Review Article

Obstructive Sleep Apnoea in Adults - The ENT Perspective

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Introduction

Obstructive sleep apnoea (OSA) is the most common type of Sleep Disordered Breathing seen in general population. It is characterized by 3 S- Snoring, Sleepiness, Significant other report of sleep apnoea episodes. In recent times it is gaining more attention from both clinician and patients because of its strong association with hypertension, cardiovascular diseases, coronary artery diseases, insulin resistance diabetes and depression.

Charles Dickens in The posthumous Papers of the Pickwick Club, published in 1837 suggested a correlation between obesity and snoring. Drs. A. G. Bicklemann and C. S. Burwell in their paper "Extreme Obesity Associated with Alveolar Hypoventilation: a Pickwickian Syndrome"[1], further supported the concept but the complete description of OSA syndrome with its adverse cardiovascular effects was given for the first time by Elio Lugaresi et al in 1970s.

Treatment of OSA has progressed from tracheostomy and weight loss to surgeries like Uvulopalatopharyngoplasty (UPPP), described by Fujita et al[2] and Simmons et al[3] but all these modalities have their own limitations with benefits at the most being only partial in most of the cases. Colin Sullivan suggested a non surgical treatment modality in the form of Continuous positive airway pressure (CPAP), which is currently the first line of treatment for this condition.

Classification

Sleep related breathing disorders range from partial airway collapse and increased airway resistance to episodes of hypopnoea and complete airway collapse with sleep apnoea.

The various sleep events are described as follows:

Apnoea is complete cessation of airflow for 10 seconds

Hypopnoea is defined as reduction in airflow (≥30%) for at least 10 seconds with a oxyhemoglobin desaturation of >=4% OR a reduction in airflow of 50% for 10 seconds with oxyhemoglobin desaturation of >=3%

Respiratory effort related arousals (RERA) refer to sleep events associated with arousals due to increased breathing efforts lasting for at least 10 seconds

Central sleep apnoea is characterized by lack of thoracoabdonimal effort associated with partial or complete cessation of airflow

Obstructive sleep apnoea is characterized by continuous thoracoabdonimal effort associated with partial or complete cessation of airflow.

Mixed type has features of both.

Snoring

Vibration of the pharyngeal soft tissues leads to snoring. It affects at least 40% of men and 20% of women and often accompanies sleep-disordered breathing (SDB)[5]. Not all patients with snoring are diagnosed to have OSA. Snoring in absence of OSA is diagnosed when habitual audible snoring occurs with an apnoea hypopnoea index (AHI) of less than five events per hour without daytime symptoms.

Upper airway resistance syndrome

Guilleminault et al first described upper resistance airway syndrome (UARS) for those patients who did not meet the criteria for OSA syndrome but had excessive daytime somnolence and other debilitating somatic complaints[6]. Some authorities don't treat UARS as a separate entity and club it with OSA because of similar pathophysiology.

Obstructive sleep apnoea syndrome

Obstructive sleep apnoea is defined by five or more respiratory events including apnoea, hypopnoea and

RERA in association with excessive daytime somnolence, waking with gasping, choking, or breath holding, or witnessed episodes apnoeas, loud snoring or both. More often than not it is the bed partner who brings the problem to notice.

Pathophysiology

Soft tissue collapse due to decreased transmural pressure at the level of nasopharynx, tongue (oropharynx) has been proposed as one of the factors for OSA. Anatomical factors like enlarged tonsils, volume of tongue, soft tissue length of soft palate, abnormal position of maxilla and mandible have also been implicated. Reduced ventilatory motor output to upper airway pharyngeal dilator muscles has been proposed to be another important factor.

Fujita et al classified[7] classified the patterns of obstruction into following types:

Type 1: collapse in retropalatal region only

Type 2: collapse in both retropalatal and retrolingual region

Type 3: collapse in retrolingual region only

Nasal obstruction though touted as a cause of OSA is rarely the sole cause, though it may contribute in worsening the symptoms. Baisch et al demonstrated in their study that surgical correction of nasal breathing led to subjective improvement in symptoms of OSA[8].

Obesity contributes in a multidirectional way to development of OSA by causing narrowing and compression of upper airway, reducing lung volume and causing a mismatch between alveolar ventilation and perfusion.

Adenotonsillar hypertrophy is a major cause of OSA affecting mainly the children. In adults it is mainly multiple structural characteristics are associated with OSA like increased distance of hyoid from mandible, decreased mandibular and maxillary projection, downward and posterior rotation of mandibular and maxillary growth, increased vertical facial length, increased vertical length of posterior airway and increased cervical angulation[9].

