REVIEW ARTICLE

RISK FACTORS AND TREATMENT MODALITIES FOR MANAGEMENT OF OBSTRUCTIVE SLEEP APNEA

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ABSTRACT

Obstructive sleep apnea is characterized by repeated airway collapse during sleep. Gold standard for diagnosis of OSA is using Polysomnography (PSG). Routine clinical examination of anthropometric and radiographic variables helps to identify the high risk subjects for sleep apnea. Among the sleep medicine team, the role of dentist has become more significant especially in managing the patients with mild to moderate OSA. Treatment method should be based on the severity of the condition, etiology and patient preferences. This review focuses on the risk factors associated with OSA and different treatment methods for managing these patients.

Key words: Obstructive sleep apnea, Behavioral modifications, Rapid maxillary expansion.

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INTRODUCTION

Obstructive sleep apnea (OSA) is a highly prevalent condition resulting in increased collapsibility of upper airway during sleep, leading to reduced or absolute cessation of airflow. Recurrent periods of complete or limited pharyngeal obstruction causing nocturnal hypoxemia, and excessive daytime sleepiness. Symptoms include snoring, witnessed apnea, excessive daytime sleepiness, morning headache, restless sleep, insomnia, nightmares, irritability, memory loss, decreased attention and concentration, performance deficiencies. OSA thus leads to functional impairment, decreased quality of life and increased risk for road traffic accidents.

According to the International Classification of Sleep Disorders[1], alternate names include OSA syndrome, sleep apnea, sleep apnea syndrome, obstructive apnea, sleep-disordered breathing (SDB), sleep hypopnea syndrome and upper airway obstruction.

The gold- standard in diagnosing OSA is by polysomnography (PSG)[2]. Severity of OSA is determined by Apnea Hypopnea Index (AHI), which measures the average number of apneas and hypopneas per hour of recorded sleep.

This review focuses on the risk factors associated with OSA, diagnosis and treatment modalities.

Types of Sleep apnea

The types of sleep apnea are central, obstructive and mixed[3]:

- Central sleep apnea is the condition in which the neural drive to all respiratory muscles are abolished resulting in apnea
- Obstructive sleep apnea is the condition in which there is an occlusion of the oropharyngeal airway for more than ten seconds during sleep
- Mixed sleep apnea is a combination of both component.

OSA can occur at one or more "levels" of the nasopharyngo-tracheal airway[4].

- Type I disease involves narrowing or collapse of the retropalatal region.
- Type II disease involves narrowing or collapse of both the retropalatal and retrolingual areas.
- Type III disease involves collapse in the retrolingual area (tongue base).

EPIDEMIOLOGY

OSA is highly prevalent disorder. Many previous population studies conducted globally reported 0.3-5.1%prevalence for OSA. However, these data are predominantly from Caucasian population and so may not be valid to others. A population based study in Delhi, India reported 2.4-4.96 % of prevalence of OSA among men and 1-2% among women[5]. Men are more predisposed to OSA than women[6]. The male-to-female ratio for prevalence of OSA among general population ranges between 2:1 and 4:1.

RISK FACTORS

Obesity is the main risk factor for the development and progression of OSA and different parameters such as altered body mass index (BMI), neck circumference, waist circumference and waist to hip ratio (WHR) are all considered as risk factors for OSA[7]. However, this relationship appears to be varied by social, environmental and different ethnic backgrounds. Hence, there is an increased attention in identification of the role of these risk factors which leads to sleep disorders so that useful interventions can be used to reduce the obligation of these conditions.

Peppard et al[8] found that 10% increase in body weight caused six times higher risk for developing OSA. Smith et al also have distinctly shown that a high BMI was an important risk factor for OSA, and longitudinal studies have shown that weight loss significantly improves the AHI in obese individuals, suggesting that obesity plays a causative role.

OSA is marked by recurrent collapses of the upper airway during sleep that occur due to reduced airway dilator muscle tone. Obesity may alter the normal upper airway mechanics and contribute to the development of OSA[10]. Fat deposition around neck region can result in change in shape of the upper airway promoting collapsibility[11]. Neck circumference measured

at the level of cricothyroid membrane also forms another parameter for determining obesity[12]. Svennson et al[13] based on a population study showed that increased neck circumference was associated with increased risk of snoring. Central obesity was estimated using waist circumference and waist hip ratio, Suwanprathes et al[14] identified that greater waist circumference increases the risk for developing OSA.

