CORRESPONDENCE

OSA in COPD: An Ignored Comorbidity

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Abstract

Obstructive sleep apnea (OSA) diagnosis in chronic obstructive pulmonary disease (COPD) patients is an ignored comorbidity. While excessive daytime sleepiness (EDS) patients are diagnosed and treated early, delayed diagnosis in non-EDS patients may lead to adverse outcomes, especially in the form of cardiovascular events. The conventional questionnaires make use of EDS and thus become nugatory in non-EDS patients. Submental ultrasound has proven beneficial for screening for OSA as well as directing its management and monitoring of treatment.

Keywords: Chronic obstructive pulmonary disease, Obstructive sleep apnea, Overlap syndrome, Sleep score.

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Introduction

Obstructive sleep apnea (OSA) remains highly underdiagnosed worldwide. It is characterized by recurrent episodes of reduced inspiratory airflow caused by upper airway obstruction during sleep. Although it occurs commonly in obese individuals, studies have revealed that non-obese individuals account for at least 20% of adult OSA cases.[1]

Chronic obstructive pulmonary disease (COPD) has been defined as a "heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, expectoration, exacerbations) due to abnormalities of the airway (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction".[2]

Obstructive sleep apnea and COPD are commonly associated diseases, affecting and affected by sleep. These are prevalent worldwide in nearly 10% population and gradually increasing.[2]

In COPD, the effect on respiratory physiology which occurs during sleep results in a myriad of sleep-related breathing disorders.[2] In emphysema, the flattening of the diaphragm is aggravated in sleep. This is compensated by the use of accessory muscles when the patient is awake. However, in the rapid-eye movement (REM) stage of sleep, since the function of accessory muscles is diminished, hypoventilation may worsen in such patients. This combination of ventilation-perfusion mismatch and hypoventilation causes sustained hypoxemia during sleep.[2]

COPD-OSA Overlap

The co-occurrence of OSA and COPD is labeled as overlap syndrome (Figs 1 and 2). As compared to either COPD or OSA, patients with overlap syndrome have a poorer quality of life and an increased risk of pulmonary hypertension. These patients also have an increased susceptibility to cardiovascular disease.[3]

Obstructive sleep apnea is usually screened for when the patient presents with excessive daytime sleepiness (EDS) in the background of risk factors. However, EDS is not universal. Studies have shown that up to 25–50% of OSA patients do not report subjective tiredness or sleepiness.[4]

USG to Screen for OSA in COPD

Ultrasound is a portable, quick, and low-cost alternative for screening for OSA in COPD patients. Ultrasound can be used to measure various tongue parameters such as tongue thickness, the distance between lingual arteries (DLAs), tongue base height (sagittal plane); as well as pharyngeal parameters such as lateral pharyngeal wall (LPW) thickness, retropalatal (RP) and retroglossal diameters. This has proven useful in various meta-analyses and reviews.[5–7] Submental ultrasound can be utilized

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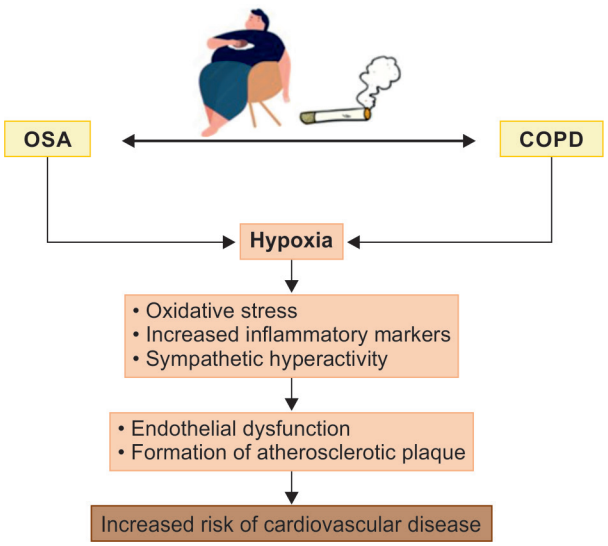

Fig. 1: Pathophysiological mechanism for development of cardiovascular disease

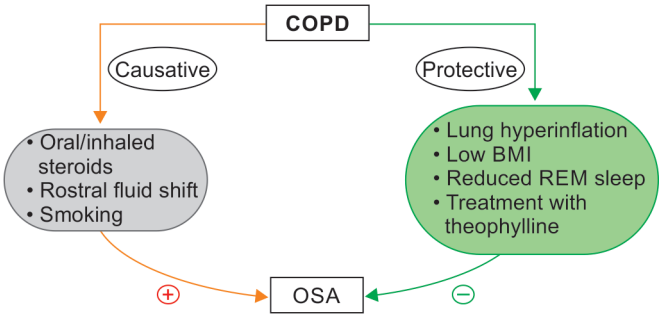

Fig. 2: COPD-OSA correlation

for the detection of OSA in COPD for early management and better outcomes.

There are no uniform guidelines for screening for OSA in COPD patients. Chronic obstructive pulmonary disease patients with OSA may not have EDS, thus challenging its early diagnosis and management to prevent adverse cardiovascular events.

There are several screening questionnaires for OSA, such as the Epworth Sleepiness Scale (ESS), STOP-Bang questionnaire, and the Berlin questionnaire (BQ). However, the sensitivity and accuracy of overlap syndrome (OS) are low. Also, as EDS is uncommon in OS patients, many of these questionnaires, which ask about subjective sleepiness, would be unreliable.

