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REVIEW

Diagnosis and management of obstructive sleep apnea in patients with chronic obstructive pulmonary disease

Diagnostic et prise en charge de l'apnée obstructive du sommeil chez les patients atteints de la broncho-pulmonaire chronique obstructive

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality and is characterized by breathing impairment and related symptoms. Obstructive sleep apnoea (OSA) is strongly associated with COPD and is a highly prevalent condition that adversely affects breathing during sleep. The co-occurrence of OSA and COPD in an individual has been referred to the overlap syndrome (OVS). Patients with COPD and OSA may have more frequent and severe nocturnal arterial hypoxemia and hypercapnia than in patients with OSA alone, as well as increased pulmonary hypertension and dysrhythmia. The presence of OSA in patients with COPD has also been implicated as a risk factor for COPD exacerbations and associated hospitalizations. Patients with overlapping syndrome, when compared with patients with obstructive respiratory disease only, had a higher Epworth score, lower total sleep time, and sleep efficiency. Age, gender, BMI and the presence of co-morbidities such as hypertension may be superior beside the traditional symptoms of OSA. Continuous positive airway pressure (CPAP) therapy reduces the mortality rate in people who have both COPD and OSA. The co-occurrence of OSA and COPD is common condition with high number of patients. The clinical signs such as snoring, nocturia and daytime sleepiness (Epworth score) and PSG are required signs and very useful to detect OSA in COPD patients. The most effective treatment option for OSA is CPAP therapy, which helps keep the airway open by providing a stream of air through a mask that is worn during sleep.

KEYWORDS: OSA; COPD; AHI; Epworth; Overlap syndrome; CPAP.

RÉSUMÉ

La maladie pulmonaire obstructive chronique (MPOC) est une cause majeure de morbidité et de mortalité et se caractérise par des difficultés respiratoires et des symptômes associés. L'apnée obstructive du sommeil (AOS) est fortement associée à la BPCO et constitue une affection très répandue qui affecte négativement la respiration pendant le sommeil. La cooccurrence de l'AOS et de la BPCO chez un individu a été appelée syndrome de chevauchement (OVS). Les patients atteints de BPCO et d'AOS peuvent présenter une hypoxémie artérielle et une hypercapnie nocturnes plus fréquentes et plus graves que les patients atteints d'AOS seule, ainsi qu'une augmentation de l'hypertension pulmonaire et de la dysrythmie. La présence d'AOS chez les patients atteints de BPCO a également été impliquée comme facteur de risque d'exacerbations de BPCO et d'hospitalisations associées. Les patients présentant un syndrome de chevauchement, par rapport aux patients atteints uniquement d'une maladie respiratoire obstructive, présentaient un score d'Epworth plus élevé, une durée totale de sommeil et une efficacité du sommeil inférieures. L'âge, le sexe, l'IMC et la présence de comorbidités telles que l'hypertension peuvent être supérieurs aux symptômes traditionnels de l'AOS. La thérapie par pression positive continue (CPAP) réduit le taux de mortalité chez les personnes atteintes à la fois de BPCO et d'AOS. La cooccurrence de l'AOS et de la BPCO est une pathologie courante touchant un nombre élevé de patients. Les signes cliniques tels que le ronflement, la nycturie et la somnolence diurne (score d'Epworth) et la PSG sont des signes obligatoires et très utiles pour détecter l'AOS chez les patients BPCO. L'option de traitement la plus efficace contre l'AOS est la thérapie CPAP, qui aide à maintenir les voies respiratoires ouvertes en fournissant un flux d'air à travers un masque porté pendant le sommeil.

MOTS CLÉS: AOS; BPCO; AHI; Epworth; Syndrome de chevauchement; CPAP.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) affects about 10% of adults and obstructive sleep apnea (OSA) is also reported to have a similarly high incidence [15]. COPD and OSA are highly prevalent diseases and thus likely to coexist in the same patient; a disorder otherwise known as overlap syndrome (OS). [24][8] According to S Duong-Quy et al (2018): OSA has apnea - hypopnea index (AHI) > 5 found in 8.5% and AHI > 15 (5.2% of cases) [21]. A recent review by Shawon et al also concluded that the incidence of OVS ranged from 2.9% to 65.9% in patients with COPD [16]. OSA was associated with overall worse outcomes in patients with COPD. Clearly, there is an increasing demand to identify OSA among the COPD population. Scientists have already tried to explore associations between these two diseases, in terms of epidemiology, overlapping pathophysiology, shared risk factors, presence of other comorbidities, clinical outcomes, and optimal management [10].

