Obstructive Sleep Apnea And Obesity

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INTRODUCTION

Obstructive sleep apnea OSA is characterized by repetitive airflow reduction caused by collapse of the upper airway during sleep. OSA alone afflicts almost 20 million Americans, yet it is under-recognized and under-diagnosed partly because it is often not self-reported.[1] OSA is one of the commonest causes of excessive day time sleepiness (EDS), occurring in approximately 4% of middle-aged men and 2% of middle-aged women.[2] OSA has been associated with cardiovascular co-morbidity and increased mortality, and recent studies report a 60% survival at 15 years for severe OSA, compared with 83% in the comparable general population.[3]

RISK FACTORS

OBESITY

Obesity is the strongest risk factor for the development of OSA. There is a dose-dependent relationship, with higher BMI indicating higher risk. A moderate to severe OSA category, defined by an Apnea Hypopnea Index (AHI) ≥ 15, was independently associated with increased BMI, neck circumference and waist circumference.[4] Recently, patients with Diabetes Mellitus Type 2 and higher BMI were shown to be more likely to have severe OSA.[5] The investigators concluded that those with obesity and DM should be evaluated for OSA even in the absence of symptoms. Increased neck circumference and increased waist to hip ratio are also markers of OSA.[6] Increases in weight over a finite period of time were associated with increased rates of development of moderate OSA (AHI ≥ 15).[7] Men with a 10 kg weight gain had a 5 fold increase in the incidence of OSA, and women with comparable weight gains had a 2.5 fold increase. A BMI of greater than 25 kg/m2 has been reported in about 60 % of patients with moderate to severe OSA.[15] However, obesity does not explain the entire picture, as there are also genetic and anatomical influences.

Prevalence of OSA depends on its polysomnography definition. In general, the Apnea – Hypopnea Index can vary depending on how hypopnea is defined. The accepted definitions include a hypopnea episode as a 50% drop from baseline airflow with 3% or 4% desaturation and/or an arousal. Variability occurs in the use of either 3% or 4% desaturation, at the discretion of the individual lab or sleep center. The prevalence tends to increase with age, and reaches a plateau beyond the sixth decade for moderate to severe sleep apnea.[8] Some researchers have found 2 to 3 fold increases in the incidence of OSA in the 65 and older age group.[2] Mortality differences in the 60+ age group are still being debated. Among the <35 age group there is about a 2 fold higher prevalence among African Americans than Caucasians, independent of weight.[15] In contrast, Asians have a prevalence similar to that of Americans despite lower overall BMIs, which suggests these variations are related to differences in craniofacial structure.[9] Genetic inheritance of obesity or craniofacial abnormalities seems to cluster with some other genetic factors rather than a single genetic mutation.[10]

PATHPHYSIOLOGY/CONSEQUENCES

In the waking state, the tendency of the upper airway to collapse with each negative-pressure inspiration is balanced by the outward forces of the pharyngeal dilator muscles driven by the central and possibly autonomic nervous systems.[10,11] In patients with obesity and OSA, especially while sleeping, the forces that maintain airway patency are overwhelmed because of obesity, anatomical factors such as the dimensions of the soft palate, tongue, tonsils and mandible, and possibly abnormal contraction of the pharyngeal muscle dilator muscles as a result of altered central control.[12] Obesity also affects this mechanism through increased deposition of para-pharyngeal fat and reduced caudal traction as a result of reduced FRC (Functional residual capacity).[13]

The pathophysiologic effect of repetitive collapse of the upper airway includes release of catecholamines. Increased respiratory effort with hypoxia and snoring are commonly associated at the end of the apnea. Both effort and hypoxia seem to play a role in catecholamine release, but which of the two mechanisms predominates is still being investigated. The effect on the autonomic nervous system that results from catecholamines is likely a major factor in the development of hypertension and cardiovascular complications.

Recurrent episodes of apnea lead to sleep fragmentation and subsequent reductions in both SWS (Slow Wave Sleep) and REM (Rapid Eye Movement) sleep.[14] These reductions are likely responsible for profound EDS in a considerable number of patients. The impact of reductions in SWS and REM are evident after CPAP therapy, in which "rebounds" of both SWS and REM occur, indicating the beginning of sleep debt recovery. The ramifications of OSA extend beyond the sleepiness that it causes and include poor neuro-cognitive performance over months to years,[15] impaired work performance, decreases in health related quality of life, increased risk of automobile accidents, and impaired vigilance.[16] All of these consequences highlight the need for better recognition and earlier treatment of OSA.

Mood disturbances, personality changes, anxiety, and depression have all been associated with OSA. CPAP treatment has been reported to improve the high rates of symptoms related to depression.[17] In depressed patients who were evaluated without regard to OSA symptoms, air flow-limiting events with de-saturation were found with a higher frequency. The authors suggested that hypoxic effects on the pre-frontal cortex may pre-dispose to mood disorders.[18] The high prevalence of obesity in conjunction with mood disturbances in this population underscores the need to look for mood disturbances when evaluating for OSA.[19] More importantly, aggressive treatment of OSA can significantly improve mood and possibly eliminate the need for anti-depressants.

