

Original Research Article

## Study on Clinical Profile of Obstructive Sleep Apnea (OSA)

Dr. Shyamala KK<sup>1</sup>, BidhataKhatri<sup>2</sup>

<sup>1</sup>Assistant professor, Department of Pulmonology, Dr. BR Ambedkar Medical College, Bengaluru, Karnataka 560045, India

<sup>2</sup>PharmD student, Acharya and BM Reddy College of Pharmacy, Bengaluru, Karnataka 560107, India

### \*Corresponding author

Shyamala KK

Email: [shyamalak@gmail.com](mailto:shyamalak@gmail.com)

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**Abstract:** Obstructive Sleep Apnea (OSA) is a major public health problem, largely unrecognized and undiagnosed. As there is paucity of data regarding the disease in India, this study was conducted to study the typical clinical features, mode of presentation, physical findings that help to identify patients with obstructive sleep apnea, documentation of associated risk factors and co- morbid conditions and to study polysomnography features. In our prospective, non-interventional study, we randomly assigned patients suspected with OSA and underwent polysomnography. Their clinical data was analysed. A total of 50 patients completed the study. The ages of patients ranged from 30-75 years, majority was in the age group 50-59 years. Males were predominant with male to female ratio of 4:1. The commonest clinical features were loud snoring (100%), excessive daytime sleeping (EDS) (90%), chokings (86%), irresistible sleep attacks (78%) and tiredness on waking (70%). All patients were found to be obese. On polysomnography test it was found majority of the patients had severe OSA. CPAP therapy lowered blood pressure and partially reversed metabolic abnormalities. Very few studies have been reported in India, which reveals lack of awareness of the disorder. It has considerable effect on patients with increased risk of other diseases. Quality of life, morbidity and mortality can be improved by effective treatment. Awareness of the disorder, pretest clinical predictors and establishment of more sleep lab centres in India will improve the situation.

**Keywords:** Obstructive sleep apnea, obesity, loud snoring, excessive daytime sleeping, polysomnography, mallampati score, positive airway pressure

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### INTRODUCTION

As there is paucity of data regarding Obstructive sleep apnea (OSA) in India, this study was conducted to study the typical clinical features, mode of presentation, physical findings that help to identify patients with obstructive sleep apnea, documentation of associated risk factors and co- morbid conditions and to study the polysomnography features.

OSA is a clinical condition characterized by recurrent episodes of complete obstruction (apnea) or partial obstruction (hypopnea) of the upper airway. It leads to increased negative intrathoracic pressure, sleep fragmentation, and intermittent hypoxia during sleep [1]. Obstructive sleep apnea syndrome (OSAS) is defined as OSA associated with daytime symptoms, most often excessive sleepiness [2].

OSAHS is largely unrecognized and undiagnosed [3, 4]. Young *et al.*; estimated that 93% of

women and 82% of men with moderate to severe OSAHS are not diagnosed. They reported that OSA is highly prevalent in general population affects 4% in male and 2% in female middle aged adults. In India study by Sharma et al reported that prevalence of OSA is similar to that of western nation [5].

The patho mechanism is unclear. The critical pathophysiological feature of OSA is sleep-related collapse of the upper airway (UA) at the level of the pharynx, the interaction of anatomic and neural state-related factors in causing pharyngeal collapse [6-9].

The major risk factors for the disorder include obesity, male gender, postmenopausal status and age. The prevalence of OSA increases with age, with a 2- to 3 fold higher prevalence in older persons ( $\geq 65$  years) compared with those in middle age (30-64 years) [10, 11].

Reasons for high prevalence in men is not clear however two factors seem to be important which are pattern of fat distribution and differences in sex hormones. Female progesterone has been shown to have impact on genioglossus muscle activity, causing upper airway less likely to collapse or narrow during sleep[12].

Excess body weight has been hypothesized to affect breathing in many ways, including alterations in upper airway structure (e.g., altered geometry) or function (e.g., increased collapsibility), disturbance of the relationship between respiratory drive and load compensation, and by exacerbating OSA events via obesity-related reductions in functional residual capacity and increased whole-body oxygen demand [10].

