When Treating Insomnia, Consider Comorbidities

BY HEIDI SPLETE
Senior Writer

WASHINGTON — Insomnia is a disorder of hyperarousal rather than one of sleep deprivation, Thomas Roth, Ph.D., said at the annual meeting of the American Academy of Clinical Psychiatrists.

"Mothers of newborn babies don't have insomnia; they simply don't have adequate opportunities for sleep," said Dr. Roth, director of research and chief of sleep medicine at the Henry Ford Hospital in Detroit.

Because 90% of people with insomnia have other comorbid conditions, insomnia was seen as a symptom rather than an independent disorder until 2005. That's when the National Institute of Mental Health declared that insomnia met the criteria for a disorder, which include impairment in function and quality of life that is associated with specific symptoms and rooted in physiology.

To meet the diagnostic criteria for insomnia, a person must report one or more of the following symptoms: difficulty falling asleep, difficulty staying asleep, or nonrestorative sleep.

When treating a patient who complains of chronic sleep problems, be sure to ask these several key questions, Dr. Roth said in an interview:

► What is the nature of the nighttime sleep problems (difficulty falling asleep, difficulty staying asleep)?

► What is the nature of daytime consequences (daytime sleepiness, impaired function)?

► What are the frequency and duration of symptoms?

► Does the patient have any comorbid medical or psychiatric conditions?

Prevalence data are limited, but about 30% of the general population has some type of disturbed sleep, Dr. Roth said.

Many patients with insomnia report that the daytime impairment and distress resulting from insomnia are more frustrating for them than their difficulty sleeping at night.

Chronic pain is a common comorbidity in insomnia patients. In addition, people with insomnia are significantly more likely to develop comorbid psychiatric disorders.

Dr. Roth cited a recent study from his laboratory in which the researchers evaluated 1,000 people who had never had a psychiatric disease and found that 240 met criteria for insomnia. At a follow-up 3.5 years later, the people with insomnia had 4.5 times the risk of developing a psychiatric disorder.

Treatment of insomnia remains a challenge, but recognition of the role of hyperarousal and the frequency of comorbidities allows for new therapeutic targets, including some sedating antidepressants. ■

Serum Testosterone Could Be Marker for OSA

CARMEL, CALIF. — If preliminary results from an ongoing study are accurate, low baseline total serum testosterone levels could be a marker for obstructive sleep apnea in older men, Yao Schmidt reported at the Western regional meeting of the American Federation for Medical Research.

The issue is important because 20%-60% of men aged 60-80 years have borderline hypogonadism.

MS. SCHMIDT

The issue is important because 20%-60% of men aged 60-80 years have borderline hypogonadism, said Ms. Schmidt, who is a second-year medical student at the University of Colorado Health Sciences Center, Denver.

As part of a larger, ongoing study on the effects of exercise on the elderly, she and her associates at the university's Center on Aging evaluated 28 men aged 60-80 years. They recorded each man's apnea-hypopnea index (AHI), baseline total serum testosterone level, age, body mass index (BMI), neck size, and LDL cholesterol level.

The mean age of the men in both groups was 67 years, mean BMI was 29 kg/m^2, mean serum testosterone level was 288 ng/dL, mean neck diameter was 16 inches, and mean LDL cholesterol level was 103 mg/dL. The researchers then divided the patients into two groups of 14 with obstructive sleep apnea. The mean baseline serum testosterone level in the men with obstructive sleep apnea was 262 ng/dL, compared with a mean of 315 ng/dL in the men who did not have obstructive sleep apnea, a difference that was statistically significant.

A total of 180 men are expected to enroll in the study through the end of 2009.

—Doug Brunk

EVIDENCE-BASED PSYCHIATRIC MEDICINE

Obesity and Obstructive Sleep Apnea

The Question

Last month, we examined the neurocognitive effects of obstructive sleep apnea in a patient with an elevated body mass index that was not to the result of muscle hypertrophy. What does the evidence show regarding the link between obesity and OSA?