CLINICAL MANIFESTATIONS

The most common symptoms include excessive daytime sleepiness (EDS), snoring, and apnea (cessation of airflow that lasts at least 10 seconds). Although OSA typically is associated with EDS a number of patients are deemed "non-sleepy" phenotypes and in many cases present with insomnia, fatigue and loss of energy. As noted earlier, systemic hypertension and pulmonary hypertension are more common in OSA, as are motor vehicle accidents. Nocturnal cardiac deaths are also more common[20] as are peri-operative complications due to difficult intubation and impaired arousal from sedatives.[21] Published results of our experience at LGH concluded that those morbidly obese (weight ≥ 299 Lb) and those with severe OSA (AHI ≥30) were identified as high risk by a scoring system. The complications were lower than expected because of monitoring guidelines.[22]

The most important risk factors associated with OSA include obesity, which is present in about 60% of those referred for sleep evaluation.[23] A pattern of central as opposed to peripheral adipose deposition is considered typical, but in practice both patterns are seen. Neck and waist circumference are both markers of central obesity and both are associated with increased risk.[24] These measurements seem to correlate with severity in a "dose-dependent" manner.

The severity of OSA is determined by symptoms, which are hard to measure, and by parameters measured by nocturnal polysomnography, which have been categorized as follows by the Apnea Hypopnea Index (AHI):

Mild - Passive or sedentary sleepiness with AHI 5 – 15 events per hour;

Moderate - Awareness of sleepiness symptoms at inappropriate times but without interference with daily activities; AHI 15 – 30 events per hour;

Severe - sleepiness that interferes with daily activities such as driving; with an AHI ≥ 30 events per hour.

The symptoms do not always correlate with the degree of disordered breathing during sleep but these parameters can be used as guidelines to help make patient-directed clinical decisions.

TREATMENT

Treatment options include continuous positive airway pressure (CPAP), surgical interventions, weight loss, treatment of nasal allergies, and positional therapies. Probably the most effective and most widely used method of treatment is with CPAP. Compliance rates have varied but for the most part it has been accepted as first line therapy, because it improves quality of life and cognition, and it reduces the AHI. Not all people can tolerate CPAP so alternative treatments should be explored.[25]

Weight loss, when successful, is an effective means of treating OSA but the reality is that without surgery few are able to achieve a significant and sustainable weight loss (10% or more of body weight). The American Academy of Sleep Medicine categorizes weight loss as a behavioral strategy that should be recommended for every patient whether CPAP or other treatments are being considered.

Weight loss should be considered as an option of treatment for mild and possibly moderate OSA, as it is effective in reducing sleep-disordered breathing if done in a structured program. In a recent study, intense lifestyle monitoring and intervention resulted in both a decrease in the AHI and the percentage of OSA cases after 1 year.[26] The results of this study applied to mild cases of OSA (AHI 5 – 15 events per hour). An earlier study, by Young et al, demonstrated an average reduction of 20-40% in the AHI if weight loss was in the range of 10-20 percent.[27]

Bariatric surgery is an effective means of sustainable weight loss and has been indicated in those with a BMI ≥ 35 with co-morbid conditions and in those with BMI ≥ 40.[28] It has been accepted as an adjunctive therapy for treatment of OSA,[29] and any bariatric surgery candidate should be assessed for OSA. The remission rate for OSA 2 years after bariatric surgery is about 40%. The treatment of OSA with CPAP in the pre- peri-operative, and post-operative periods is very important, and repeat CPAP trials are warranted after about 6 months.

Other forms of non-CPAP treatments include positional therapy, avoidance of sedatives and alcohol, and treatment of nasal allergies. For those who don't tolerate CPAP, and who have mild to moderate disease, dentally designed oral appliances are an effective alternative. A more invasive surgical approach that involve resection of palatal soft tissue, known as uvulopalatopharyngioplasty (UPPP), is a good treatment for snoring but not OSA, as it is generally associated with a high rate of recurrence. Surgical reconstruction of the maxilla and mandible, and tracheotomy, are reserved for those failing all CPAP methods.

Conservative and or non-first line treatments are usually more effective in patients with mild OSA than in those with severe OSA and should be used as adjunct treatments to CPAP and surgical intervention.[30,31] Treatment should always be individualized and continued long-term follow-up with a sleep specialist is also important for effectiveness and compliance.

SUMMARY

The concerns about OSA are just part of the many complications that arise from obesity and with the obesity epidemic likely to continue, OSA will make an immense contribution to the health care burden. We should look for OSA in anyone who snores; is overweight, tired, or sleepy; suffers with mood or cognitive disturbances; and/or has risk factors for cardiac disease. Treatment is usually initiated with CPAP but weight loss and surgery are also viable options.

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