Obstructive Sleep Apnea Hypopnea Syndrome – Indian scenario

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ABSTRACT

Obstructive sleep apnea syndrome is far more common than generally believed. The Indian population where obesity rates are also increasing also has a very large population with undiagnosed OSAHS. The association of OSAHS with cardiovascular abnormalities further complicates the disease. Understanding the pathophysiology of OSA in obese and non-obese persons helps in evaluation, diagnosis and management in the individual group. With wider availability of PSG tests and newer treatment modalities, diagnosis and management of OSAHS has become easier. Increasing awareness regarding OSA and OSAHS among the general population and clinicians, wider availability of PSG studies and decreasing the costs of diagnostic tests and treatment can go a long way in identifying and treating these patients.

Key words: Obstructive sleep apnea hypopnea syndrome,Obstructive sleep apnea, Polysomnography, Obese.

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INTRODUCTION

Obstructive sleep apnea hypopnea syndrome (OSAHS) is a disease characterized by snoring, snoring disrupting the sleep, repeated episodes of complete or partial pharyngeal obstruction during sleep resulting in nocturnal hypoxemia, frequent arousals leading to cardiac abnormalities and excessive daytime sleepiness.[1] OSAHS is a far more prevalent disease than generally believed. The global prevalence varies from 0.3 % to 5.1% in general population.[2] In Indian studies obstructive sleep apnea (OSA) varied from 4.4% to 13.7% and OSAHS varied from 2.4% to 2.8%.[3] OSA in Indian males varied from 4.4 % to 19.7% and in females it was between 2.5% to 7.4% from various studies. OSAHS was observed in males from 2.4% to 7.5% and in females it was 1% to 2.1%.[3] The new classification of OSA given by the American Association of Sleep Medicine is[4]

1. Obstructive and central sleep apnea syndromes.

2. Sleep related hyperventilation/hypoxic syndromes due to congenital, idiopathic or medical conditions and

3. Other unspecified sleep related breathing disorders.

Definitions

OSA: Five or more apneas per hour of sleep, each apnea being ≥10 seconds duration.[5]

OSAHS**:** OSA with daytime sleepiness is called as OSAHS.

Hypopnea: 50% reduction in flow with 4% decrement in oxygen saturation.[6]

Respiratory related arousals (RERA**)**: Detectable changes in EEG are for more than 3 seconds. This term also includes micro arousals.[7,8]

Diagnosis

The gold standard test for OSAHS is overnight in laboratory multichannel Polysomnography (PSG).[2,9] This test includes surface leads for Electroencephalography (EEG), Electrooculography (EOG), Electromyography(EMG), Electrocardiography (ECG), nasal pressure transducer (or a thermistor) for detection of nasal airflow, thoracic and abdominal impedance belts for respiratory effort, Pulse oximetry, tracheal microphone for snoring and sensors for leg and body positions.

Pathophysiology

Factors leading to obstructive sleep apnea include[10]

1. A compromised often completely closed extrathoracic upper airway (obstructive event).

2. Marked reduction or cessation of brain stem respiratory center motor output (central event)

3. A combination of obstruction and central events.

These events produce intermittent hypoxemias leading to arousals from sleep, sleep fragmentation and further causing over compensatory responses of autonomic nervous system. The common sites of obstruction are retropalatal, retropharyngeal and hyypopharyngeal areas. Bony abnormalities like reduced mandibular body length, inferiorly positioned hyoid bone, retroposition of maxilla, increased vertical length of the upper airway [10] can lead to OSA. An enlarged soft palate and tongue would reduce the anteroposterior diameter where as enlarged pharyngeal walls reduces the lateral plane.

Obesity and OSA: Obesity indirectly contributes to upper airway narrowing during sleep because the lung volumes are markedly reduced by increased abdominal fat mass and decreased lung volumes in recumbent position. Obesity also gives rise to fat free muscle tissue. Leptin, an adipocyte derived factor affecting respiratory stimulant action and abnormalities in leptin signalling pathway can affect the respiratory pattern.[11,12] Many adipose tissue released inflammatory mediators like TNF-α, IL-6 are elevated in OSA and decreases with treatment with continuous positive airway pressure(CPAP).[13,14] The disease is also more common in supine position [15,] REM sleep, alcohol intake or sedative usage at bed time. [16] Pregnant females also have a high snoring percentage, which was 13.5% in an Indian study conducted in pregnant females, also showed a high sleep disordered breathing (SDB) of 9.5%.[17] Incidence of preeclampsia, meconium stained liquor, lower appearance, pulse rate, reflexes, activity, respiratory rate (APGAR score), lower birth weight was observed in third trimester females on follow up in New Delhi. Abnormal lung function tests (spirometry) are also common in OSA patients. Decrease in (FVC), FEV1/FVC, ratio of forced expiratory volume in first second and forced vital capacity, forced expiratory flow (FEF) 25-75%, PIFR and a ratio of the peak inspiratory flow rate and peak expiratory flow rate PIFR/PEFR≥ 1was observed in 92% of Indian OSA patients. A negative correlation was observed in daytime arterial PaO2 and respiratory distress index (RDI), and arousal index.[18] The effects of OSA include systemic hypertension, pulmonary arterial hypertension, hypercapnea, coronary artery disease, cardiac arrhythmias. Daytime systemic hypertension as a consequence of severe OSA can be explained by enhanced chemoreceptor sensitivity causing excessive sympathetic vasoconstrictor activity on vessels.[10]

