Obstructive Sleep Apnea: A Review

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Introduction

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder that involves a decrease or complete halt in airflow despite an ongoing effort to breathe. It occurs when the muscles relax during sleep, causing soft tissue in the back of the throat to collapse and block the upper airway.

This leads to partial reductions (hypopneas) and complete pauses (apneas) in breathing that last at least 10 seconds during sleep. Most pauses last between 10 and 30 seconds, but some may persist for one minute or longer. This can lead to abrupt reductions in blood oxygen saturation.A common measurement of sleep apnea is the apnea-hypopnea index (AHI). This is an average that represents the combined number of apneas and hypopneas that occur per hour of sleep.

Prevalence increases between middle and older age. OSA with resulting daytime sleepiness occurs in at least four percent of men and two percent of women. About 24 percent of men and nine percent of women have the breathing symptoms of OSA with or without daytime sleepiness. About 80 percent to 90 percent of adults with OSA remain undiagnosed. OSA occurs in about two percent of children and is most common at preschool ages[1].

OSA has three types. Mild OSA: AHI of 5-15, Moderate OSA: AHI of 15-30, Severe OSA: AHI of more than 30[1].

The risk groups include people who are overweight (Body Mass Index of 25 to 29.9) and obese (Body Mass Index of 30 and above), large neck sizes: 17 inches or more for men, 16 inches or more for women, middle-aged and older men, and post-menopausal women, abnormalities of the bony and soft tissue structure of the head and neck, down Syndrome, children with large tonsils and adenoids, family member with OSA, acromegaly and hypothyroidism, smokers, those suffering from nocturnal nasal congestion due to abnormal morphology, rhinitis[1].

Adults with OSA snore loudly and waking from sleep with a choking sensation. This abnormal sleeping pattern leads to daytime drowsiness and many other complications. Effects[1] of OSA include the fluctuating oxygen levels, increased heart rate, chronic elevation in daytime blood pressure[4], increased risk of stroke[2,3], higher rate of death due to heart disease, impaired glucose tolerance and insulin resistance[4], Impaired concentration[5], mood changes, Increased risk of being involved in a deadly motor vehicle accident[6], Disturbed sleep of the bed partner .Edwards et. al. stated the underlying pathophysiology of OSA may vary by age, with increased upper airway collapsibility being a common cause in older patients[7].The perioperative period is a time of particularly high risk for patients with OSA because of theadverse effects of anesthesia, narcotics, and sedatives on OSA. Patients with OSA undergoing cardiac surgery have a higher incidence of postoperative hypoxia, respiratory failure, and cardiac events.

Pediatric OSA presents different symptoms than OSA in adults and is defined differently. The most common cause of OSA in children is adenotonsillar hypertrophy,and adenotonsillectomy is the primary treatment for this condition, curing OSA in 70% of children[8,9]. OSA is also associated with obesity and chronic upper airway inflammatory disorders, including sinusitis, allergic rhinitis, and asthma[10].In children OSA often presents as learning difficulties, behavioral problems, and hyper activity[11].In severe cases hypertension, cardiac dysfunction, and failure to thrive may be seen[12]. Breathing is primarily controlled by central respiratory pacemakers in the medulla that interact with central and peripheral chemoreceptors.

Four tools validated for screening for OSA in surgical patients are the Berlin questionnaire , the ASA checklist, the STOP questionnaire, and the STOP-Bang questionnaire[13].Although the STOP questionnaire is the easiest to use, the STOP-Bang modification improves the sensitivity and negative predictive value of the STOP questionnaire American Academy of Pediatrics recommends that all children and adolescents be routinely screened for snoring at routine office visits, and should have a polysomnogram[14]. A pediatric screening questionnaire called I'M SLEEPY[15]. A score more than 3 is sensitive for adiagnosis of OSA.

Treatments

Sleep apnea is first diagnosed at a sleep center or lab during an overnight sleep study or polysomogram. The study charts vital signs such as brain waves, heart beat and breathing. The treatments include CPAP, oral appliances, surgery, behavioral changes and position therapy.