Craniofacial and upper airway morphology is also increasingly accepted as an important interacting factor in OSA pathogenesis[15]. The most frequently reported abnormalities include, a posteriorly placed maxilla and mandible, an enlarged tongue and soft palate, an inferiorly positioned hyoid bone and a reduced velopharyngeal cross-sectional area[16]. In children, enlarged tonsils and adenoids causes abnormal facial growth pattern (adenoid faces) leading to increased risk for developing OSA.[7]

Smoking and alocohol consumption are also possible risk factors for OSA but only few studies have been reported. Wetter et al[18] showed that smoking causes three times more risk for OSA than non-smokers. Nagayoshi et al[19] also supported that alcohol consumption, and cigarette smoking were positively associated with habitual snoring for both men and women.

Comorbid conditions associated with OSA include hypertension, diabetes, coronary heart disease, congestive heart failure and stroke[20].

DIAGNOSIS

Berlin questionnaire helps to predict sleep apnea and has a sensitivity of 86% in the detection of OSA. Excessive Daytime sleepiness is evaluated using Epworth Sleepiness Scale (ESS)(21). This is a simple questionnaire measuring the general level of daytime sleepiness, called the average sleep propensity. This measures the probability of falling asleep in a variety of situations. ESS scores distinguished patients with primary snoring from those with obstructive sleep apnea syndrome (OSAS), and

EPWORTH SLEEPINESS SCALE

Chance of dozing

0 - Would never doze

1 – Slight chance of dozing

2 – Moderate chance of dozing

3 – High chance of dozing

Situations

1. Sitting + Reading ☐
2. Watching TV ☐
3. Sitting inactive in a public place (eg. Theatre or a meeting) ☐
4. As a passenger in a car for an hour without having a break ☐
5. Lying down to rest in the afternoon ☐
6. Sitting + Talking to someone ☐
7. Sitting quietly after lunch when you have had no alcohol ☐
8. In car, while stopped in a traffic ☐

Figure 1- Epworth sleepiness scale

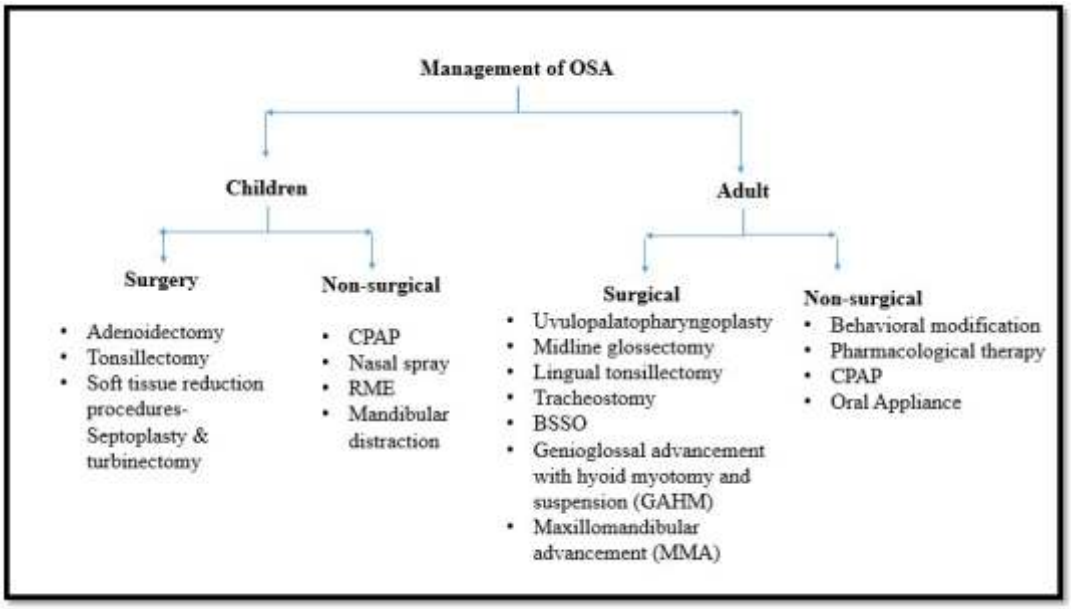

Figure2- flowchart

Figure 3-RME - before expansion

Figure 4- RME- after expansion

ESS scores increased with the severity of OSAS. (fig 1)

The most widely used technique is polysomnography (PSG), which monitors the sleeping state, respiration, electrocardiogram, move¬ments of the legs, oximetry and snoring. In addition, PSG records the distribution of the stages of sleep, the number of awakenings, the number of apneas or hypopneas, the starting time of sleep, and the hours of efficient sleep(hours asleep/hours in bed). PSG also provides the apnea / hypopnea index (AHI)

AHI >30 -severe OSA,

AHI 15-30 - moderate OSA

AHI < 15 - mild OSA

The various anthropometric measurements of obesity, like BMI, waist and neck circumference, can be used as clinical predictors in routine clinical examination for suspecting high risk candidates for OSA. Radiographic examination of upper airway and craniofacial features can be evaluated using lateral cephalograms (2D), computed tomography (CT), cone beam computed tomography, Magnetic resonance imaging (MRI).