Risk Factors: Obesity, male sex, age, heritable factors, craniofacial anatomic predisposition are the risk factors. Approximately 60-70% of OSA are obese. The occurrence of OSA in non obese exhibits different characteristics. In Indian children Adenoids and tonsillar enlargement (72%) were the causes for snoring, whereas disproportionate upper airway anatomy at multiple levels like long thick soft palate (62%), long and edematous uvula (62.8%), webbing of tonsillar pillars (40%) were the causes.[19] Incidence of OSA increases with age from 2% at age 30 for females to 28% at 60 years and from 4% at age 30 to 67% at 60 years of age for males.[12] Elderly also have high snoring and high excessive daytime sleepiness (EDS), with 27% being habitual snorers.[20] The prevalence of obesity is increasing in Asians.[21] The increase in prevalence from 2.3% to 19.6% occurred over a period of a decade up to 1998 and rate further increased when a lower cutoff value of 23- 25kg/M^2 was given for Asians. The national family health survey, India (NFHS) report shows that 12.1% of men and 14.8% of women in India are overweight or obese.[22] According to a study in industrial workers in a city in India 30.9% of men and 32.8% of women were obese.

Evaluation

Evaluation should include a comprehensive questionnaire regarding Snoring, intensity of snoring, choking during sleep, witnessed breathing pauses during sleep, recurrent awakenings. Prior medical history, drug usage, alcohol consumption and smoking history, history regarding cardiovascular status should be carefully looked into because higher incidence of coronary artery disease, diastolic dysfunction, left ventricular dysfunction, pulmonary arterial hypertension arrhythmias and coronary artery plaques were observed in Indian patients with OSA in a study conducted in 2009.[23] A comparative study between obese and non-obese OSA patients in Lucknow revealed that obese had more smokers, alcoholics, diabetics, hypertensives and snorers.[24]

LV dysfunction was also observed in 46.8% of OSA patients by R Paul in another Study.[25] Increase in both systolic and diastolic hypertension was observed with increasing severity of OSA in a study conducted in Mumbai.[26] Measurements of Body mass index, neck circumference, thyromental distance and estimation of Mallampati score must also be done.

OSAHS in Indian studies

Majority of PSG centers and institutes where studies on OSA or OSAHS in Indian population are being done, are in big cities in India. Prevalence of sleep disordered breathing (SDB) and OSAHS in middle aged urban Indian men were studied in a two phased cross sectional study by Udwadia et al, in Mumbai.[27] SDB was found in 19.5 %, OSA in 7.5% and EDS in 22%.The snorers had high body mass index (BMI), neck girth, waist girth and hip girth compared to non snorers. Obese individuals also had higher desaturation index, higher arousal index, 86.6% of obese individuals had SDB, 6.6% RERA's,80% had OSA and OSAHS in 37.5%.[25,28] In another study in Mumbai SDB was found in 3.42% of general population with habitual snoring in 6.4%.[29] In New Delhi, snoring was seen in 39.5% (males 49.5% and females 29%) with habitual snoring in 28.2%, SDB in 4.3%, EDS in 48.6% and a high BMI of 13.7%.[30] EDS increased with increasing severity of Bronchial Asthma (30.8% in asthmatics vs 11% in nonasthmatics) was observed by Ghoshal et al.[31]A study in paediatric group in New Delhi showed 12.7% of school children being snorers with SDB in 4.8% and EDS in 24.3%. In children with snoring and mouth breathing, 60% on PSG showed severe SDB and 30% moderate SDB.[32]

Acceptance of nasal CPAP in Indian OSA patients is low, cost being the major impediment. Discontinuation of CPAP was observed in 25% with leakage from the nasal mask being the major cause.[33] Oral appliances were successful in CPAP uncomfortable patients as observed by Jayan et al.[34] Children with OSA responded better to appropriate surgery after careful selection of patients.[35]

CONCLUSION

Sleep related disorders are high in Indian population but accessibility for diagnosis and treatment is still at a very low level due to various reasons. Increasing awareness regarding OSA and OSAHS among the general population and clinicians, wider availability of PSG studies and decreasing the costs of diagnostic tests and treatment can go a long way in identifying and treating these patients.

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