During sleep, there are changes in breathing patterns, decreased respiratory drive due to blunted chemoreceptor sensitivity, and altered lung mechanics in terms of fall in functional residual capacity (FRC).[8] These changes are predominantly seen in REM sleep.[9] These lead to alveolar hypoventilation, hypoxemia, and hypercapnia. Normal subjects can easily compensate for these changes. However, in COPD patients, who are predominantly dependent on the use of respiratory muscles, this could result in oxygen desaturations during sleep. Nocturnal oxygen desaturations (NOD) are more frequent and often severe in overlap syndrome than in COPD or OSA alone. Nocturnal oxygen desaturations can be diagnosed as ≥30% of the total recording time (during sleep) spent with a transcutaneous SaO_2 <88% or 90%.[10,11] Various underlying mechanisms postulated include ventilation perfusion mismatch, hypoventilation at the alveolar level, and altered lung mechanics during sleep.[12]

Amongst these, hypoxia is the preeminent mechanism, as it causes the release of systemic inflammatory cytokines.[12] Reactive oxygen species (ROS) are released which cause endothelial dysfunction and accelerated atherosclerosis. This increases the risk of cardiovascular events, pulmonary hypertension, and increased mortality. Since these patients are less often sleepy, they hardly undergo polysomnography and the diagnosis is delayed.

In a study by Mohammad OI et al.,[13] the OSA was found to co-exist in 50% of COPD patients, the proportion being even higher in severe COPD cases and obese patients. There was no difference in the efficiency of sleep between non-obese and obese OSA cases. Forced expiratory volume in 1 second (FEV1), body-mass index (BMI), and oxygenation-desaturation index (ODI) were found to be predictors of underlying OSA in COPD patients. Even in the study by Gunduz C et al.[14] mild COPD patients with no sleep apnea symptoms, showed a high prevalence 26/45 (58%) of OS. In another study by Venkateswaran S and Tee,[15] the prevalence of overlap syndrome was 63.6%. The majority of patients with COPD and concomitant OSA were non-obese and non-sleepy and such patients presented with cardiac events. This makes it essential to diagnose these patients early. In a study by Marin JM et al.,[16] it was seen that patients who were not treated with PAP therapy in overlap syndrome had higher mortality and greater risk of exacerbations of COPD.

Polysomnography undoubtedly remains the gold standard for diagnosis of OSA or OS,[17,18] however it has certain impediments like overnight in-laboratory stay and cost. Also, PSG data may be hampered by the 'first night' effects (i.e., decreased or absent slow wave and REM sleep and increased wakefulness and stage 1 NREM sleep). Availability of PSG in low-economic countries like India is also limited.

In children, MRI has been used to evaluate upper airway abnormalities and obstruction in patients with OSA. It carried no radiation hazard. However, longer acquisition times and costs remain the limitations.[19]

Ultrasonography is available across small centers around the country and has a simple learning curve, no risk of radiation, is cost-effective, time-effective, and can be done at the bedside. It has shown good sensitivity as a screening tool for OSA in several studies.[5–7]

Submental ultrasonography can help in:

- Evaluation of muscle activity in the submental region.
- Identifying airway collapsibility by detecting changes in airway caliber and collapsibility.
- Assessing tongue position in patients where the tongue is the culprit in airway obstruction.
- Treatment monitoring by tracking changes in upper airway dynamics on follow-up.

Upper airway length (UAL) significantly correlates with apnea-hypopnea index (AHI), as demonstrated by Shu et al.[6] The RP diameter was minimum in cases of severe OSA and maximum in those without OSA on expiration during forced inspiration, tidal breathing, and Müllers maneuver. The severe OSA patients documented the maximum thickness of the tongue and the longest UAL.

In a meta-analysis done by Singh et al.,[7] the distance found in between the lingual arteries (>30 mm; 67% sensitivity and 59% specificity); mean resting tongue thickness (>60 mm; 85% sensitivity and 59% specificity); tongue base thickness during Müller maneuver (59% sensitivity and 78% specificity); and a combination of neck circumference and RP diameter shortening during MM (100% sensitivity and 65% specificity) correlated well with moderate–severe OSA.

An increase in the thickness of the lateral para pharyngeal wall (LPWT) and DLAs were statistically significant in OSA patients, as shown by Hussein SA et al.[20] In OSA patients, a significant decrease in the retropalatal pharynx transverse diameter (RPD) was also seen. Thus, a combined measurement of LPWT and RPD can help in achieving high sensitivity along with high specificity in the detection of OSA. There are currently no studies evaluating the role of submental ultrasonography in screening for OS in patients with COPD.

Conclusion

Early diagnosis of comorbidities in patients with COPD is essential to improve morbidity and reduce mortality. Obstructive sleep apnea is a common association, early treatment of which can help prolong life expectancy in patients with COPD. Since a significant number of overlap syndrome patients are non-EDS, they rarely undergo polysomnography and often remain undiagnosed in the early stages before the onset of any complications. Later these

may directly present with some cardiac events and have adverse outcomes. Thus, it is essential to find an effective screening method for OSA in this important subset of patients. Submental ultrasonography is a potential tool for this purpose, however, sufficient evidence is lacking at this point in time.

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