Sometimes skeletal or soft tissue abnormalities, including dysmorphism related to mandibular or maxillary size and position, narrowed nasal cavities and tonsillar hypertrophy, play an important role in the development of sleep apnea [12].

Several studies have shown increased risk of OSA in families of patients with OSA and also explain the ethnic differences. Apolipoprotein E (APOE) is associated with increased risk in younger subjects less than 65 years [13]. Higher OS A prevalence was found in non-obese Asian patients relative to white patients [10]. Other risk factors for OSA are smoking, alcohol, sedatives, nasal obstruction, tonsillar hyperplasia and endocrinology [14, 10]. Endocrinal diseases include hypothyroidism and acromegaly [15].

Snoring is a common symptom reported in the general population. Occurs in 35–45% of men and 15–28% of women.<sup>3</sup> Snoring is the most frequent symptom of OSAHS, occurring in 70–95% of patients [16]. Snoring is caused by vibration of the palate and other soft tissues in the oropharynx, indicates partial obstruction of the airway [17].

Excessive day time sleepiness is the other common symptom of obstructive sleep apnoea [16].

EDS is defined as sleepiness (the urge to sleep) that occurs in a situation when an individual is normally expected to be awake and alert. 30–50% of the general population without OSAHS report moderate to severe sleepiness; and reported in 90 % of patients. Measures of excessive daytime sleepiness[18] can be performed by subjective and objective tests. Subjective measures are Epworth Sleepiness Scale, Stanford sleepiness Scale and Karolinska Sleepiness Scale. There is no gold standard test to assess EDS. The most practical is the Epworth sleepiness scale [19]. Objective tests include multiple sleep latency test (MSLT), the maintenance of wakefulness test (MWT) and Osler test [18].

Other symptoms include witnessed apnea and nocturnal breathing pauses, choking, insomnia and arousals, unrefreshed sleep, tiredness on waking, early morning headache, personality and mood changes, impaired memory, impotence and enuresis [20, 21].

The co morbid conditions associated with OSA are endocrinological disorder like hypothyroidism and acromegaly. Hypothyroidism is much common than acromegaly [22]. Heart failure is a major risk factor for sleep related breathing disorders [23]. Hypertensive patient's especially non dippers (absent fall in nocturnal BP) have higher incidence of OSA [24]. Type-2 is often associated with OSAHS. It is not clear OSA is casual in the development of diabetes [25]. Other co-morbid conditions are COPD, ESRD, Epilepsy etc [26-28].

There are no specific signs to indicate OSA for physical assessment; however certain physical finding abnormalities are found to be associated [29]. Mallampati score is used for oropharyngeal assessment. The Mallampati classification may help quantify the degree of oropharyngeal anatomical obstruction. The classes are determined by the visible structure (fig 1)  
Class I, the soft palate, fauces, uvula, and pillars are visible.  
Class II, the soft palate, fauces, and a portion of uvula are visible.  
Class III, the soft palate and base of uvula are visible.  
Class IV, only hard palate is visible.

