CLINICAL APPROACH TO OBSTRUCTIVE SLEEP APNEA (OSA)

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Received:- 17/08/2023
Revised:- 23/08/2023
Accepted:- 30/09/2023

Repetitive collapse of the upper airway during sleep leads to sleep-disordered breathing disorders which present as obstructive sleep apnea (OSA) and obstructive sleep apnea and hypopnea syndrome (OSAHS). In Indian studies, the prevalence of OSA varied from 4.4% to 13.7% and that of OSAHS varied from 2.4% to 2.8%. Obstructive sleep apnea is being increasingly recognized as a major public health problem worldwide, including India (1). Awareness among the general public and even among doctors is very low in India. This disorder is becoming increasingly common among obese individuals, patients with metabolic syndrome, children, and postmenopausal women.

Patients with OSA have episodic upper airway obstruction and hypoxemia, which form the basis for symptoms like loud snoring, witnessed apneas, excessive daytime sleepiness (EDS), and an increased propensity for accidents. OSA is also associated with complications such as hypertension, ischemic heart disease, and stroke. It also results in poor quality of life and an increase in health care costs. OSA is now a proven causal factor for various cardiovascular complications such as stroke, myocardial infarction, arrhythmias, and sudden cardiac death (2). It also results in cognitive dysfunction and accidents due to difficulty in concentration and excessive sleepiness while driving.

OSA is the occurrence of an average five or more episodes of obstructive respiratory events per hour of sleep with either sleep-related symptoms or co-morbidities or ≥15 such episodes without any sleep-related symptoms or co-morbidities. OSAHS is defined as OSA associated with daytime symptoms, most often excessive sleepiness.

The American Academy of Sleep Medicine (AASM) defines apnea as a reduction in airflow of ≥ 90% lasting at least 10 seconds. A hypopnea is defined as a reduction in airflow of ≥ 30% lasting at least 10 seconds and associated with a ≥ 4% decrease in pulse oxygenation, or as a ≥ 30% reduction in airflow lasting at least 10 seconds and associated either with a ≥ 3% decrease in pulse oxygenation or with arousal (3).

PATHOPHYSIOLOGY

The pathogenesis of OSA can be multifactorial, and complex and is not completely understood. Certain factors like obesity, thickened lateral pharyngeal walls, nasal congestion, enlarged uvula, facial malformations, and tonsillar hypertrophy contribute to the development of OSA. Repetitive collapse of the upper airway during sleep results in diminished airflow or cessation of airflow leading to sleep fragmentation and arousals. Disordered breathing during sleep leads to intermittent hypoxia-induced activation of oxidative stress and chronic inflammation that is postulated to result from fragmented sleep, intermittent hypoxia, hypercapnea, intrathoracic pressure swings, and increased sympathetic nervous activity (4).

Associated risk factors

The following risk factors have been identified in the development of OSA (5)

- Male gender (up to age 65),
- Increasing age
- Menopause,
- Overweight and truncal obesity reflected by several markers including BMI,

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Dariya S S, Inamdar A V
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- neck circumference (>17 in men & >16 in women),
- waist-to-hip ratio,
- craniofacial abnormalities,
- hypothyroidism
- upper airway anatomy,
- smoking, alcohol, and genetic predisposition

CLINICAL PRESENTATION:

Patients usually present with a history of habitual loud snoring along with a sensation of choking. Hypersomnolence (Excessive daytime sleepiness) especially dosing off during reading and watching TV is classic symptom of OSA. Other common complaints are falling asleep while driving, morning headaches, difficulty in concentration and loss of libido.

The Epworth sleepiness scale (ESS) is a short self-assessment to identify how likely patients are to fall asleep during the daytime.

EPWORTH SLEEPINESS SCALE

How likely are you to dose off or fall asleep during the following situations?

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

	Score
1. Sitting and reading	0 1 2 3
2. Watching TV	0 1 2 3
3. Sitting, inactive in a public place	0 1 2 3
4. As a passenger in a car for an hour without a break	0 1 2 3
5. Lying down to rest in the afternoon when circumstances permit	0 1 2 3
6. Sitting and talking to someone	0 1 2 3
7. Sitting quietly after a lunch without alcohol	0 1 2 3
8. In a car, while stopped for a few minutes in the traffic	0 1 2 3
Total	

Epworth Score Interpretation:

0-7 : It is unlikely that you are abnormally sleepy.

8-9: You have an average amount of daytime sleepiness.

