A Comprehensive Perspective on Obstructive Sleep Apnoea

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Abstract: Obstructive sleep apnoea (OSA**)** is sleep associated disorder of breathing with an episodic reduction or complete airflow obstruction despite an ongoing effort by patient for breathing. OSA seems to affect especially middle-aged and elderly men its prevalence has increased substantially over the last two decades. Few predisposing factors that affect OSA are obesity, craniofacial abnormalities, increasing age, male predilection, family history, smoking, micrognathia, enlarged adenoids and tongue. With rising awareness of OSA and the increasing prevalence of obesity, OSA is increasingly recognized as a major contributor to cardiovascular morbidity including systemic and pulmonary arterial hypertension, heart failure, acute coronary syndromes, atrial fibrillation, and other arrhythmias. Management primarily includes the use of continuous positive airway pressure (CPAP) or Bi-level positive airway pressure (BiPAP) devices during sleep. Alternate options such as mandibular devices and surgical procedures are considered for certain patient populations.

Keywords: Sleep Apnoea, Obstructive, Sleep Disorder, Airway obstruction

Introduction:

Obstructive sleep apnoea (OSA) is characterized by recurrent collapse of the pharyngeal airway during sleep, resulting in substantially reduced (hypopnea) or complete cessation (apnoea) of airflow despite ongoing breathing efforts. These disruptions to breathing lead to intermittent blood gas disturbances (hypercapnia and hypoxemia) and surges of sympathetic activation. Loud snoring is a typical feature of OSA and in most cases the culmination of a respiratory event is associated with a brief awakening from sleep (arousal). These events

result in a cyclical breathing pattern and fragmented sleep as the patient oscillates between wakefulness and sleep. In severe cases respiratory events can occur more than 100 times per hour and typically each event lasts 20–40 seconds.

OSA can be definitively diagnosed only by a physician, the orthodontist may be called on to screen for OSA, contribute to the identification of underlying dentofacial components, and assist the physician in managing the disease. A cooperative shared effort between the orthodontist and other medical professionals is preferred to optimize care of patients with OSA as orthodontic screening may reveal the need for further evaluation by a physician.[1]

Epidemiology and Prevalence:

Obstructive sleep apnoea can occur in any age group, but it prevails mainly in adults. OSA seems to affect especially middle-aged and elderly men.9 The prevalence of OSA is similar in both Caucasians and Asians, this indicates that OSA is not only common in developed but also in developing countries.[2] However, the disease prevalence is higher in the subgroups with overweight or obese subjects, elderly people and those of different ethnic origins.

Epidemiologic data have shown a strong association between untreated obstructive sleep apnoea and incident cardio and cerebrovascular morbidity and mortality.[3,4]

Predisposing Factors:

The predisposing factors that affect OSA are as follows:

- Obesity/Visceral Obesity- It is the major risk factor for the development of OSA. Increase in adiposity around the pharynx and body, predispose to upper airway obstruction during sleep.[5]
- Craniofacial Abnormalities- Differences in craniofacial morphology such as micrognathia, enlarged adenoids, retrognathia, enlarged tonsils, enlarged tongue may explain some of the variation in risk for OSA in different ethnic groups.
- Increasing age- In elderly, it appears after 65 years of age. When the prevalence is controlled for body mass index, the severity appears to decrease with age.
- Male gender- OSA is more common in men than women. This can be attributed to anatomical and functional properties of the upper airway and in the ventilatory response to the arousals from sleep.[6]
- Family history and genetic predisposition- First degree relatives of those with OSA increases the relative risk compared to those without OSA
- Smoking

- Enlarged tongue and adenoids

Pathophysiology-

OSA results from a combination of structural upper airway narrowing and abnormal upper airway neuromotor tone.[7] It is believed that the upper airways collapse more easily in OSA patients and occurs at slightly negative intra-thoracic pressures or even positive pressures.[8] There is increasing evidence that the quantity and pattern of ventilation plays a substantial role in airway collapse as well as the presence of upper airway neuropathology.[9]

Signs and Symptoms of OSA :

Snoring- Snoring is the cardinal complaint of the patients with sleep apnoea. Nearly all patients with OSA are heavy snorers and this close relationship between snoring and sleep apnoea points the need to enquire about snoring during the medical interview.