Diagnosis

OSA symptoms generally begin insidiously and are often present for years before the patient is referred for evaluation. It progresses through various stages of what is known as Sleep Disordered Breathing Continuum. The initial manifestation is just snoring. Untreated, it gradually progresses to upper airway resistance syndrome, which may subsequently progress to OSA[10].

Nocturnal symptoms may include the following:

- Snoring, usually loud, habitual, and bothersome to others
- Witnessed apnoeas, which often interrupt the snoring and end with a snort
- Gasping and choking sensations that arouse the patient from sleep
- Nocturia
- Insomnia
- Restless sleep, with patients often experiencing frequent arousals and tossing or turning during the night

Daytime symptoms may include the following:

- Nonrestorative sleep (i.e., "waking up as tired as when they went to bed")
- Morning headache, dry or sore throat
- Excessive daytime sleepiness (EDS) that usually begins during quiet activities (e.g., reading, watching television); as the severity worsens, patients begin to feel sleepy during activities that generally require alertness (e.g., school, work, driving).
- Daytime fatigue/tiredness
- Cognitive deficits; memory and intellectual impairment (short-term memory, concentration)
- Decreased vigilance
- Morning confusion
- Personality and mood changes, including depression and anxiety
- Sexual dysfunction, including impotence and decreased libido
- Gastroesophageal reflux
- Hypertension
- Depression

Excessive Daytime Somnolence is most frequently assessed with Epworth Sleepiness Scale (ESS). An ESS score greater than 10 is generally considered sleepy.

All patients must undergo a detailed examination including calculation of Body mass index, blood pressure measurement and neck circumference measurement along with assessment of body habitus and craniofacial proportions. This can been supplemented with fiberoptic nasopharyngoscopy in multiple positions to get an idea of the extent of airway compromise and the level of obstruction- nasal, retropalatal, or retrolingual.

The site of obstruction can be better identified in patients with OSA, if the above examinations are supplemented with drug induced sleep videoendoscopy.

Imaging modalities haven't shown much promise in delineating OSA patients from non OSA patients though cephalometric X rays, Ct scans and MRI are used frequently to assess the skeletal and soft tissue components of the airway[11, 12].

Nocturnal Polysomnography is currently the gold standard for diagnosing OSA.

Other pathologies like sinonasal polyposis, asthma, central sleep apnoea, chronic obstructive pulmonary disease, depression, gastroesophageal reflux disease,

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

Situation Chance of Dozing (0–3)

Situation	Chance of dosing (0-3)
Sitting and reading	
Watching television	
Sitting inactive in a public place (e.g. a theater or meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	
TOTAL SCORE	

Total Score - Score Results:

1-6 Congratulations, you are getting enough sleep!
7-9 your score is average
10 and up Very sleepy and should seek medical advice

hypothyroidism, narcolepsy and periodic limb movement should be ruled out before embarking on the treatment for OSA.

Treatment

The approach to treatment of an OSA patient is in a step wise manner with medical management first and surgical management reserved for later.

Medical

- Weight loss should be recommended for all overweight patients with OSA.
- Bariatric surgery can be considered when treating patients who are morbidly obese. Surgically induced weight loss significantly improves obesity-related OSA and parameters of sleep quality[13], and this improvement can occur as early as 1 month after surgery[14].
- Continuous positive airway pressure (CPAP) is considered the gold standard treatment for moderate to severe OSA. However, patient adherence remains a significant obstacle.
- Bi-level positive airway pressure (BiPAP) and autoadjusting positive airway pressure (APAP) may be used to treat patients with neuromuscular disorders and ventilatory disease.
- Oral appliances may also be used in some patients with mild, moderate, and some severe OSA
- Modafinil is a central stimulant of postsynaptic alpha1-adrenergic receptors, which acts by promoting alertness. It is used for treatment of narcolepsy and idiopathic hypersomnia. Some studies have shown it to be effective in OSA[15].
- Nasal dilator strips and topical decongestants may be used in patients who have OSA and severe nasal obstruction.

Surgical

Surgical modality to be used for treatment of OSA should always be decided after taking patient's wishes and expectations into consideration and all the patients should be counseled for the need of tracheostomy. Some of the criterias used by surgeons to decide upon the surgical modalities are failure of medical therapy, significant cardiac arrhythmias, AHI>15and oxyhemoglobin desaturation <90%.

Steinhart and colleagues evaluated 117 OSA patients and found that 100% had retropalatal obstruction and 77% had retroglossal obstruction, thus illustrating that a majority of patients have a combination of the two[16]. In 2005, den Herder and colleagues evaluated 127 patients and found that 88% of patients had retropalatal obstruction while 49% had retroglossal obstruction. In this study, 51% exclusively had palatal obstruction whereas only 12% solely had obstruction at the base of tongue[17]. These studies demonstrate that most of these patients have a multilevel problem which should be identified diligently with endoscopies and other diagnostic modalities and treatment should be planned accordingly.