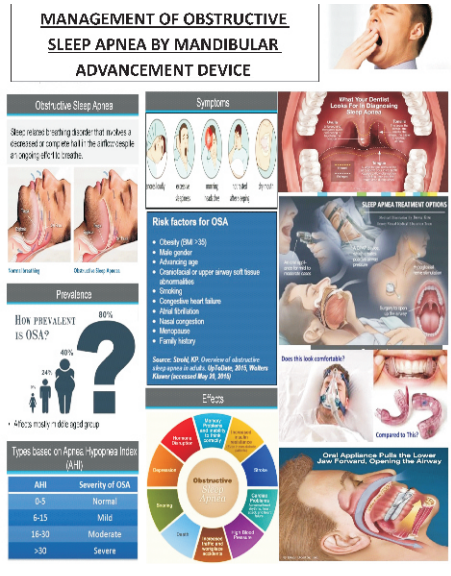
Continuous Positive Air Pressure (CPAP)

CPAP is the standard treatment option for moderate to severe cases of OSA. First introduced for the treatment of sleep apnea in 1981, CPAP provides a steady stream of pressurized air to patients through a mask that they wear during sleep. This airflow keeps the airway open, preventing pauses in breathing and restoring normal oxygen levels. While the efficacy of CPAP is reliant on patient adherence, non-adherence rates ranging from 29 to 83% are reported, with many patients rejecting therapy within the first months after initiation. Issues with mask discomfort, nasal dryness or congestion, and difficulty adapting to the pressure have been identified as barriers to patient compliance in CPAP therapy[16, 17].

Oral Appliance Therapy

An effective treatment option for people with mild to moderate OSA. and a feasible alternative for patients intolerant to CPAP treatment[16,18].Among these devices, the mandibular advancement device. (MAD) is the most commonly used and studied appliance. MADs cover the upper and lower dentition and maintain the mandible in a protruded position during sleep. By enlarging the upper airway and reducing upper airway collapsibility (e.g., by improving upper airway muscle tone), these devices help in sustaining upper airway patency[19,20]. In awake OSA patients, MAD insertion significantly increases electromyogram(EMG) activity of the genioglossus, geniohyoid, and masseter muscles.Due to a lack of dentition to support and retain a MAD, these devices are considered unsuitable for edentulous OSA patients. When MAD treatment is initiated, possible side effects that could hamper compliance include excessive salivation and poor retention on the short term and TMJ pain and my of acial discomfort on the long term[21].

Mandibular repositioning appliance (MRA) has the possibility of titration causing mandibular advancement. Adjustable MRA appears to be more effective than mono block or single arch positioning device, which does not allow titration[22]. Number of studies have proven the efficacy of MRA use in decreasing apnea-hypopnea index (AHI), increasing oxy-hemoglobin saturation (SAO2) during sleep, reducing blood pressure and improving heart rate variability[23-27]. According to American Academy of Sleep Medicine, MRA use is the first treatment option for snoring, upper airway resistance syndrome, and mild to moderate OSA, as well as the second option when patients with severe OSA refuse CPAP therapy[28].



Fixed Oral appliances are typically set to advance the mandible between 50%and 80% of its maximal protrusion and fabricated in a permanent position for therapeutic use. Fixed Oral appliances offer the advantages of lower cost, greater ease to obtain and fit, and a decrease in time to therapy compared to adjustable Oral appliances. However, because fixed oral appliances cannot be titrated, further adjustments cannot be made once the device is set. This may limit the degree of mandibular advancement/protrusion oranterior displacement of the oropharynx, which may lead to inadequatere solution of obstructive events in patients with OSA[22].

Tongue retaining/reposition devices (TRDs) do not require support from dentition and can be used as an alternative in patients lacking sufficient dentition. TRDs use suction forces to hold the tongue in a forward position without mandibular repositioning[29]. Deane et al. investigated the efficacy of TRDs compared to MAD therapy in a randomized controlled trial and reported a similar effectiveness for both devices in terms of AHI reduction. However, side effects including soft tissue irritation, dryness of mouth, and excessive salivation were reported in the majority of patients treated with a TRD,with MAD therapy showing a substantial higher compliance and patient preference[30]. The use of dental implants has been proposed to improve retention, as poor device retention remains a majorobstacle of oral appliance treatment in edentulous patients[31].A different intraoral approach was suggested by deCarlos et al.[32], involving the use of ortho-implants as an alternative to patients intolerant to CPAP and lacking sufficient dentition.

