Asthma and Obstructive Sleep Apnea
At Different Ends of the Same Airway?

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. Asthma symptoms are often worse at night. Many possible explanations have been put forth for the nocturnal deterioration of asthma symptoms, including alterations in the autonomic tone, changes in hormonal secretion, circadian changes in inflammatory cells and cytokines, and the possible role of acid reflux in the worsening of asthmatic symptoms at night.

A relationship between asthma and OSA was noted 25 years ago. Hudgel and Shucard described severe hypoxemia in coexistent asthma and OSA. Catterall et al. noticed more hypoxemic episodes, especially during rapid eye movement sleep, in a group of 10 asthmatic patients compared to control subjects. Interestingly, they noticed the appearance of apneas and hypopneas but commented that these asthmatic patients did not have “classical sleep apnea syndrome.” In a later study, Fitzpatrick et al. noted that 11% of asthmatic patients reported frequent snoring (ie, ≥4 nights a week) and frequently reported falling asleep while driving or operating machinery. In another questionnaire study of 2,202 subjects (267 asthmatic subjects), the presence of asthma was a stronger predictor of self-reported apneas than the other common risk factors like male sex, body mass index, and age. Despite these earlier observations, there has been a paucity of literature addressing comorbid OSA in asthmatic patients until recently.

What predisposes asthmatic patients to the development of OSA? In this issue of CHEST (see page 1125), Teodorescu and colleagues have taken a step further to address this important question. In a questionnaire-based cross-sectional study of 244 well-characterized patients from a specialty asthma clinic, they used the sleep apnea scale of sleep disorders questionnaire to assess OSA risk and daytime and nighttime asthma symptoms to assess asthma severity. The diagnosis of comorbidities like gastroesophageal reflux disease (GERD), sinusitis, and rhinitis was based on chart review. Importantly, they quantified doses of inhaled corticosteroids (ICSs) as low, medium, or high based on the National Asthma Education and Prevention Program guidelines for analysis.

The strengths of the study by Teodorescu and colleagues are that the patients were drawn from a specialty clinic and National Asthma Education and Prevention Program guidelines were used to define the severity of asthma and the classification of ICS doses. However, the lack of polysomnographic diagnoses of OSA is a limitation of the study, since the sleep apnea scale of sleep disorders questionnaire has not been validated in asthmatic patients. Further, the diagnosis of GERD was based on chart review and not on predefined criteria. Despite these limitations, this is the first study to suggest that the use of ICSs is correlated with the risk of OSA in asthmatic patients in a dose-dependent manner. The odds ratio of having OSA was 1.99 with low-dose ICS therapy and 6.83 with high-dose ICS therapy. The odds ratio was altered only slightly when asthma severity was taken into consideration. Interestingly, female gender was associated with a higher risk of OSA in this population of asthmatic patients. In contrast to some of the earlier studies, the association was similar with either daytime or nighttime asthma symptoms.

Since this is a cross-sectional study, a firm inference cannot be drawn about whether ICSs cause an increased risk of OSA. This question warrants further investigation in separate studies. Although laryngeal muscle dysfunction has been reported with the use of ICSs, a myopathic effect on pharyngeal dilators has not been reported. If ICSs were to cause pharyngeal muscle myopathy, some effects like dysphagia or dysarthria would be expected during the daytime. Also, some of these patients would be expected to have concomitant dysphonia due to the proximity of laryngeal muscles to the pharynx. To complicate the issue, the use of nasal steroids, some portion of which drain into the throat, has been associated with improvement of OSA, at least in children. Another potential mechanism by which ICSs increase the risk...
of OSA is possible increased fat deposition in phar-
ynx. Finally, it is possible that perhaps the dose of
ICSs was a surrogate marker of asthma severity.
However, Teodorescu et al\(^5\) found that even after
the inclusion of asthma severity and FEV\(_1\) in the
analysis, ICS dose remained a significant predictor
for the risk of OSA.

Portable monitoring should allow for easier
screening of asthmatic patients for OSA. Polysom-
ography and volumetric or imaging studies of the
upper airway before and after the initiation of ICS
therapy could provide evidence needed in favor of a
causative role for ICSs in increasing the risk of OSA.
If ICS therapy does increase the risk of OSA, newer
technologies for minimizing pharyngeal deposition and
different formulations to decrease the local effects
would need to be developed.

The authors found that asthmatic women were more
prone to the development of OSA. This finding has
important implications. Women have been shown\(^7\) to
require more urgent care visits despite better knowl-
dge and more frequent use of ICSs. Asthmatic women
have also been shown\(^8\) to have a higher incidence of
cardiovascular disease. Studies are needed to address
the role of OSA in the increased morbidity of asthma in
women and the increased risk of cardiovascular disease
in asthmatic women, and to understand the underlying
mechanisms. Obesity and GERD are likely to play an
important intermediary role in defining relationship
between asthma and OSA. OSA has been shown to
contribute to obesity which in turn is associated with
worsening of asthma and OSA.\(^9,10\) There is preliminary
evidence\(^11,12\) that positive airway pressure treatment
for OSA can improve asthma control, although this
needs to be studied in large prospective trials. As the
epidemics of asthma and obesity increase, it is not the
time to miss the boat, but to get to know the ropes and
study these issues urgently, not only in adults but also in
children.

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Oral Decontamination to Prevent Ventilator-Associated Pneumonia

Is It a Sound Strategy?

The development of ventilator-associated pneu-
monia (VAP) requires microbial pathogens to
gain access to the lower respiratory tract. The GI and
upper respiratory tract are thought to represent the
primary source of these pathogens in intubated
patients.\(^1\) Interruption of this process by preventing
colonization with pathogenic organisms represents a
potential target for preventing VAP.

A variety of strategies for decontamination of the
oropharynx and GI tract have been studied. The
most well-studied strategy is that of selective
digestive decontamination (SDD). SDD typically in-

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