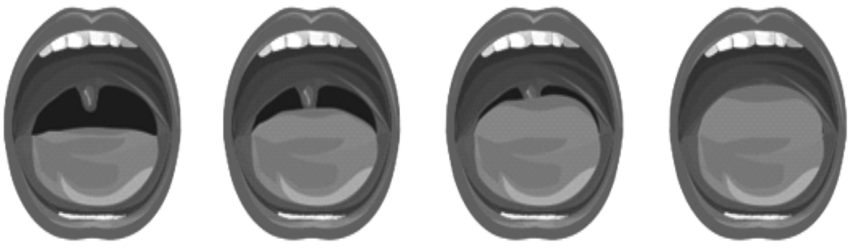
10-15: You may be excessively sleepy depending on the situation. You may want to consider seeking medical attention.

16-24: You are excessively sleepy and should consider seeking medical attention.

PHYSICAL EXAMINATION:

All patients should undergo a proper ENT examination to rule out septal deviation and enlarged tonsils. Higher Mallampati airway class is associated with increased severity of OSA.

The Mallampati Score



CLASS I Complete visualization of the soft palate

CLASS II Complete visualization of the uvula

CLASS III Visualization of only the base of the uvula

CLASS IV Soft palate is not visible at all

DIAGNOSTIC TESTING:

The gold standard for diagnosis is **Polysomnography (PSG),** or, sleep study. This test is performed in a sleep laboratory while the patient is asleep, and it monitors brain waves, blood oxygen levels, heart rate, and breathing along with eye and leg movements. Data is analyzed for sleep staging, frequency of respiratory events, and limb movements. Nowadays most sleep studies are performed as `Split studies' where the first few hours of the study are used for diagnosis of OSA and the second part of the study is used for CPAP titration if the AHI is consistent with moderate to severe OSA (6).

TABLE. APNEA-HYPOPNEA INDEX SCORE CLASSIFICATION FOR ADULTS	
APNEA SEVERITY	APNEA-HYPOPNEA INDEX (AHI) (EVENTS/HOUR OF SLEEP)
Normal	<5
Mild	5 ≤ AHI < 15
Moderate	15 ≤ AHI <30
Severe	≥30

OSA is diagnosed based on a sleep study (polysomnography) where the apnea–hypopnea index (AHI) is calculated, that is, the number of apneas and hypopneas per hour of sleep. An apnea is defined as the absence of airflow (reduction to less than 10% of baseline for ≥10 seconds), and hypopnea is defined as a reduction in the airflow by ≥30% of baseline for ≥10 seconds and is accompanied by a ≥3% decrease in oxygen saturation or arousal.

Recent consensus states that the event is obstructive if any of the following criteria are met: 1) there is snoring during the event; b) there is increased inspiratory flattening of the nasal pressure waveform; and/or c) there is associated paradoxical motion of the chest and abdominal respiratory inductance plethysmography excursions. The event is central if none of these criteria are met. An OSA syndrome is present if OSA is accompanied by symptoms of daytime sleepiness, as assessed by the Epworth sleepiness scale (ESS) with a cut-off of a score of 10 points (maximum 24). Apart from AHI, the arousal frequency and the degree of nocturnal desaturation are found to be related to relevant pathophysiological aspects of the disease. It should also be noted that the definitions of hypopnea have changed over time and that depending on these definitions, the prevalence of OSA can vary significantly (7). Some studies have used the respiratory disturbance index (RDI) to define OSA, a parameter calculated from respiratory polysomnography, describing the number of events per hour of registration (not sleep).

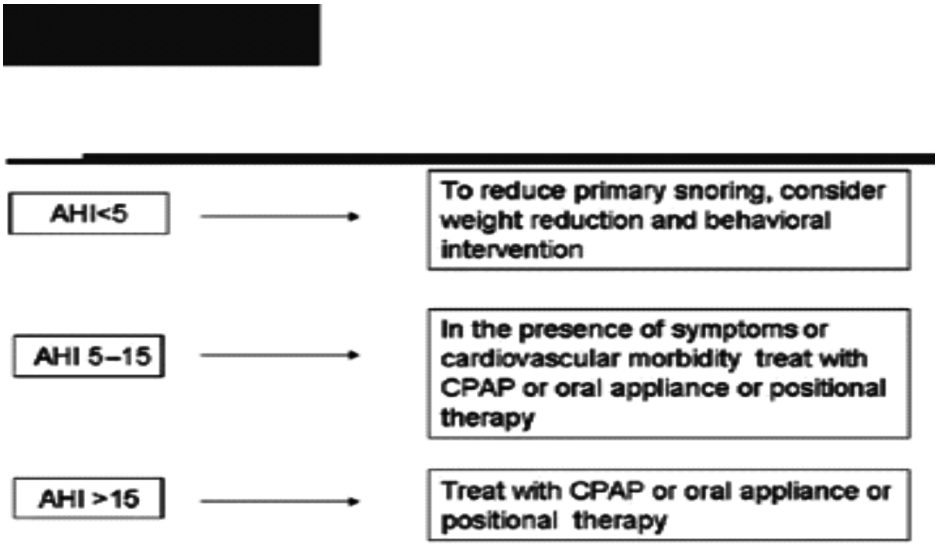
Differential diagnosis for daytime sleepiness include sleep deprivation, hypothyroidism narcolepsy, and medication side effects. One should also rule out COPD, CHF, and GERD.