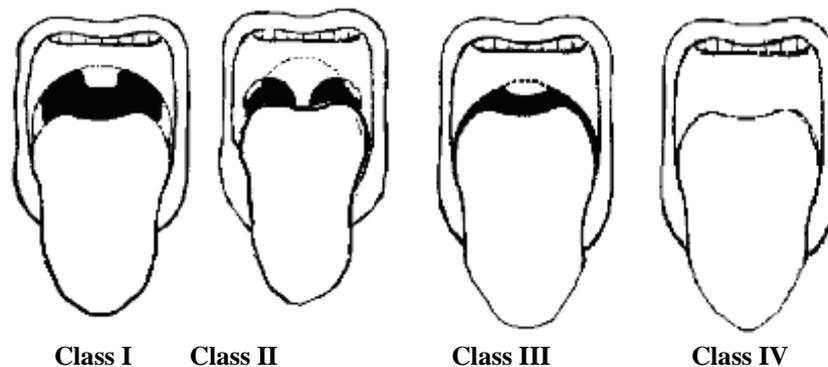


Fig 1: The Mallampati classification

The higher the mallampati classification, the greater is the likelihood of oropharyngeal obstruction. The classification assigned may vary if the patient is in the supine position. Other findings includes tonsillar enlargement which is defined as the presence of lateral impingement of greater than 50% of the posterior pharyngeal airspace (Clinical grade 2+ or greater). The uvula enlarged if it is more than 1.5 cm in length or > 1.0 cm in width. The soft palate considered low-lying if greater than one-third of the uvula extended below the level of the mandibular occlusal plane, lateral peritonsillar narrowing which is defined as impingement of greater than 25% of the pharyngeal space by the peritonsillar tissues, excluding the tonsils. Tongue is considered enlarged if it is above the level of the mandibular occlusal plane. Craniofacial abnormalities are over jet, retrognathia and micrognathia. Over jet was noted if there were greater than a 3-mm anterior-posterior distance between the upper and lower incisors during occlusion. Retrognathia is defined as a greater than 0.5cm retro position of thegnathion (the most inferior point in the contour of the chin) relative to the plane of the nasion (the deepest point of the superior aspect of the nasal bone) and subspinale (the deepest point on the Prem axillary outer contour). Micrognathia is hypoplasia of the mandible with foreshortening of the floor of the mouth and reduction of the size of the oral cavity.

Obesity can be measured by body mass index and central obesity. About 30% of patients with BMI >30 have OSA, and 50% of patients with BMI >40 have OSA. A 10% increase in weight predicted a 6-fold increase in the odds of developing moderate-to-severe OSA [30]. Waist to hip ratio (WHR) of > 0.95 in men and > 0.88 in women were used as parameters of obesity.<sup>5</sup> Neck circumference correlates with increased dimensions of pharyngeal fat pads, measured at the level of the superior border of the cricothyroid membrane. A neck circumference greater than 43 cm (17 in) in men and 37 cm (15 in) in women has been associated with an increased risk of OSAHS [16]. Neck circumference corrected for height is a useful predictor of obstructive sleep apnoea than general obesity [31].

For nasopharyngeal assessment septal deviation, polyps, and tumors contribute to nasal obstruction that predispose to OSA [12]. Other systemic examination may be normal or abnormal either as a result of the consequences developing from OSA or due to the associated co morbid illness.

The main Consequences of OSA are on central nervous system and cardiovascular system [32]. OSA is associated with a number of neurocognitive and behavioral outcomes including depression, sleepiness and poor quality of life [33]. In patients with OSAS, sleepiness can impair social function and can have a major impact on the ability to carry on daily life activities [32]. Diminished cognitive function occurs in

the areas of attention/vigilance, learning and short and long term memory, and executive function. Impaired QOL is common among patients with OSAS. These patients may experience moodiness, anxiety, depression, lack of motivation, and compromised performance in social function, all of which may contribute to a lower QOL that are reversible with treatment [20]. Drivers with OSAS are two to seven times more likely to have a motor vehicle accident compared with normal drivers. The cause being related to sleepiness and decreased performance [18, 34]. Epidemiological data suggest a strong relationship between OSAS and acute cerebrovascular events [35]. OSA is an independent risk factor for cardiovascular and cerebrovascular morbidity and mortality [36]. There is strong evidence that OSAS is an independent risk factor for systemic hypertension [37].

Untreated OSAS worsens the prognosis of patients with coronary artery disease (CAD) [38]. OSAS can lead to heart failure. It has also been recognised as a potential cause of arrhythmias during sleep. Metabolic abnormalities due to OSA include increased insulin resistance and type-2 diabetes [35].

According to the recently updated International Classification of Sleep Disorders published by the American Academy of Sleep Medicine [39] diagnosis of OSAHS can be made if

The respiratory disturbance index (RDI) is  $\geq 15$ , independent of occurrence of symptoms or whenever an RDI >5 is associated with any of the following:

1. Sleep attacks, excessive daytime sleepiness (EDS), unrefreshing sleep, fatigue or insomnia;
2. Awakenings with a choking sensation; or
3. Witnessed heavy snoring and/or breathing pauses referred by the partner.

