asthma of at least 12 month duration. Data were collected regarding demographic and clinical factors, spirometry, general health information including relevant comorbid conditions such as (Gastroesophageal reflux disease (GERD), rhinitis or sinusitis) and current asthma medication.

Patient control was assessed according to the latest GINA guidelines and the level of control was divided into well controlled, partly controlled or uncontrolled [11].

12 healthy adults matched for age, sex and BMI were included as a control group. No sleep-related information was used in the recruitment process.

Exclusion criteria for both groups included the following: current smoker or ex-smoker with a greater than 10 pack-year smoking history, comorbidities that could potentially interfere with the study or other pulmonary diseases.

Nocturnal sleep studies

All subjects underwent overnight polysomnography. The patients underwent polysomnography at a time of relative clinical stability, and at least 2 weeks after recovery from any exacerbation or intervention.

The analysis was carried out automatically and manually. Respiratory events were scored using standard criteria [12]. The apnea hypopnea index (AHI) was defined as the total number of apneas and hypopneas per hour of sleep. As indices of nocturnal hypoxemia we considered the oxygen desaturation index (this is the number of times that the oxygen saturation falls by more than 3 or 4 percent per hour of sleep), T90 (the fraction of sleep time spent below an oxygen saturation of 90%) and the minimal value recorded during sleep (minimal SaO₂). The OSA was defined as an AHI of ≥ 5 events/h.

Assessment of daytime sleepiness

The Epworth Sleepiness Scale (ESS) was used for assessing daytime sleepiness. This is a commonly used self-administered scale with eight items about how easily the respondent would fall asleep in different situations. The items are scored on a 0–3 scale, which are added to give an overall score of 0–24. Higher scores indicate more sleepiness. ESS score 2–10 is considered ‘normal’ and > 10 indicative of pathological sleepiness [13].

All subjects were enrolled in the study after a written informed consent according to a protocol approved by the Ethics Committee of the Hospital.

Results

Subjects' characteristics

30 asthmatic patients and 12 healthy controls matched for age, sex and BMI were enrolled in the study. Subjects' characteristics are shown in Table 1. Asthmatic patients showed significantly more comorbidities, snoring and ESS.

Polysomnographic respiratory parameters of the studied subjects

OSA, defined by an AHI of ≥ 5 events/h, was significantly more prevalent in the asthmatics in comparison with the control group ($p < 0.001$). The polysomnographic parameters of the studied subjects are shown in Table 2.

Table 1 Subjects' characteristics.

	Asthmatic patients	Control	<i>p</i>
Age	49.12 \pm 3.43	47.8 \pm 0.56	NS
Sex(M/F)	13/17	6/6	NS
BMI	28.13 \pm 4.22	26.88 \pm 2.42	NS
GERD n(%)	12(40)	2(15)	< 0.001*
Rhinitis/sinusitis	20(67)	3(25)	< 0.001***
ESS	8.89 \pm 2.8	4.85 \pm 2.15	< 0.01**

Variables are expressed as mean \pm standard deviation.

M = male, F = female, BMI = body mass index, GERD = gastroesophageal reflux disease, ESS = Epworth sleepiness scale, NS = not significant.

* Statistically significant at $p < 0.05$.

** Statistically significant at $p < 0.01$.

*** Statistically significant at $p < 0.001$.

Download English Version:

<https://daneshyari.com/en/article/3399989>

Download Persian Version:

<https://daneshyari.com/article/3399989>

[Daneshyari.com](https://daneshyari.com)