OSA: An awaking disorder!

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Abstract: OSA is characterized by repeated narrowing or collapse of the upper airway during sleep. It is the most common sleep disordered breathing seen among general population and contributes to increased morbidity and mortality. Recent lifestyle modifications have made Indian population more prone to this chronic disorder. Epidemiological studies show an increase in OSA prevalence among Indian population. OSA is associated with various co-morbid conditions which can be easily prevented by early diagnosis for which awareness should be brought among practicing Indian physicians and general population.

Keywords- CPAP, co-morbidities, obstructive sleep apnea, polysomnography (PSG), risk factors

I. INTRODUCTION

Recent lifestyle modifications due to rapid growth in science and technology have led to various changes in sleeping pattern of human, which is an essential component of life. Disturbance in sleep pattern hinders various physiological functions initially and subsequently leads to lifestyle disorder. Sleep disordered breathing (SDB), a lifestyle disorder is characterized by frequent ventilatory overshoots and undershoots with swings in arterial blood gas levels [1]. One such type of sleep disordered breathing is sleep apnea. Apnea is a condition in which absence of spontaneous breathing occurs more than 10seconds with decrease in airflow \geq 90% and hypopnea is decrease in airflow \geq 30% more than 10seconds [2]. Sleep apnea is a condition in which apnea occurs frequently during sleep causing disturbance in body homoeostasis. Generally, sleep apnea (a SDB) is divided into central, obstructive and mixed. Central sleep apnea(CSA) is due to suppression of central respiratory centers leading to reduced or cessation of motor output to pharyngeal muscles causing collapse of the airway [1].

Obstructive sleep apnea (OSA) or pickwickian disease is a potentially disabling condition characterized by repeated narrowing or collapse of the upper airway during sleep. Episodes of complete or partial airway narrowing are often associated with oxygen desaturation and terminated by an arousal from the sleep [3]. Mixed type is a combination of central and obstructive sleep apnea and seen in very few patients. OSA is the most common SDB seen in the general population and recognized as increased cause of morbidity and mortality [4]. Severity of OSA is diagnosed using Apnea-Hypopnea index (AHI). AHI is defined as the number of apneas and hypopneas per hour of sleep. Mild OSA - AHI between 5-15, moderate OSA - AHI between 15-30 and severe OSA – AHI >30 per hour of sleep. Obstructive sleep apnea syndrome (OSAS) is clinically characterized by AHI > 5 with excessive daytime sleepiness (EDS). Snoring is recognized as a characteristic feature of OSA [5], but, all snorers are not diagnosed with OSA. Snoring in absence of OSA is diagnosed when habitual snoring occurs with AHI <5 episodes per hour of sleep and absence of daytime symptoms.

Various unidentified forms of sleep apnea were reported in 19th century itself. In 1870's British physicians described OSA as 'fruitless contraction of inspiratory and expiratory muscles against closed glottic obstruction with accompanying cyanosis during sleep' [6]. Finally, Gastaut et al [7] recognised OSA in obese cases in 1960's, following which physiological research in sleep and breathing exploded in a huge manner. From 1990's, there is an increase in physiological research and clinical approach towards this chronic debilitating clinical disorder.

1.1 Classification

OSA is due to obstruction of the upper airway that leads to apneic episodes during sleep. Fujita et al [8] had classified the anatomical obstruction levels of OSA as follows:

Type 1: collapse in retropalatal region only

Type 2: collapse in retropalatal and retrolingual region

Type 3: collapse in retrolingual region only.

Retropalatal region is seen as the most common site of collapse [1]

1.2 Prevalence of OSA

Worldwide, OSA has been recognized as a major contributor to morbidity and mortality [4]. OSA prevalence is not the same among various ethnicities. Epidemiological surveys say that prevalence of OSA worldwide is 4% in males and 2% in females [9]. The prevalence is found to be more in developed and in developing countries. India being a fast developing country has prevalence of OSA also rising. A study conducted in semi-urban Delhi population suggested OSA prevalence to be 19.7% in men and 7.4% in women [4]. Another study conducted in northern India revealed an overall prevalence of 9.3% for OSA with 13.5% in male and 5.5% in female [10]. These studies suggest that OSA is undoubtedly increasing in India

II. RISK FACTORS OF OSA

OSA is a multifactorial disorder. Various risk factors include increasing age[4,5], male gender[4,6,11], snoring[5,12,13], habitual smoking[4], alcohol intake[4,5], obesity[4,5,12], high waist-hip ratio(WHR)[5,13], increased Neck circumference(NC)[11,13,14,15], percent predicted neck circumference (PPNC) – a height corrected neck measure of neck circumference which is calculated using formula PPNC = (1000* NC in cm)/(0.55* height in cm+310) [16]>90

Epidemiological studies shows that prevalence of OSA in higher in men than women [9,10,11]. The exact mechanism by which the male predominance exists is still unclear. But, various anatomical and physiological factors have been suggested to support this male predominance. In general males have increased NC, pharyngeal airway length and volume and increased soft palate volume than females which can cause OSA [17,18]. Though, men have larger pharynx than women there exists a vast change in pharyngeal area with lung-volume change than women which predisposes to airway collapse [19]. Pharyngeal airway in men was found to be more collapsible during sleep than women [20]. Testosterone, male sex hormone also increases upper airway collapsibility during sleep and induce/aggravate OSA in men [21,22,23].