Severity is classified depending on AHI [40]

- Mild - 5-15 episodes per hour
- Moderate - 15-30 episodes per hour
- Severe - More than 30 episodes per hour

The gold standard technique for the diagnosis of sleep apnoea and related disorders is overnight Polysomnography. Home-based sleep studies by using of limited diagnostic systems in clinical practice, which assess cardio respiratory variables during overnight studies can be utilized for diagnosis of OSA [41].

Diagnostic criteria for apnea and hypopnea are recommended by AASM Task force [40]. The mainstay of medical treatment of OSAHS is administration of non-invasive positive airway pressure (PAP) during sleep. It is the safest and first line therapy.

Three varieties of Positive pressure devices available in treatment of OSA.

1. Continuous Positive Airway Pressure (CPAP) is most commonly used. It acts as a pneumatic splint to the upper airway during sleep and corrects the obstruction. A
2. Bi-level positive airway pressure (BIPAP) and
3. Auto titrating positive airway pressure (APAP).

Other therapies are usually unsuccessful [14, 42, 43]. It includes oral appliance therapy like tongue repositioning devices and mandibular repositioning appliances. Surgical therapy remains an option for patients with intolerance for CPAP. It includes oropharyngeal surgery and minimally invasive techniques.

Different types of oropharyngeal surgery[42] are uvulo pharyngo palatoplasty, tonsillectomy, nasal surgery, and tracheostomy and jaw advancement techniques. Minimally invasive techniques include genioglossus advancement, tongue-base suspension and multilevel radiofrequency tissue ablation.

Adjunctive medical therapies[14, 43] include use of drugs like protriptyline and modafinil for OSAHS. Different lifestyle modification like weight reduction, avoidance of alcohol and sedative, smoking cessation, good sleep hygiene can also help in improving patient's condition [14]. Weight reduction even modest reductions in body weight are associated with changes in obstructive sleep apnea [44]. Correcting underlying medical disorders such as hypothyroidism with thyroid replacement hormone, growth hormone suppressant therapy for acromegaly, medroxy progesterone and estrogen therapy in postmenopausal women has shown some benefits [43]. Miscellaneous devices and aids like neck collars, nasal valve dilators, nasopharyngeal tubes,

pharyngeal lubricant, electrical stimulation of upper airway dilator muscles, Trans tracheal air insufflation, and even atrial pacing are also beneficial [14].

### MATERIALS AND METHODS

The study was a prospective, observational type of study. Patients who attended outpatient department of pulmonary medicine, Dr. BR Ambedkar Medical College, Bangalore between January 2015-December 2015 with high index of suspicion of OSA (Loud snoring, daytime sleepiness, etc) after detailed examination completing the questionnaire as per the proforma and underwent polysomnography were included in the study. Permission was obtained from the ethical committee to carry out the study. 55 patients agreed to undergo PSG. Out of 55, 50 had OSA as per the detailed examination and PSG; their characteristic features were analyzed and presented.

#### Method of Statistical Analysis:

The following methods of statistical analysis have been used in this study. The Excel and SPSS (SPSS Inc, Chicago) software packages were used for data entry and analysis. The results were averaged (mean + standard deviation) for each parameter for continuous data and numbers and percentage for categorical data presented in Table and Figure.

### RESULTS:

Among 50 patients enrolled, their ages ranged from 30-75 years. Number of cases was increasing with increase in the age up to 60 years. Majority of the patients were in the age group 50-59 years. The mean age of the study group was  $49.74 \pm 11.59$ . It showed males (41/50) were predominant with male to female ratio of 4:1.

