Obstructive Sleep Apnea and Depression: Issues for Psychiatrists

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What are the effects of sleep apnea on depression--and depression on sleep apnea? Insights here.

Premiere Date: August 20, 2016
Expiration Date: February 20, 2018

This activity offers CE credits for:
1. Physicians (CME)
2. Other

ACTIVITY GOAL
The goal of this article is to clarify the effects of sleep apnea on depression and vice versa.

LEARNING OBJECTIVES
At the end of this CE activity, participants should be able to:
• Discuss the relationship of obstructive sleep apnea (OSA) and depression
• Recognize OSA symptoms
• Understand the strategies for diagnosing OSA
• Describe the treatment of choice (positive airway pressure) as well as alternative treatments

TARGET AUDIENCE
This continuing medical education activity is intended for psychiatrists, psychologists, primary care physicians, physician assistants, nurse practitioners, and other health care professionals who seek to improve their care for patients with mental health disorders.

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Obstructive sleep apnea (OSA) is recognized as one of the mental disorders in the sleep-wake disorders category in DSM-5. OSA hypopnea syndrome is defined as either:
1. Evidence by polysomnography (PSG) of at least 5 obstructive apneas or hypopneas per hour of sleep and either of the following sleep symptoms/nocturnal breathing disturbances: snoring, snorting/gasping, or breathing pauses during sleep; or daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that is not better explained by another medical disorder and is not attributable to another medical condition.
2. Evidence by PSG of 15 or more obstructive apneas and/or hypopneas per hour of sleep regardless of accompanying symptoms.

The more widely used diagnostic criteria in International Classification of Sleep Disorders – Third Edition (ICSD-3) have a definition of OSA very similar to that in DSM-5.

Prevalence
The prevalence of OSA in the general population differs by age and gender. In men aged 30 to 49 years, the prevalence is 10%; in older men (aged 50 to 70), it is 17% among the general population. The prevalence in women aged 30 to 49 is 3%, and in women aged 50 to 70 it is 9%. OSA is associated with an increased risk of drowsy driving and motor vehicle accidents, hypertension, stroke, coronary artery disease, insulin resistance and type 2 diabetes mellitus, and perioperative complications. Persons with OSA have a 2- to 3-fold increased risk of all-cause mortality compared with those who do not have OSA.

Studies also show a co-occurrence of MDD and OSA with a higher prevalence of one if the other is present. A 4-year prospective study by Peppard and colleagues that included 1400 community-dwelling patients revealed a 2-fold increase in depression with mild OSA and a 2.6-fold increase in depression with moderate to severe OSA. A large national study by Wheaton and associates showed a high association of sleep apnea and an elevated Patient Health Questionnaire-9 (PHQ-9) score of more than 10 with an odds ratio of 2.4.

CASE VIGNETTE
Z is a 50-year-old woman who has had MDD for much of her adult life, with worsening of symptoms during postpartum periods. When she presents for an initial evaluation, her PHQ-9 score is high. She reports that she raised her children as a single mother, worked long hours, and functioned with very little sleep for years.

She has had no prior pharmacologic treatment for depression, and she quickly responds to an antidepressant. After the initial surge of improvement, it becomes evident that the medication helps with select symptoms, but she continues to struggle with fatigue, lack of motivation, and feeling that she has lost her resilience.

At subsequent appointments thematic symptoms are recognized, including anhedonia, hypersomnia, lack of motivation, use of sleep as a coping mechanism, poor concentration, irritability, and poor mood regulation. Further questioning about sleep reveals a decades-long history of snoring that the patient never considered pathological. Her total sleep time each night is 7 to 8 hours, but she wakes up feeling tired each morning and attributes the fatigue to her depression and unhappiness.

Comorbid OSA and MDD
The mechanism of the relationship between OSA and MDD is not well established. There are many considerations including fragmentation of sleep, hypoxia, and disruption of the sleep-wake cycle. It has also been hypothesized that depression may exacerbate or impede recovery of the neuronal injury caused by OSA, and this way of adding injury to insult may happen specifically in brain regions responsible for affect and cognition.

Other considerations include the overlap of neurotransmitters for both sleep and mood regulation, such as serotonin, dopamine, norepinephrine, and g-aminobutyric acid. Serotonin in particular is interesting given its role in depression and also the fact that it influences the strength of muscles in the upper airway that contribute to OSA and plays an important role in causing arousals from sleep. Regarding the relationship between MDD and OSA, are there confounders or a root cause for both MDD and OSA? Certainly, there are possible culprits including age, gender, obesity, hypertension, diabetes, cardiovascular disease, and cerebrovascular disease. For now, this is an interesting area
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for contemplation and research and helps guide clinical thinking.

