

Obstructive sleep apnea and asthma: is it just a matter of CPAP

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Obstructive sleep apnea (OSA) may occur concomitantly with asthma. Approximately 74% of asthmatics experience nocturnal symptoms of airflow obstruction secondary to reactive airways disease. There is a higher prevalence of OSA symptoms in an asthmatic population when compared to a primary care population independent of the severity of disease (1). Sleep deprivation, chronic upper airway edema and inflammation associated with OSA may further exacerbate nocturnal asthma symptoms. In addition, steroid therapy leads to the development of alkalosis which results in greater hypoventilation during sleep (2).

There is a strong consensus that continuous positive airway pressure (CPAP) is the gold standard treatment for OSA. Currently, there is a growing interest in understanding the use of CPAP to improve both daytime and nighttime peak expiratory flow rates in patients with concomitant OSA and asthma. This is a great field for pulmonologists in studying how OSA may complicate diagnosis and treatment of asthma and in understanding the potential contributions of CPAP. This is particularly interesting if there is a history of sleep disorder and a high clinical suspicion of OSA in asthma patients, who are refractory to standard treatment regimens (3).

We were pleased to read the recent report of Teodorescu et al. which reported an original study of medical records that were reviewed for an established diagnosis of OSA and CPAP use among asthma patients. Major conclusions were that, high OSA risk was associated with persistent daytime and nighttime asthma symptoms and management of OSA led to adequate control of asthma symptoms (4). This implication is very favorable for the adequate control and prognosis of the OSA and opens up a new connection via. During episodes of OSA, nocturnal hypoxemia and hypercapnia occur repeatedly in addition to an inflammatory response leading to increased bronchial hyperreactivity resulting in difficult control of symptoms in these patients. However, we would like to comment especially on the practical implications involved in the treatment of both diseases:

1. There is no clear correlation between tests for OSA and asthma, such as the apnea hypopnea index (AHI) and bronchial reactivity testing. There are also no adequate correlation oximetry values for patients with OSA and asthma symptoms (5).

2. Daytime and nighttime asthma symptoms could be influenced by other comorbidities. In this study comor-

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bidity informations were obtained retrospectively from medical reports and they were not adequately separated in the population tested leading to a potential bias in the severity of symptoms reported.

3. In this study the pulmonary functional tests and CPAP trials in OSA, are important in understanding the observed changes to patient symptoms. However, there were no results of positive or negative correlation analysis provided. This is relevant because we know from previous studies that functional tests do not always correlate to improvement in patient symptoms (6).

4. Also in this study CPAP setting and criteria used, and the degree of correlation with asthma control is unknown. Another essential aspect is to know the level of adherence to CPAP in these patients. This was not reported in this study (7).

5. The questionnaire used for evaluation of OSA, along with its correlation to polysomnography studies are essential for the proper diagnosis of OSA. This was not clearly revealed in this study.

6. Influence of body mass index (BMI), especially a BMI > 35 kg/m², was shown in other studies to have a significant correlation with symptoms. This information was not available to interpret the results of this study (8).

In conclusion, although the results of this study contributes to the early application of CPAP in patients with asthma and OSA, a larger prospective trial is needed to identify specific factors and specific patient population that would benefit from this treatment approach.

CONFLICT of INTEREST

None declared.

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