TREATMENT OF OSA

RISK MODIFICATION:

1. Lifestyle modification
2. Weight loss
3. Avoid alcohol and sleep deprivation
4. Treat nasal obstruction (if present) and postural therapy
5. The choice of treatment for OSA depends on

-OSA Severity
-Symptoms
-Comorbid medical conditions
-Patient preferences (affordability)



Positive Airway pressure devices (PAP) devices function as pneumatic support that allows one to maintain upper airway patency by increasing the upper air-way pressure above a 'critical' value (pressure value below which the airways collapse) (6).

CPAP is the first-choice treatment for patients with moderate to severe OSA. Continuous PAP (CPAP), generally administered through the nose (nCPAP), delivers a single pressure to the posterior pharynx throughout the night and acts as a pneumatic splint that maintains the patency of the upper airway in a dose-dependent fashion. The best pressure for CPAP treatment is typically determined during an in-laboratory attended sleep study. CPAP therapy is indicated in all patients with an AHI greater than 15, independently from the presence of comorbidities, type of work, and severity of symptoms; if the AHI is above 5 and below 15, CPAP is indicated in the presence of symptoms (i.e. sleepiness, impaired cognition, mood disorders) or in the presence of hypertension, coronary artery disease or previous cerebrovascular accidents (7,11). CPAP is a highly cost-effective intervention that reduces the risk of cardiovascular events and it also improves metabolic syndrome associated with OSA. With the use of CPAP other associated conditions with OSA like hypertension, nocturia, polycythemia, and peripheral edema may also improve.

BiPAP machines are the preferred treatment for OSA patients who have complex sleep-related breathing issues, including severe OSA that requires very high pressure levels and intolerance to CPAP. It may also be recommended for people with sleep-related hypoxemia, where the blood oxygen levels are too low. Certain BiPAP modalities are the recommended treatment for patients who have a combination of obstructive and central sleep apnea.

Modafinil or armodafinil may improve daytime sleepiness in patients with persistent symptoms despite adequate CPAP use.

Oral Appliances

The most frequently used oral appliances are **mandibular advanced splints** (MAS). These splints reduce pharyngeal collapsibility and the risk of overall apnoeic events will be lowered. Significant reductions in AHI have been reported with the use of MAS. Young people, women, patients with small necks, and milder OSA will benefit from the use of these oral appliances (8).

Surgical Treatment

The surgical treatment removes the cause of upper airway obstruction and widens the airway, after precise detection of the obstruction site. The most common sites of obstruction are the oropharyngeal tract (collapse of the retropalatal and retrolingual regions due to macroglossia, low-lying soft palate or enlarged tonsils) and the nose (congestion, polyposis, chronic rhinitis). Tonsillectomy and adenoidectomy are the most commonly used highly effective surgical procedures to treat OSA in children.

Another widely used surgical procedure for the treatment of OSA is **Uvulopalatopharyngoplasty (UPPP**), either conventional or laser-assisted (LAPP). UPPP is the most commonly performed surgical treatment of mild to moderate OSAHS patients who do not respond to medical therapy (9). Tracheotomy especially maxillomandibular advancement (MMA) is a highly effective surgical treatment for OSA and must be carried out in patients with severe OSA for whom all other treatment approaches have failed.

Bariatric Surgery:

Various observational studies have shown the beneficial effects of weight loss in reducing obstructive sleep apnea severity. A significant reduction in AHI score is possible with a decrease in body weight. In patients with morbid obesity (BMI > 40) bariatric surgery, including gastric bypass and bandage, is a good alternative. When conservative treatments like CPAP, oral devices, and upper airway surgeries fail, Bariatric surgery is the optimal alternative for achieving considerable weight reduction (10).

Avoidance of risk factors such as tobacco, alcohol consumption (particularly in the evening), use of sedatives, and hypnotics should be emphasized. Weight loss for obese and overweight patients and the use of nocturnal CPAP for severe OSA should be regularly followed.

A multidisciplinary approach and the implementation of educational programs will significantly improve the management of OSA.

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