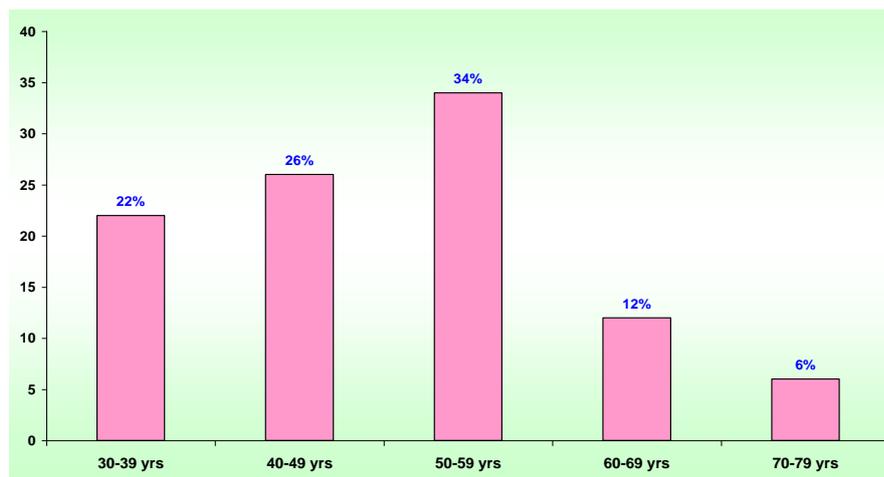


Fig-1: Distribution of Age

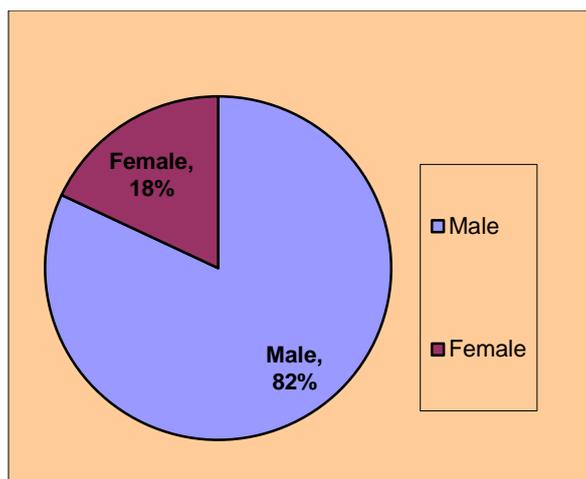


Fig 2 : Gender Distribution

Table 1: Clinical features of OSA

Complaints	Frequency	Percent
Loud Snoring (LS)	50	100
Excessive Daytime Sleepiness(EDS)	45	90
Chokings	43	86
Irresistible Sleep Attacks	39	78
Tiredness/ Fatigability	35	70
Nocturnal Enuresis	22	44
Impaired Memory /Concentration/ Mood Changes	21	42
Unrefreshed Sleep	14	28
Difficulty in falling sleeps	12	24
Witnessed Apnoea	12	24
Morning Headache	9	18
Early Awakenings	6	12
Problem in Reinitiation of Sleep	3	6
Leg Movements	2	4
Loss of libido	1	2

As shown above the commonest clinical features were Loud snoring (100%), 90% with EDS, Chokings in 86%, irresistible sleep attacks in different situations was found in 78%, 70% had tiredness on waking, nocturnal enuresis was reported in 44% of the subjects, was noted among patients of Diabetes, IHD and hypertension on diuretics, neurocognitive dysfunction were observed in 42%, unrefreshed sleep in 28% and witnessed apneas by the bed partners were reported in 24%. Bed partner reported snoring/ gasps/ choking sounds in 62% of patients.

It was observed 26% had the habit of alcohol consumption and 28% had smoking habits including current and ex-smoker and 2% with use of sedatives. It was found that 54% had associated systemic hypertension, 36% had COPD/RAD with associated hypercapnic respiratory failure in 4%. 11% with Diabetes Mellitus, 16% found to have CRF secondary Diabetes/hypertension and were not on dialysis, 14% had IHD, 6% with hypothyroidism and Epilepsy was noted in 4%.