- **Excessive daytime sleepiness-** Excessive daytime sleepiness is a chief clinical consequence among patient with OSA.[10]Patients with OSA fall asleep easily under the most inappropriate circumstances: for example, while talking, eating, driving short distances, or engaging in sexual activity. Daytime sleepiness directly relates to the severity of sleep apnoea. A standard instrument, Epworth Sleepiness Scale (ESS), is a useful tool to assess the degree of self-rated sleepiness. A value between 1-6 is considered as normal, values between 7-8 is considered as subnormal and the value above 10 is considered abnormal.
- **Morning symptoms-** Morning tiredness, fatigue, and lack of refreshing sleep are other associated symptoms.
- **Restless sleep-** Agitation, restlessness, and abnormal body movements are common during episodes of obstructive apnoea.
- **Other symptoms-** Other, progressively less common, symptoms include intellectual deterioration, depression, impotence, sleep walking and enuresis.

Signs

The most common physical sign of OSA, is the excess weight. More than half of the people with obstructive sleep apnea are either overweight or obese, which is defined as a Body Mass Index (BMI) of 25-29.9 or 30 or above, respectively.[11]

Each unit increase in BMI is associated with a 14% increased risk of developing sleep apnoea and a 10% weight gain increases the odds of developing moderate or severe obstructive sleep apnoea by 6 times.

BMI is not the sole marker of obesity. Men with a neck circumference above 17 inches (43cms) and women with a neck circumference above 15 inches (38cms) also have a significantly increased risk of developing OSA.

Obesity may worsen OSA because fat deposition at specific sites. Fat deposition in the tissues surrounding the upper airway appears to result in a smaller lumen and increased collapsibility of upper airway, predisposing to apnea. Moreover, fat deposits around the thorax (Truncal Obesity) reduces chest compliance and functional residual capacity, and may increase oxygen demand. Visceral obesity is common in subjects with OSA. Factors such as reduced activity levels and increased appetite, particularly for refined carbohydrates, may contribute to weight gain in OSA patients.

Anatomical abnormalities which cause narrowing of the nasal or pharyngeal airway, such as enlarged adenoids, deviated nasal septum (DNS), nasal valve obstruction, tonsilar hypertrophy, macroglossia, and retro- or micrognathia are rare, but should be looked for during physical examination.

Diagnosis:

As per the norms set by American Association of Sleep Medecine (AASM), sleep history is taken to evaluate OSA symptoms and to determine patients who present high-risk levels. A sleep examination is directed at modifying the OSA probability based on the history, looking for associated or complicating disease, and excluding other potential causes for symptoms.

The steps for the diagnosis of OSA includes

1. Clinical Features
2. Objective testing

1. Clinical features

Excessive Daytime Sleepiness (EDS)- EDS is caused by sleep fragmentation due to frequent arousals at night. It is still a very subjective symptom that overlaps significantly with other factors such as tiredness and lethargy.[12] Sleepiness may occur during “passive” conditions, such as watching television or, in severe forms, during “active” conditions, such as verbal conversation or driving.

Objective laboratory sleep tests, like multiple sleep latency test (MSLT) or maintenance of wakefulness test (MWT) are also used for EDS assessment, but their limits are principally related to their costs and duration.

Snoring and witnessed apnoea’s with choking or gasping-The presence of snoring alone is a poor predictor of OSA but its absence does not exclude OSA. Severe snoring can

affect social relationship and become one of the main complaints of patients. Talking to the partner and family members can be very helpful; they can often report signs, such as apnoea or falling asleep unintentionally.

2. Physical Examination-

Obesity- A BMI ≥ 30 kg/m^2 and a neck circumference >17 inches in men and >16 inches in women are used as critical values.[13]

Hypertension- The finding of hypertension in a patient with symptoms suggestive of OSAS increases the likelihood of the disorder.

Craniofacial examination- Clinical examination should include anatomical features of craniofacial and oropharyngeal structures as they can compromise airway patency.

3. **Objective Testing-**

Polysomnography (PSG)- Polysomnography is the golden standard method for diagnosing OSA. It is done in the laboratory. It records sleep breathing pattern and oxygen saturation overnight via a minimum of 12 channels of physiological signal such as electroencephalogram (EEG), electrocardiogram (ECG), electromyogram (EMG) , oronasal airflow, electro-occulogram (EOG), respiratory effort, body position and oxygen saturation39. The most important data from the polysomnography is the apneahypopnea index (AHI). It represents the severity of OSA by relating the values of apneas and hypopneas. It is equal to the sum of hypopneas and apneas multiplied by sixty and divided by total sleep time in minutes. A result less than 5 corresponds to no or minimal OSA. A result equal to or greater than 5 but less than 15 is mild. An AHI greater than or equal to 15 but less than 30 is moderate. Finally, an outcome greater than or equal to 30 correlates to severe OSA.

Portable Monitors (PM)- Unlike PSG that is expensive and labor intensive, PM is performed at home and thus offers greater convenience for patients. PM may be used as an alternative to PSG for diagnosis of OSA.