Surprisingly, body mass index (BMI) and smoking pack years, but not the Epworth Sleepiness Scale (ESS)' score, were found to be predictors of coexistent OSA in a group of COPD patients [19]. Moreover, increased risk for OSA in patients with COPD was related to higher scores in COPD Assessment Test and more prevalent cardiometabolic disease. In addition, the degree of airflow limitation is another issue to consider, as moderate-to-severe COPD is related to a high prevalence of OSA [17]. Of note, hyperinflation and emphysema are inversely correlated with the severity of OSA in COPD patients,[10] reinforcing the fact that the chronic bronchitis phenotype of COPD is strongly associated with OSA. In a landmark study [9], nontreated patients with OS carried a higher mortality and hospitalization risk compared to patients with COPD only.

PREVALENCE OF OSA AND COPD OVERLAP

More than three quarters of patients with COPD report nocturnal discomfort symptoms. Sleep complaints increase with more severe illness [6]. S Duong Quy reported that the prevalence of OSA in COPD patients is higher than in non-COPD group (23% versus 10%) and there was a significant correlation between AHI and snoring during sleep, nocturia and Epworth score ($r = 0.614$, $p < 0.05$, $r = 0.672$, $p < 0.05$, $r = 0.526$, $p < 0.01$) [20]. The high prevalence of OSA in lower airway diseases (asthma, COPD and asthma-COPD overlap - ACO) was from 35.5% to 64.4%.

A study of Pavel Turcania (2014) noted that the OSA rate among COPD patients who hospitalized for acute exacerbations was 51.4% [14]. And if patient has a sustain increase in CO2, the OVS rate is up to 82% according to Cristina Miralles (2013).

PATHOPHYSIOLOGY OF OSA AND COPD OVERLAP

There is a decrease in blood oxygen saturation at night compared to COPD patients without OSA or OSA alone. Both COPD and OSA are associated with a range of overlapping physiological and biological disturbances that include hypoxia and inflammation, which likely contribute to cardiovascular and other comorbidities. Thus the probability is high that the overlap syndrome will be associated with a greater risk of comorbidity than is true with either disease alone. Actually, the risk of respiratory failure increase with higher CO2 and pulmonary hypertension although the bronchial obstruction is mild or moderate level. COPD has long been recognized to be associated with oxygen desaturation during sleep, which may exceed that associated with maximum. **Figure 1** shows the overlap between asthma, COPD and OSA.

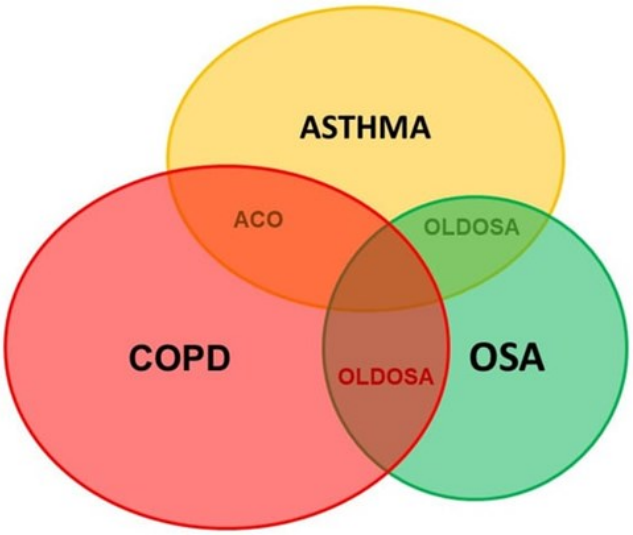

FIGURE 1. ***Comorbid obstructive lung disease and obstructive sleep apnea.***
ACO: Asthma–COPD overlap; COPD: chronic obstructive lung disease; OSA: obstructive sleep apnea; OLDOSA: obstructive lung disease and obstructive sleep apnea.

Notably, OSA and COPD share common risk factors and pathophysiological mechanisms. Age, obesity, and tobacco exposure are well-recognized risk factors. Synergistically, both disorders are associated with systemic inflammation, oxidative stress, excitations in sympathetic activity, and endothelial dysfunction. Because OSA is characterized by intermittent whereas COPD by sustained hypoxia, impaired oxygenation during both wakefulness and sleep is observed in OSA, and it is more pronounced compared to COPD or OSA alone. These characteristics can increase the risk for the development, or progression of already-established in OSA.

The model predicts the effects of transmural pressure on airflow dynamics and the severity of upper and lower airway obstruction during sleep as follows *(Figure 2)*.