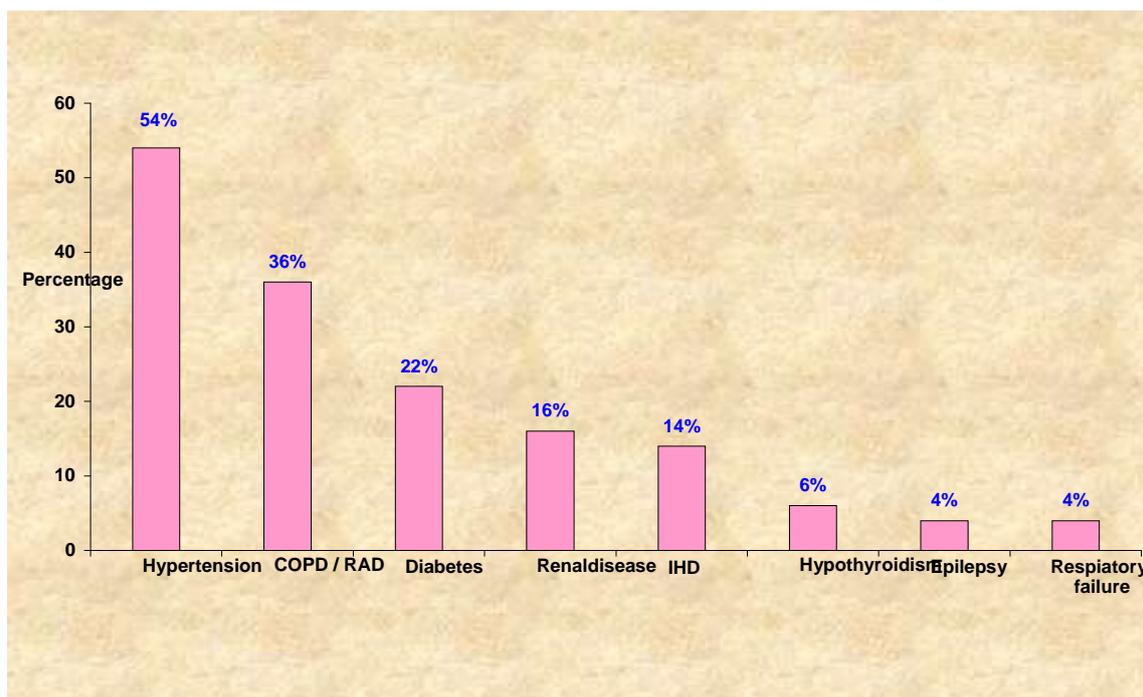


Fig-3: Associated co morbid diseases

Table-2: Assessment of Obesity

	Mean	Median	Mode	Std. Deviation	Minimum	Maximum
<b>WHR</b>	0.9582	.9500	.88	.0921	.80	1.27
<b>BMI Kg/m<sup>2</sup></b>	32.00	30.00	30	5.18	26	46
<b>NC, cm</b>	41.76	42.00	43	3.29	36	52

The study observed mean BMI of 32.00 ± 5.18 kg/m<sup>2</sup> (range, 26 to 46 kg/m<sup>2</sup>), Waist Hip ratio was also used to assess obesity found mean value of 0.95. (BMI-

25 kg/m<sup>2</sup> and WHR for >0.95 men and >0.88 females measure of obesity as defined by WHO standards for Indian population.

Table 3: physical findings

Total (N=50)	Obese	Micrognathia	Retrognathia	Nasopharyngeal abnormality
<b>Frequency</b>	50	5	3	12
<b>Percent</b>	100	10	6	24

All the 50 study patients were found to have obesity based on BMI and WHR measurements. Associated Retrognathia and Micrognathia were seen in 6-10%. Nasopharyngeal abnormality in the form of either DNS or hypertrophied turbinates was noted in 12 patients (24%).

**Mallampatti classification**

Majority (56%) of the patients were in class IV while 5% were in class I, 6% were in class II and 11% were in class III.

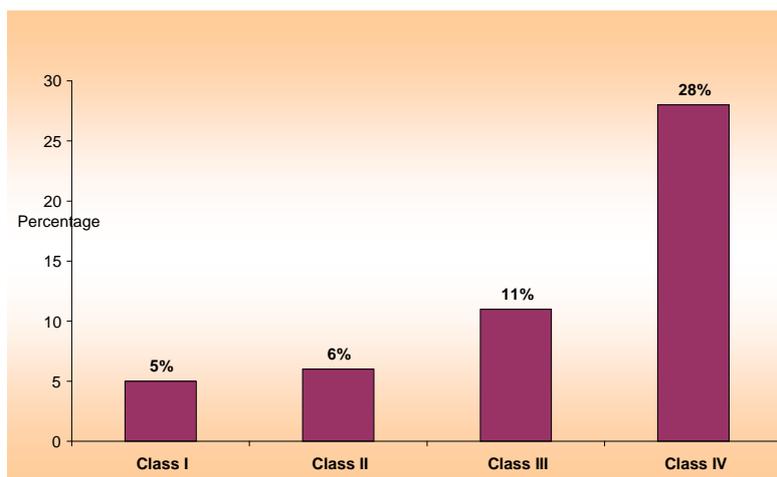


Fig-4: Mallampatti classification

**Soft tissue abnormality**

Total	Abnormality of Uvula		Low lying soft palate		Macroglossia		Tonsillar enlargement	
	No	percent	No	percent	No	percent	No	Percent
50	41	82 %	34	68%	29	58%	6	12%