Nasal surgeries like septoplasty, turbinate reduction and sinus surgeries may improve the symptoms of OSA though they rarely are curative by themselves. The one definitive advantage they do offer is a more physiological breathing after the nasal obstruction has been relieved and also improve the adherence to CPAP.

UPPP aiming to eliminate palatal obstruction by resecting redundant palatal and pharyngeal tissue was described by Fujita et al[2]. Though one of the most commonly performed surgical procedure for OSA, it

has shown a success rate of not more than 50% due to its misuse as the first line surgical therapy for OSA regardless of coexistent patient factors such as obesity, retrognathia, and the existence of other sites of obstruction[18]. Friedman et al demonstrated the value of staging OSA patients for the prediction of success for UPPP[19]. They used palate position (based on the modified Mallampati staging), tonsil size, and BMI to stratify patients.

Modified Mallampati palate position divides the view into 4 positions:

1. The entire uvula can be seen with the tongue at rest.
2. A partial view of the uvula is seen.
3. Only the soft and hard palate can be seen.
4. Only the hard palate can be seen.

Stage I patients have an 80% success rate, stage II patients have a 40% success rate, and stage III patients have only an 8% success rate.

Complications associated with UPPP include temporary nasal reflux, postoperative bleeding, infection, and rarely altered speech.

Various modifications like Woodson's transpalatal advancement pharyngoplasty[20] and Friedman's Z-palatoplasty[21] have also been described especially for patients with persistent symptoms after UPPP.

Other lesser invasive procedures like palatal implants have also been advocated by various authors to reduce the morbidity and cost of treatment of OSA[22].

The management of retrolingual narrowing requires some specific surgical procedures like partial midline glossectomy, lingualplasty, and radiofrequency tongue base ablation. Most of these procedures aim at reducing the volume of tissue at the base of tongue or producing scar formation in the area thereby resulting in effective widening of retrolingual space.

Hypopharyngeal airway narrowing is addressed by procedures like Genioglossal advancement, Hyoid myotomy. The aim of these procedures is to prevent tongue collapse in the airway.

The hyoid bone is mobilized by inferior myotomy and is fixed anteriorly and inferiorly to the thyroid cartilage. The patients who are refractory to the above mentioned procedures may be considered for maxillo-mandibular advancement.

Despite of all the advancements in the management of OSA, tracheotomy still is the gold standard treatment because it bypasses the portion of airway where the obstructive symptoms arise. But the stigma associated with tracheostomy rarely makes it a surgical option of choice, although it must be considered in patients in the severity of symptoms warrants it like morbidly obese patients or on temporary basis in patients undergoing base of tongue surgery.

Conclusion

OSA if left untreated can cause cardiovascular problems like hypertension, coronary heart disease, congestive heart failure, arrhythmias, sudden death, pulmonary hypertension and stroke. It is also known to be an independent risk factor for insulin resistance23. Further it is associated with significantly higher incidence of Gastroesophageal reflux disease (GERD)[24].

Apart from this the patients suffer from daytime somnolence, decreased attention and executive functions which may lead to serious consequences in the form of automobile and workplace accidents. Impaired mood and neurocognitive deficits have also been noted. Bed partner dissatisfaction is also a common complaint among this patient group.

Treatment of OSA with CPAP and other surgical modalities has been shown to improve quality of life both for the patient and the bed partners25. Considering all the above facts and that the treatment has a well documented positive impact on the quality of life, utmost effort should be made to diagnose, evaluate the problem and offer the appropriate treatment to such patients.

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Eat Curry to Curb Mets!
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Turmeric (Curcuma Longa; "Indian Saffron") is an important spice of curry and many other dishes of Indian and other Asian cuisine. Its medicinal value has long been recognised in Ayurveda. The beneficial effects are considered to be due to one of its active ingredients, Curcumin. Several recent studies have highlighted its anti-inflammatory property and also a possible benefit when used in Parkinsonism. Now, in a study conducted in Ludwig-Maximilians-Universität (LMU) in Munich, Germany,Dr. Beatrice Bachmeier and team showed that curcumin averts the development of metastasis in patients with prostate cancer. Curcumin is safe and upto 8 gram may be consumed daily. In mice, it appears to inhibit the expression of chemokines CXCL1 and CXCL2, powerful pro-inflammatory mediators that promote metastasis. So, its action is mainly anti-inflammatory. It may also be useful in breast cancer. However, it is not a replacement for conventional therapy. The study has been published in Carcinogenesis.

- Dr. K. Ramesh Rao