Thus during routine clinical examination, these anthropometric as well as radiographic variables help us to identify the high risk subjects for sleep apnea.

TREATMENT

Based on the severity of sleep apnea, the sleep medicine team describes the different treatment options to patients with OSA.(fig 2) summaries the It includes behavioral modifications, Continuous positive airway pressure (CPAP) and surgical option for moderate to severe OSA cases, Oral Appliance for mild to moderate cases and for patients who are non-compliant to CPAP or refuses surgery.

In children with OSA, Rapid maxillary expansion (RME) is considered as an effective treatment method (fig 3& 4). According to American Academy of Sleep medicine, the PSG diagnosis of OSA in children is defined as apnea hypopnea index AHI>1/h. Clinically these patients have long and narrow face, constricted maxilla, enlarged tonsils, narrow upper airway and retrognathic mandible leading to unfavourable facial growth and dental malocclusion. RME helps in the expansion of maxilla by splitting midpalatal suture leading to the correction of posterior crossbite and nasal stenosis[28]. Asok et al showed an improvement in sleep parameters with an increase in sleep efficiency, decreased in arousal and desaturation index after expansion in children[29].

Among adults,

A. Behavioural Modification

- Weight loss
- Positional therapy
- Avoid smoking and alcohol

B. Continuous Positive airway pressure

The ideal treatment of OSA should be capable of reducing excessive daytime sleepiness by normalizing the breathing pattern during sleep. The first line choice of treatment is CPAP. It is often associated with numerous problems like nasal congestion, discomfort secondary to air leak and pressure sensation, mask intolerance, claustrophobia, and issues relating to prolonged use in younger and less severe patients[22].

C. Oral Appliance (OA)

In 1990, mandibular advancing oral appliance was the most popular treatment for OSA. Clinically, OA is considered as an effective and low risk alternative for CPAP. According to American Academy of Sleep Medicine (AAOSM), OA is recommended for patients with mild to moderate OSA and also to patients who are intolerant to CPAP and refuses surgery[23]. OA helps to advance the mandible, thus, modifying the posture and their by enlarging the airway leading to reduced upper airway collapsibility.

Numerous designs are available which include

- Mandibular advancing device
- tongue protrusion devices
- tongue repositioning or retaining devices
- soft-palate lifters, tongue trainers
- combination of OA and CPAP(24)

Figure 5- Custom made Oral appliance

Custom made OA (fig 5) are fabricated using thermoplastic material in the vaccum pressure molding device. Posterior bite blocks are made similar to twin block on the upper and lower thermoplastic material. In the lower

incisal lingual area ball end hooks are placed that engages into the acrylic trough in the upper anterior palatal region while closing. Hooks are also placed in the lateral side on to which interarch elastics are engaged which prevents mouth opening during sleep. Various studies have evaluated the efficiency of OA. Cilil et al showed that the custom made OA showed significant improvement in sleep parameters using PSG indicating reduced upper airway collapsibilty.[25] Bonham et al also advocated an increase in velopharyngeal area following the use of OA.[26]

D. Surgical Method

- Uvulopalatopharyngoplasty
- Midline glossectomy
- Lingual tonsillectomy
- Tracheostomy
- Bilateral sagittal split mandibular ramus osteotomy
- Genioglossal advancement with hyoid myotomy and suspension (GAHM)
- Maxillomandibular advancement (MMA)

MMA increases the retropalatal and retroglossal portion of upper airway, preventing its collapse. MMA advances both maxilla and mandible forward along with its muscle attachments. Prinsell et al suggested 10-15mm advancement of maxilla for lefort I and BSSO mandible with 50% decrease in the AHI index in post-operative patients[27].

CONCLUSION

Undiagnosed OSA represents a major health problem for the public. Disturbed sleep often leads to weakened neurocognitive function, predisposes to motor vehicle and workstation accidents and poor quality of life. Nevertheless, the condition still remains mostly undiagnosed. It is very important to spread alertness not only among the general public but also to physicians of developing countries, regarding the common clinical features, risk factors and different treatment options associated with OSA.

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