Majority (82%) of OSA patients had abnormality of uvula either it was elongated or thickened, 68% with low lying soft palate, 58% had Macroglossia and 12% Tonsillar enlargement.

**Physical Assessment**

**A. OSA Severity:**

Among 50 patients, 41 had severe OSA, 8 moderate and 1 with mild OSA.

OSA Severity	Frequency	Percent
Mild	1	2.0
Moderate	8	16.0
Severe	41	82.0
<b>Total</b>	50	100.0

The Adjusted neck circumference 52 obtained by measuring the actual neck circumference and adding 4 cm if the patient has hypertension, 3 cm if the patient is a habitual snorer, and 3 cm if patient is reported to

choke or gasp most night. All the 50 patients had adjusted neck circumference in the range of 43-55 cm with intermediate and high probability of OSA.

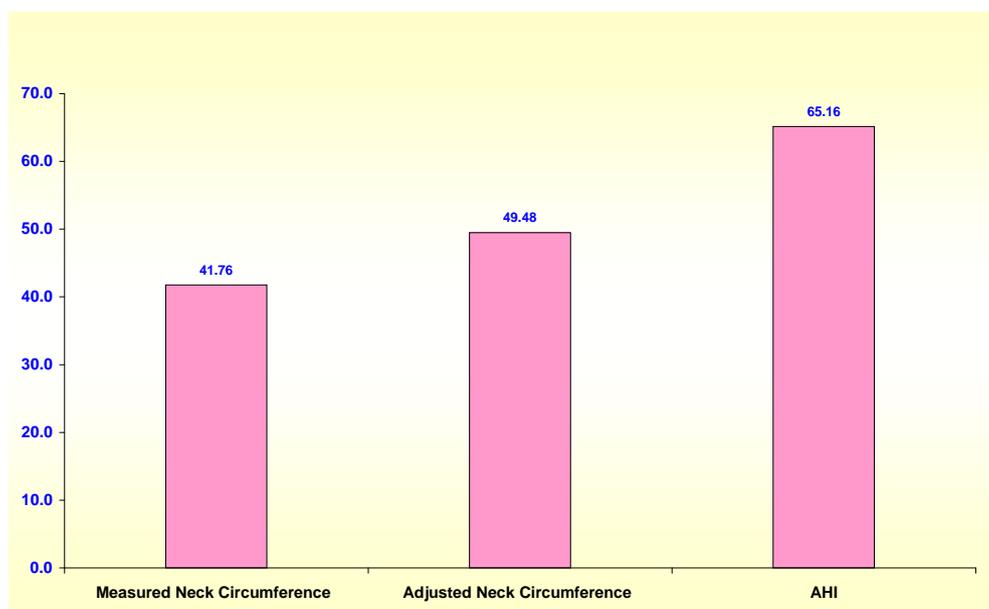


Fig-5: Clinical prediction of OSA using Neck circumference measurement

## DISCUSSION

The study was conducted in department of pulmonary medicine. Out of total 50 cases, 41 (82%) were males, with male to female ratio of 4:1. In a study conducted by Sreedharan SA, out of 152 patients, 119 were males and 33 were females [45]. Among the females studied 5 out of 9 were in the postmenopausal state. Age group of patients ranged from 30-75 years with a mean age of 49.74 years similar to the observation of Sharma[5] and Udwardia [46].