Obesity is considered as an important risk factor of OSA [12,24]. In India, obese individuals have four times higher risk of developing OSA than non-obese individuals [4,10]. Obesity predisposes an individual to OSA by increasing fat deposits in the body leading to increase in WHR and NC. Increased adiposity in the neck directly mass loads the airway and predisposes to collapse, hence OSA [25]. But, 50% of the OSA patients were reported to be non-obese too [24]. The proposed reason is that in non-obese male OSA patients muscle mass content is almost less but the neck fat content remains the same. In addition, fat content in male neck is proportionately higher than other parts of the body which may also influence OSA preponderance [26].

III. CONSEQUENCES OF OSA

Studies on OSA patients have shown that it is associated with co-morbid conditions like cognitive, metabolic and cardiovascular consequences and affects the quality of life.

3.1 Cognitive consequences

OSA as it is characterized by intermittent hypoxia results in decrease in grey matter and network integrity in brain lobes causing impairment of vigilance, memory, psycho-motor performance, attention etc., [1]. Sleep fragmentation, a characteristic feature in OSA induced by frequent apnea causes dysfunction of neural networks in frontal lobe thus disturbing memory and cognitive functions [27].

3.2 Metabolic consequences

OSA by various mechanisms results in metabolic disturbances which ultimately disturbs the body homeostasis. OSA patients tend to be more prone to Type 2 Diabetes Mellitus. The probable mechanism could be that chronic intermittent hypoxia leads to high sympathetic activity which increases insulin resistance and decreased glucose tolerance and predisposes to DM in untreated patients of OSA [1,28].

3.3 Cardiovascular consequences

OSA patients have been reported to have increased risk of cardio-vascular diseases, OSA patients were found to have high hypertension. The probable mechanism is that OSA due to intermittent hypoxia leads to excessive daytime sympathetic vasoconstrictor activity, increased free radicals or reactive oxygen species, elevated levels of pro-inflammatory mediators such as C-reactive protein, TNF- α , IL-1 β , IL-6 and adhesion molecules which causes local and systemic inflammation promoting CAD, pulmonary hypertension and even Left ventricular failure [1,29].

OSA patients are prone to have metabolic syndrome, which is a combination of type-2 DM, Hypertension and dyslipidaemia. A combination of metabolic syndrome and OSA has been classified as Syndrome Z [30]. Treating OSA patients with CPAP (Continuous Positive Airway Pressure), the most effective treatment for OSA, decreases lipid profile [29,30].

3.4 Decreased quality of life

Lack of nocturnal sleep, a main feature in OSA patients, results in increased day time somnolence. It leads to decreased memory, attention, work performance, nervousness, impaired sexual performance [13], increased vehicular accidents [31], psychological depression [32] and decreased immune and general health which can affect physical and mental well being of an individual.

IV. DIAGNOSIS

OSA initially manifests as snoring [12], if untreated subsequently progresses to OSA. OSA patients exhibit variety of symptoms. Nocturnal symptoms include loud snoring [4,13], frequent apneas commonly witnessed by bed partner, gasping and choking leading to arousals, and restless sleep. Daytime symptoms include EDS (Excessive daytime sleepiness), morning headache, sore throat, decreased memory and depression [13]. EDS is assessed using Epworth Sleepiness scale. A score >10 is considered sleepy. There are other questionnaires also like Berlin questionnaire, STOP questionnaire, etc.,

A complete physical body examination including BMI, waist hip ratio, neck circumference and blood pressure gives data related to obesity and OSA. Oropharyngeal patency is assessed using modified Mallampati score which grades the positions accordingly.

- I Entire uvula seen with tongue at rest
- II Partial view of uvula seen
- $\ensuremath{\text{III}}-Soft$ and hard palate seen
- IV Only hard palate seen

Polysomnography (PSG) is the 'gold standard' method to diagnose OSA. It records various parameters related to sleep and helps in diagnosing OSA. It is performed as a single night sleep study either in a sleep lab or in home under supervision of a sleep technician. It records electroencephalogram (brain activity), electrocardiogram (heart activity), electooculogram (eye movements), submental and tibialelectromyogram (muscle activity), respiratory movements, nasal airflow and oxygen saturation level. Apneic episodes can be easily recorded using PSG studies.

V. TREATMENT

Treatment includes medical and surgical management. Medical management includes weight loss, Continuous positive airway pressure (CPAP) and oral appliances. Obesity is directly related to OSA [12]. Hence weight loss decreases fat deposition in the body especially in the neck, chest and abdomen, which prevents pharyngeal collapse and increases tracheal traction and lung volume. CPAP is the 'gold standard' medical management for OSA. Application of CPAP acts as a pneumatic splint maintains positive pressure at pharyngeal level and prevents collapse. CPAP usage has also reported to reduce OSA related co-morbid condition [33]. Application of oral appliances may also be used in some patients.

Surgical techniques include Uvulopalatopharyngoplasty(UPPP) the most common surgical procedure for treating OSA, in which palatal obstruction is eliminated by resecting redundant palatal and pharyngeal tissues [34]. Other procedures include lingualplasty, glossectomy and radiofrequency tongue ablation, genioglossal advancement and hyoid myotomy.

5.1 Hypoglossal-nerve stimulation

Reduced upper airway muscle activity leads to OSA. Hypoglossal nerve innervates genioglossus the important upper airway dilator. Hence stimulation of hypoglossal nerve which is usually done using Transcutaneous electrical nerve stimulator (TENS), causes contraction of genioglossus leading to airway opening and preventing its collapse.

VI. CONCLUSION

Lifestyle modifications have resulted in an increase in prevalence of OSA among Indian population. OSA has been associated with various co-morbid conditions which if goes unnoticed shall become detrimental to the individual. Awareness among Indian practicing physicians on SDB was only 10% [35] which should be rectified by updating sleep medicine among practicing physicians to identify, diagnose and treat patients with the awaking disorder - OSA.

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