Diagnosis
The co-occurrence of OSA and MDD can be difficult to diagnose because many symptoms overlap. While PSG remains the gold standard for diagnosis of OSA, there are clinical screening tools that can be used to determine whether a patient has an increased risk of OSA and would benefit from PSG. In a general clinic setting, formal screening tools such as STOP Bang questionnaire or the Berlin Questionnaire can be utilized. Nevertheless, the decision to use diagnostic PSG should be based on the presence of symptoms and signs suggestive of OSA (Table 1), such as snoring, insomnia, unrefreshing sleep, and excessive daytime sleepiness. There is not yet a screening tool for OSA in patients with MDD, and scales may be affected by this side-by-side nature of the 2 disorders. However, patients with comorbid depression tend to report more sleepiness, which is also evidenced by higher Epworth Sleepiness Scale scores.

Treatment
Once a patient completes PSG, reading the sleep study report can be highly informative and relaying this information to patients can be very powerful (Table 2). Patients with untreated OSA often have difficulty sleeping on their back, since OSA worsens in the supine position. They also tend to have less REM sleep because OSA significantly worsens during REM sleep. These compensatory phenomena of less supine sleep and less REM sleep can be reversed once treatment is started. The treatment of choice for OSA is positive airway pressure (PAP) therapy, including continuous positive airway pressure (CPAP), bi-level PAP (BPAP), or auto-PAP (APAP). For those who are unable to tolerate PAP therapy, alternative treatments—such as a mandibular advancement device, upper airway surgery, or hypoglossal nerve stimulation therapy—might be considered. Improvement in depressive symptoms has been reported after the treatment of OSA.

CPAP delivers a constant pressure based on a pressure titration study. The pressure keeps the airway open as a pressure column. BPAP delivers 2 different levels of pressure—one for inspiration and the other for expiration (IPAP and EPAP). With APAP the machine can automatically adjust the pressure slowly within a prescribed range based on the patient's need. The mandibular advancement device is an oral device that the patient wears inside of the mouth while asleep. This device pulls the mandible forward and opens up the airway in the back of the tongue. Hypoglossal nerve stimulation therapy uses an implanted device to stimulate cranial nerve XII (the hypoglossal nerve); the result is tongue protrusion, which opens up the airway in the back of the tongue as well.

CASE VIGNETTE CONT'D
After it was suspected that Z was struggling with both MDD and untreated OSA, a sleep study was ordered. The study confirmed the diagnosis of OSA with predominantly supine position–related OSA (Figures 1 and 2). CPAP therapy using 6 cm water pressure was recommended (Figures 3 and 4). The results showed rebound of REM sleep and complete resolution of obstructions, including those that occurred during prolonged supine REM sleep.

After 4 months of CPAP therapy, Z has had a marked improvement in her symptoms. She reports feeling refreshed in the morning and more motivated to take on the day. Her PHQ-9 score improved without further medication adjustment. It is clear that just as OSA can worsen symptoms of fatigue, treatment of OSA can ameliorate fatigue, lack of motivation, and anhedonia.

Conclusion
Untreated OSA has well-known adverse outcomes, including a doubled risk of a motor vehicle accident; an increase in morbidity and mortality, in part through increased metabolic syndrome; and cardiovascular disease. The implications of comorbid MDD and OSA are far-reaching. It would be helpful to know whether untreated OSA contributes to resistant MDD or antidepressant treatment failure. It would also be helpful to know whether patients with untreated OSA are less able to adhere to MDD treatment, including clinic follow-ups, therapy engagement, and regular use of medications. Because cardiovascular morbidity is higher for patients with OSA and MDD, it would be useful to know whether these 2 risk factors are synergistic. The questions and opportunities for further research are endless. Given these possibilities, it is important to appreciate the relationship between MDD and OSA.
CME POST-TEST

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Table 1 – Common symptoms/signs of OSA in patients with MDD

Table 2 – Important information from a diagnostic PSG report

Figure 1. Histogram of a diagnostic polysomnography of the case patient...

Figure 2. Example of obstructive events during N2 sleep while in supine...

Figure 3. Histogram of CPAP titration PSG of the same patient

Figure 4. CPAP recording during supine REM sleep

Disclosures:

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Obstructive Sleep Apnea and Depression: Issues for Psychiatrists

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References:


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