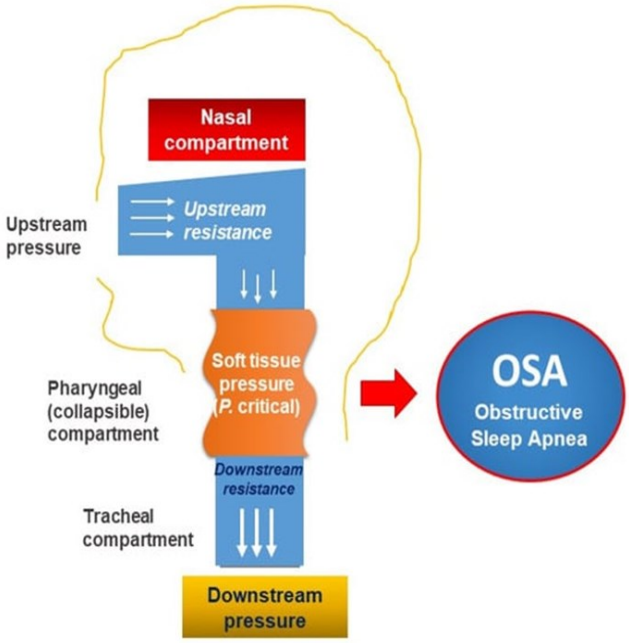

FIGURE 2. The interaction between lower airway and OSA.

The epidemiological relationship of COPD and OSA are related different clinical phenotypes. Different clinical COPD phenotypes influence the likelihood of coexisting OSA in that the increased lung volumes and low BMI associated with the predominant emphysema phenotype protects against OSA, whereas the higher likelihood of peripheral edema and increased BMI associated with the predominant chronic bronchitis phenotype promotes OSA [11].

PATHOPHYSIOLOGY OF OSA AND COPD OVERLAP

The diagnosis of OSA in patients with COPD requires awareness of relevant clinical features, and screening questionnaires may help identify suitable patients for further overnight study. Well-accepted screening tools for OSA are not always valid in COPD individuals [18]. Nevertheless, among them, the STOP-BANG questionnaire (SBQ) was found to be the most accurate [26]. Interestingly, BMI ≥25 kg/m2 and the presence of CVD perform better than other screening tools, such as the Epworth Sleepiness Scale (ESS) or the SBQ, in identifying OSA in COPD [18].

STOP-BANG questionnaire (Source: University Health Network, Toronto, Ontario, Canada (www.stobang.ca/osa/screening'php). Used with permission from Sauk Prairie Healthcare.

STOP-BANG	Yes	No
Snoring (Do you snore loudly?)		
Tiredness (Do you often feel tired, fatigued, or sleepy during the daytime?)		
Observed Apnea (Has anyone observed that you stop breathing, or choke or gasp during your sleep?)		
High Blood Pressure (Do you have or are you being treated for high blood pressure?)		
BMI (Is your body mass index more than 35 kg per m²?)		
Age (Are you older than 50 years?)		
Neck Circumference (Is your neck circumference greater than 40 cm [15.75 inches]?)		
Gender (Are you male?)		
Score 1 point for each positive response. Scoring interpretation: 0 to 2 = low risk, 3 or 4 = intermediate rick, ≥5 = high risk.		

Epworth Sleepiness Scale [4]

0	1	2	3
No chance	Slight chance	Moderate chance	High chance

Situation	Chance of dozing
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g., a theater or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

Whereas COPD can easily be diagnosed by spirometry, OSA diagnosis requires a sleep study, which is more expensive and time-consuming. Sleep physicians should choose between the gold standard polysomnography (PSG) and respiratory polygraphy. The diagnostic criteria for adult OSA as defined by the American Academy of Sleep Medicine (AASM) [1]. The patient suspected of OSAS must fulfill criterion A or B, plus criterion C. These are as follows:

A. Excessive daytime sleepiness that is not better explained by other factors
B. Two or more of the following that are not better explained by other factors:

- Choking or gasping during sleep
- Recurrent awakenings from sleep
- Unrefreshing sleep
- Daytime fatigue
- Impaired concentration

C. Overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep. These events may include any combination of obstructive apneas/hypopneas or respiratory effort–related arousals, as defined below.

They also proposed a grading of severity of OSAS based on the frequency of abnormal respiratory events during sleep: Mild: ≥ 5 but <15 events/hour of sleep; Moderate: 15–30 events/hour of sleep; Severe: More than 30 events/hour of sleep.

Effective treatment of OSA with the appropriate type of positive airway pressure (PAP) device is another matter of debate. Different strategy approaches should be applied to OSA patients with severe OSA and mild COPD than to those with mild OSA and severe COPD. Where OSA predominates, continuous PAP (CPAP) is the preferred modality, while bi-level PAP (BPAP) would be more appropriate when COPD prevails in OSA. Interestingly, the severity of nocturnal hypoxia and daytime hypercapnia in OS patients precludes the success of treatment with CPAP and shows that BPAP should be the first choice in such patients [7].