Oximetry- Another method to evaluate suspected OSA is the continuous recording of saturated oxygen (SaO2) during sleep, and it is often sufficient in severe cases because of the characteristic pattern of repetitive desaturation.

Treatment of OSA–

A variety of different treatment modalities has been suggested, including cardiac pacemakers, submental electrical stimulation, weight reduction programmes, bariatric surgery, nasal dilators,, various means of avoiding sleep in the supine position and life style modifications. Treatment options can be broadly divided into:

1. **Behavioural Interventions-** The patients with obstructive sleep apnoea who are obese should be advised to undergo weight reduction therapy as it improves symptoms of OSA and other related disorders. Smokers should be advised to stop smoking for general health. Alcohol should not be used and drugs and sleeping tablets should be avoided as this may decrease airway dilator function and worsen OSA.

2. Non-surgical Approach

Continuous Positive airway pressure (CPAP)-Continuous Positive airway pressure (CPAP) is the mainstay of therapy for most adults, as well as in a selected groups of paediatric patients with OSAS. It is the first treatment choice and the most widely used . The term CPAP (continuous positive airway pressure) means the application of ventilatory support in the form of continuous positive pressure generated by the device[14]. Treatment with CPAP is a safe, effective and tolerated therapeutic modality. Continuous distending airway pressure is applied during the sleep using a nasal, nasal-oral or face mask and small compressor. CPAP acts as a pneumatic splint to maintain airway patency. By simultaneously increasing the functional residual capacity, this pressure also helps prevent oxygen desaturation even if airway obstruction breaks through.

Newer modalities of positive airway pressure ventilation-

Bilevel PAP ventilation provides two different levels of pressure(higher during inhalation and lower during expiration) and can potentially treat OSA at a lower mean pressure than CPAP, at the same time improving lung ventilation *via* a pressure support.

Bilevel PAP, although more expensive than CPAP, is therefore a valid alternative in patients intolerant to CPAP and in patients with associated hypoventilation or chronic obstructive pulmonary disease.

Mandibular Repositioning Appliances (MRA)- A number of different mandibular repositioning appliances (MRAs) have been suggested for the treatment of snoring and obstructive sleep apnoea.[15]

The upper airway is widened by anterior displacement of the base of the tongue, epiglottis and soft palate, produced by the MRA. The device also prevents posterior displacement of the mandible in the supine position.

The devices are individually fabricated and advance the mandible by about 5 mm or 50–75% of maximum protrusion. Protrusion by 75% of maximal range has been shown to decrease AHI more than protrusion by 50% in patients with severe OSAS. This effect was

not seen in patients with a mild to moderate form of the disease. The appliances may be of a soft or hard material, either monoblock or adjustable two-piece devices. Patients must have teeth, and children should not be treated due to the high risk of dental displacement. Patients are usually treated and followed up by dentists. A sleep apnoea investigation during treatment is needed to monitor the effect on apnoea and hypopnoea reduction.

Other oral devices suggested as a treatment of OSAS include tongue retaining devices that hold the tongue anteriorly in a plastic bulb, mouth shields to prevent mouth breathing and soft palate lifters to reduce soft palate vibrations.

Surgical Interventions-

Surgery is considered when non-invasive therapy such as CPAP and oral appliances has been not successful. It is done in a situation when there is any deformity in anatomic structure that can be later on corrected to eliminate the breathing problems.

Uvulopalatopharyngoplasty (UPPP):

It is the reconstruction of the throat by resection of posterior margins of the soft palate and unwanted mucosa present on the pharyngeal walls.[16]

Consequences of Untreated OSA-

Obstructive sleep apnoea negatively impacts quality of life and is also associated with a number of adverse safety and health consequences including cardiovascular disease and motor vehicle crashes. Short habitual sleep duration can result in excessive daytime sleepiness and reduced neurocognitive function. Sleep loss may have long-term health consequences and may lead to premature death, cardiovascular disease, and the development of diabetes. Patients may also experience impaired concentration due to tiredness, increased irritability, depression and mood changes. There is an increased risk of high blood pressure and may have a slightly increased risk of angina, heart attacks and strokes.

Conclusion-

OSA is a common breathing disorder, which affects all age groups. It is a serious public health problem. Because of its potential pathophysiological consequences, it associates alteration of quality of life, decreased economic potential and increased morbidity and mortality in affected patients. Assessment of OSA requires a thorough clinical examination as well as overnight testing to determine its presence and severity before initiating treatment. Polysomnography remains the most common and reliable test for OSA diagnosis. Treatment modalities of OSA are aimed at increasing life expectancy, decreasing disease problems and improving the quality of life. CPAP is still the mainstay for treatment of moderate to severe

OSA. However, medical or surgical alternatives can be used in case of failure or non-compliance of the patients.

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