Surgical Treatment

Several soft tissues surgical procedures like uvulopalatopharyngoplasty (UPPP) and radio frequency ablation(RFA) are available for OSA patients, although accompanied by varying success rates[33, 34]In patients with severe OSA intolerant to CPAP or in whom oral devices have been found ineffective, maxillo mandibular advancement (MMA) surgery has favorable efficacy regarding surgical treatment options[35]. No studies could be identified reporting results of soft tissue surgical procedures in edentate patients. Limited evidence is available regarding treatment and morbidity outcomes of MMA surgery in the edentate patient.

Discussion

10% of weight gain is associated with a 6-fold increase in risk of OSA. Increased body weight over time increases the risk for OSA and accelerates its progression. Plasma ghrelin levels (appetite or "hunger hormone") are significantly higher in OSA patients than in BMI-matched controls, but decrease to levels similar to those of obese patients without OSA after Continuous Positive Airway Pressure (CPAP) treatment. Weight loss improves OSA by several mechanisms, including reduction in fatty tissue in the throat (i.e. parapharyngeal fat) and the tongue. Loss of abdominal fat increases mediastinal traction on the upper airway making it less likely to collapse during sleep. Importantly, weight loss benefits commonly associated co-morbidities in obese OSA patients such as hypertension and insulin resistance. Studies have also shown that compliance with CPAP treatment improves leptin (hunger inhibitory hormone) imbalance. The National Institutes of Health (NIH) and the Canadian Task Force on Preventive Health Care recommend the use of BMI and waist circumference to screen adults for obesity. The associated risks can be prevented by starting the conversation about weight loss as part of education on OSA pathophysiology and treatment during the first visit once OSA is diagnosed. Do not delay CPAP initiation or other treatments in patients with OSA. Identify patients with co-morbidities such as hypertension that can further benefit from weight loss. Weight-loss and weight-maintenance therapies should include a reduced-calorie diet, increased physical activity, and behavioral therapy. Consider the use of weight-loss medications as part of a multi component program in patients with a BMI greater than 27 kg/m^2 and comorbid medical conditions. Consider using structured methods of follow up, such as the "5 A's" (assess, advice, agree, assist, and arrange).While denture wearing may help restore the natural anatomy in edentulous OSA patients, the effect of overnight denture wearing on OSA remains controversial.

Various treatment options have been proposed to treat OSA, ranging from oral appliance therapy to surgical interventions[36]. Morphological alterations in the edentate patient however may hamper the outcome of or compliance to these interventions when applied in edentulous patients.By addressing the major concern of poor retention due to insufficient dentition, implant-retained MADs appear to be a promising treatment option in patients with mild to moderate OSA. Excessive pressure of the MAD on the maxillary alveolar ridge however was related to wearing discomfort and reduced maximal mandibular advancement. Hoekema et al., these complaints were resolved by the placement of maxillary dental implants[31]. The increased rate of alveolar bone resorption in edentate patients however should be taken into consideration, as this limits the ability of maxillary implant placement and retention[37]. Similar concerns are present in surgical treatment with ortho implants[32]as increased bone resorption could compromise treatment success due to implant loss.

Oral Appliances offer advantages over CPAP in that they do not require a source of electricity and are less cumbersome, especially with travel. Adjustable oral appliances are superior over fixed Oral Appliances in their ability to reduce the AHI among a large cohort of patients with a wide range of OSA severity. The ability to titrate OAs and allow progressive advancements in mandibular protrusion increase the efficacy of Oral appliances. Adjustable devices offer the advantage of being able to produce this advancement without having to recreate or reset thedevice. Fixed Oral appliances offer advantages over adjustable OAs. They are typically less expensive, do not require a period of adjustment, and offer treatment sooner than adjustable devices. However, despite these advantageous features, fixed OAs may be less efficacious.

Conclusion

It should be noted that OSA patients in general have an increased risk of postoperative complications including postoperative desaturation, respiratory failure, postoperative cardiac events, and ICU transfers. These general risks should be taken into account when considering surgical treatment, especially in the treatment of elderly patients as they are prone to increased risks of postoperative complications. Unfortunately, there is a lack of clinical trials assessing the application of OSA therapies in edentulous patients. In order to improve clinical management, future studies in the form of randomized controlled trials (RCT) that assess the potential of implant-retained MAD therapy and other inventions are crucial. Adequate treatment is of key importance in an effort to reduce potentially serious complications and improve both the overall health and quality of life of these usually elderly patients. Literature review presented in this paper demonstrates the deficiency in the effective evidence based therapeutic modalities for edentulous OSA patients and thus require further clinical studies to improve clinical management.

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More References are available on request at editor@healtalkht.com.