The Analysis

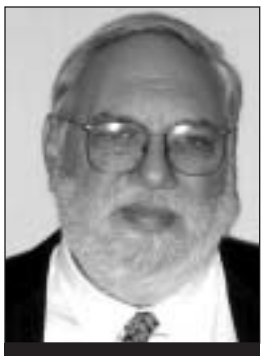
A search of the Cochrane Database of Systematic Reviews (www.cochrane.org/reviews) uncovered no review articles on this topic. We then performed a Medline search combining "sleep apnea" and "risk factors."

The Evidence

Three relevant review articles were found: CMAJ 2006;174:1293-9; Int. J. Clin. Pract. 2004;58:573-80; and Curr. Opin. Pulm. Med. 2000;6:471-8.

Being overweight or obese is defined as having a body mass index (kg/m^2) greater than 25 or 30, respectively. However, using BMI alone to determine obesity may underestimate or overestimate health risks in certain adults, such as those who are highly muscular bodies.

BY JAN LEARD-HANSSON, M.D.



BY LAURENCE GUTTMACHER, M.D.

We also looked at several individual studies. In one, Italian investigators performed a controlled trial examining predictors of OSA (Int. J. Obes. Relat. Metab. Disord. 2001;25:669-75).

A total of 161 obese patients (104 women and 57 men with a mean BMI of 43.4) were enrolled with 40 control subjects (25 women and 15 men with a BMI less than 27). After polysomnography, subjects were divided into three categories: no apnea, moderate sleep apnea, and severe sleep apnea.

Moderate sleep apnea was seen in 26% of the obese subjects, and severe sleep apnea was seen in 25%. Most of the subjects with moderate to severe sleep apnea had BMIs greater than 40. Stepwise multiple regression analysis showed that neck circumference in men and BMI in women were the strongest predictors of sleep apnea. In men, BMI correlated with sleep apnea to a lesser extent.

In a similar study conducted several years earlier, researchers at Pennsylvania State University, Hershey, recruited 200 women and 50 men with a mean BMI of 45.3 and 128 controls (BMIs not given) who were matched for age and sex (Arch. Intern. Med. 1994;154:1705-11).

The frequency of severe sleep apnea in morbidly obese patients (BMI greater than 39) was about twice as high as the frequency in severely obese patients (BMI 35-39) for both men and women (50% vs. 20% for men; 3.5% vs. 2.4% for women).

Belgian investigators evaluated the effect of continuous positive airway pressure (CPAP) and weight loss on the severity of sleep apnea in 95 patients with a baseline apnea-hypopnea index (AHI) of 10 per hour (Chest 1996;109:138-43).

Of the 95 patients originally enrolled, 39 were compliant with CPAP and thus remained in the study. Average weight loss was 9 kg, excluding three patients who underwent gastroplasty during the study. A significant improvement was found in terms of reducing the AHI and the duration of each episode.

The drop in number of apneic episodes correlated with the reduction in BMI. (AHI is an index of sleep apnea severity that is calculated by dividing the number of apnea and hypopnea events by the number of hours of sleep.)

In a prospective 4-year study conducted by investigators at the University of Wisconsin, Madison, 690 subjects with a baseline BMI of 29 (56% male) were evaluated to determine the effect of weight change on sleep-disordered breathing (JAMA 2000;284:3015-21).

A 10% weight gain predicted an approximate 32% increase in the AHI, whereas a 10% drop in weight predicted a 26% decrease in the AHI. A 10% increase in weight also predicted a sixfold increase in the odds of developing moderate to severe OSA, which was defined as an AHI of 15 per hour.

Sleep-disordered breathing was also examined in a group of 52 National Football League players (N. Engl. J. Med. 2003;348:367-8). The authors of this letter to the editor estimated a league prevalence of 14% overall—remember, last month we noted that the prevalence is 2%-3% in the general population—with 85% of cases occurring in offensive and defensive linemen, who also had the largest neck circumferences (average of 19.1 inches) and highest BMIs (average 36.6).

The Conclusion

Obesity alone is not sufficient to cause OSA, but it is one risk factor.

The data in this area are limited by several factors, including study size, but we can draw the following conclusions: For patients with elevated BMIs (without muscle hypertrophy) of 30, a further increase in body fat raises the risk of developing OSA; those with severe obesity (BMI of 35) have about a 25% chance of having OSA; those with morbid obesity (BMI greater than 40) have about a 50% chance of having OSA; and those with BMIs greater than 50 are almost assured of having OSA. ■

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