DIAGNOSIS APPROACH

The possibility that COPD may predispose to OSA and OSA to COPD has been explored in many studies over the past 2 decades. COPD and OSA are both highly prevalent, which implies that both disorders occurring together (OVS) is likely to be common based on chance association alone. Both COPD and OSA are associated with similar physiological and molecular consequences, such as hypoxia and systemic inflammation, that contribute to cardiovascular and other comorbidities, and pulmonary hypertension is highly prevalent in patients with the overlap syndrome [28-30].

Sleep quality is typically poor in COPD with diminished amounts of rapid eye movement (REM) and slow-wave sleep, which may contribute to the daytime fatigue frequently reported by these patients and may also contribute to reduced survival. A recent report has demonstrated that sleep impairment in COPD is linked to worse pulmonary function and lower daytime activity levels [12]. Lung hyperinflation also appears to relate to poor sleep quality in patients with COPD. In accordance with these findings, in this issue of this journal [12] the authors provide important evidence to support the screening of OSA in COPD patients with EDS. In this cross-sectional study, the authors assessed for the presence of EDS (based on ESS score) in 301 stable COPD patients with severe and very severe airflow obstruction. Those with EDS (n = 47) underwent an attended PSG. A high prevalence of concurrent OSA in the subgroup of sleepy COPD participants was noted (70.2%), while the overall prevalence of OSA was approximately 10%. Of note, the selective evaluation with PSG of sleepy-only COPD patients, might have underestimated the true prevalence of OSA in this cohort. Collectively, these findings add significant evidence to the existing literature on predicting the risk of OSA and highlight the need for further research on this topic [8].

TREATMENT STRATEGY

Considering the fact that OSA is fully reversible after treatment, its diagnosis is highly important, as previous data has shown that in patients with OS treatment of OSA can improve COPD related outcomes and increase patients' survival [9]. COPD is not a uniform disease but represents a spectrum of clinical phenotypes. These range from the hyperinflated patient with low BMI who typically presents with predominant symptoms of dyspnea and relatively well preserved gas exchange (predominant emphysema phenotype) to the patient with higher BMI and cor pulmonale (right-sided heart failure) who typically presents with productive cough and hypoxemia (predominant chronic bronchitis phenotype) [31-34].

Objective sleep evaluation should be offered to all COPD patients, not only in the presence of well-known risk factors or symptoms of OSA [12], but whenever sleep-related issues are mentioned [2]. Indeed, accumulating evidence shows that OS patients typically complain of fatigue, whereas insomnia is also a salient feature of sleep-reported issues in COPD patients. Patients with the overlap syndrome will present with the clinical features of each disorder to a greater or lesser extent, depending on the balance between the COPD and OSA components. However, there are also likely to be additional clinical features to reflect the higher prevalence of hypoxemia, hypercapnia, and pulmonary hypertension.

Patients with COPD and nocturnal hypoxemia benefit from inhaled long-acting beta-agonist and anticholinergic therapy with the mean nocturnal oxygen saturation being about 2% to 3% higher on each medication compared with placebo. Both agents have no significant impact on sleep quality. Theophylline also reduces nocturnal hypoxemia and has been shown to benefit OSA by reducing the AHI. The recognition of coexisting OSA in patients with COPD has important clinical relevance, as the management of patients with overlap syndrome is different from the management of COPD alone, and the survival of patients with overlap syndrome that is

not treated with nocturnal PAP is significantly inferior to that of patients with overlap syndrome that is appropriately treated.

CONCLUSION

The coexistence of OSA and COPD is common condition and increase medical burden than OSA or COPD alone. STOP-BANG and Epworth-score have screening implications for identifying patients with OVS and PSG are required signs and very useful to diagnose OSA in COPD patients. The most effective treatment option for OSA is CPAP therapy, which helps keep the airway open by providing a stream of air through a mask that is worn during sleep. Future research in this topic is needed so as to shed more light on the associations between OSA and COPD.

CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

DATA AVAILABILITY STATEMENT

This review did not have the raw data.

ETHICS STATEMENT

This review followed the Declaration of Helsinki – Ethical Principles for Medical Research involving human subjects.

AUTHOR CONTRIBUTIONS

D.NH, S.DQ, N.NTY, Q.DM, N.DT, B.LV, T.HA, Q.VTT, T.VPM , and V.NN: conceptualization, validation, and writing–original draft preparation. D.NH, S.DQ, N.NTY, T.VPM, and V.NN: methodology and writing–review and editing. All authors contributed to the article and approved the submitted version.

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