In the present study The major symptoms included loud snoring (100%) often reported by bed partner (62%), Excessive daytime sleepiness (90%), chokings (86%), irresistible tendency to sleep during different activity was reported in 78% of patients, tiredness on waking (70%), neurocognitive dysfunction (42%), unrefreshed sleep in 28% and witnessed apnoeas was reported in 24%, early morning headache (18%) and loss of libido in 2%. Loud snoring, Excessive daytime sleepiness, chokings and witnessed apnoeas are the major symptoms reported in other studies Duran[47], and Sharma [11].

In the Clinical assessment by Epworth Sleepiness Scale, present study found a mean value of  $14.08 \pm 5.09$ , when compared with severity of OSA it was found that mean value increased from 3 to 14.29 from mild to moderate OSA and not much change between moderate to severe OSA. Gottlieb Daniel[48] in their study reported that the mean ESS score and the ESS score of  $\geq 11$  increased from 7.8 to 9.3 and 28% to 35% from mild to severe OSA respectively.

The physical assessments revealed all patients were obese with BMI of  $32 \pm 5.18 \text{ kg/m}^2$ , and WHR with mean of 0.95. Neck circumference was used as a

measure of central obesity, noted a mean value of  $41.76 \pm 3.29$ , and results were similar as reported by Sharma[5] and Udwardia [46]. Oropharyngeal soft tissues abnormality observed were majority with enlargement of uvula, Low lying soft palate, macroglossia were observed in most of the patients. The study of Joseph[29] found lateral pharyngeal narrowing to be commonest cause for upper airway narrowing. Assessment of oropharynx by using Mallampatti score has higher likelihood predicting the OSA. In our study it was found that 28 out of 50 cases were in class IV of which 26 had severe OSA suggesting increase in the severity with increase in the score. In a study performed by Uzma N in which higher mallampatti score was observed in patients with severe OSA than in moderate and mild OSA patients [49].

Obesity was found in all the patients confirmed by BMI and WHR. It is considered as the major risk factor [10]. Majority 54% of patients had Hypertension similar to the observation reported by McNicholas[35] and Sharma[5]. Other associated co morbid conditions noted were 36% of COPD/ RAD, higher than that reported by Chaouat[26], Diabetes mellitus was found in 22% of patients; higher incidence compared to the observation of Reichmuth[47], but was similar to the results obtained in the study of Udwardia [8].

It was noticed 26% had history of consuming ethanol and 28% were smokers (14% current smokers) in the study group could have contributed to the increase in the severity of OSA. In a study conducted it was found that 44% had history of alcohol intake and 36% had history of smoking [45]. It was found that 4% had reported to have RTA/ Near misses in the study

group, lesser incidence compared to that reported by George[34] in his study.

Our study group had 41 cases with severe OSA, 8 were moderate, and 1 was mild OSA, Finding was similar to the study by Uzma N with majority of patients (56%) with severe OSA [49]. CPAP is the safest and first line therapy in obstructive sleep apnoea. In a study conducted by sharma, CPAP therapy lowered blood pressure and partially reversed metabolic abnormalities [44].

#### SUMMARY AND CONCLUSION

The study was conducted in a teaching hospital in south India. Prospective analysis of cases of obstructive sleep apnea confirmed by overnight polysomnography was studied. Observations of our study showed OSA is a disorder of middle age. OSA is a common sleep disorder that can present in a variety of ways. Our observation found majority of the subjects with severe OSA suggesting patients approach to the clinic/other specialty could be due to the severity of symptoms/comorbid condition. Mortality may be high among these patients if not treated. There is need to improve the awareness and further studies to recognize the milder form of the disease with regular sleep assessment should be carried out in routine practice.

Very few studies have been reported in India, which indirectly reveals the lack of awareness of the disorder among the medical fraternity, patients and the public. OSAHS has increased risk of other diseases. The quality of life, morbidity and mortality can be improved by effective treatment, and is so often unrecognized, it is important to improve the way these patients are diagnosed. Stroke cases were not found in the study group. Further studies with more sample size and control group are required to study the risk factors, co morbid conditions and the PSG features.

Understanding of its typical clinical features, risk factor, existing of co-morbid condition and the clinical consequences helps in prevention, early identification. Prompt initiation of treatment using nasal CPAP will reduce the morbidity, mortality and improve the quality of life. Awareness of the disorder, the pretest clinical predictors and establishment of more sleep lab centers in India will improve the situation.

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