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RESEARCH ARTICLE
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"A STUDY ON OBSTRUCTIVE SLEEP APNEA AND ANATOMICAL UPPER AIRWAY OBSTRUCTION"

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ABSTRACT

OBJECTIVE: To study anatomical upper airway obstruction in Obstructive sleep Apnea patients using Diagnostic Nasal Endoscopy and Video laryngoscopy.

In recent years, it has been observed that Obstructive sleep apnea is amongst one of the disorders with rising prevalence ranging from 9 to 38%. This study aimed to study the anatomical upper airway obstruction in Obstructive Sleep Apnea.

To assess the risk factors for Obstructive sleep apnea patients in the study population.

To assess severity of OSA in the study population.

SUBJECTS AND METHODS: 50 confirmed cases with the signs and symptoms suggestive of anatomical upper airway obstruction coming to the Department of ENT, Maharaja Yashwantrao Holkar Hospital, Indore, were selected. After observing required symptoms, patients were evaluated for risk factors. Diagnostic nasal endoscopy and Video laryngoscopy was done.

RESULTS: In our study, 76% (n=38) of the total patients were categorized as OSA group. Snoring was found in 66%(n=33) of the cases. Mouth breathing found in 64% (n=32) of the cases. Difficulty in breathing at night was found in 78%. BMI of more than 30 was found in 72%. Neck circumference of more than 17 inches was found in 48%. In this study, 68% (n=34) population were hypertensive, and 62% (n=31) patients had diabetes.

CONCLUSION: Obstruction at certain anatomical levels contributes towards obstructive sleep apnea severity. Multi-level obstruction is more prevalent in obstructive sleep apnea and is associated with increased severity. Risk factors increases the index of suspicion and encourage physicians to embark on a diagnostic path to investigate possible OSA.

KEYWORDS: Obstructive sleep apnea, Diagnostic Nasal Endoscopy, Video Laryngoscopy, Polysomnography.

INTRODUCTION

OSA is defined by the presence of repetitive episodes of upper airway obstruction that result in reduced or absent breathing during sleep[1].These episodes are termed "apneas" when breathing is completely or near-fully stopped, or "hypopneas" when breathing is reduced in part.[2] When OSA is

associated with symptoms during the day, the terms obstructive sleep apnea syndrome (OSAS) or obstructive sleep apnea– hypopnea syndrome (OSAHS) can be used to refer to it. [3, 4]

An AHI of equal to or greater than 5 events/h is commonly used to define OSA, with obstructive or mixed (rather than central) events comprising more than 50% of the total.[5]

In recent years, it has been observed that OSA is amongst one of the disorders with rising prevalence ranging from 9% to 38% Although the Indian prevalence was seen to be slightly on the lesser side, around 13.7%[7]. Interestingly, the prevalence has been recorded as way higher in men as compared to women[8]

One or multiple airway levels are associated with OSA to cause a reduced flow of air that originates from either an anatomical and/or functional reduction in the size of the airway lumen. Sometimes, this narrowed lumen is significant enough to create a turbulent airflow, then in that case airway obstruction is manifested as respiratory noises (e. g. stridor, wheezing), along with variable degrees of respiratory distress.[9]

The involvement of the nasal region in OSA will result in switch from nasal to mouth breathing due to increased nasal airway resistance and also due to nasal ventilatory reflex being impaired, that will reduce spontaneous ventilation. Breathing from the mouth results in narrowing of the pharyngeal lumen, reduced retroglossal diameter because of posterior displacement of the tongue and increased length of soft palate.

The apneic episodes have an effect on the oxygen saturation levels of blood that in the long-term impact vital systems like cardiovascular, pulmonary, and neurocognitive systems.[10] OSA is closely seen to be associated with cardiovascular diseases and HTN.

Furthermore, the number of cases that are identified as OSA can be said to be only the tip of an iceberg as the number of undiagnosed cases of the condition are far more in number.

Other than this, patients with OSA show impairments in behavior, cognition, and physical skills. The diagnosis of OSA is of prime importance. Talking about the modalities of diagnosing OSA, Polysomnography (PSG) has been recognized as the current gold standard for diagnosis of obstructive sleep apnea.

Keeping that in mind other diagnostic modalities like diagnostic nasal endoscopy and video laryngoscopy are also considered which involve a less complicated approach as compared to polysomnography.

METHODS:

STUDY SUBJECTS

50 confirmed cases with the signs and symptoms suggestive of upper airway obstruction coming to the Department of ENT, Maharaja Yashwantrao Holkar Hospital, Indore, were selected. After observing required symptoms , patients were evaluated for risk factors. Diagnostic nasal endoscopy and Video laryngoscopy was done.

INCLUSION CRITERIA

- Patients giving consent for study.
- Age Group- Above 20-60 years.
- Patients with signs and symptoms suggestive of upper airway obstruction were included in this study.

EXCLUSION CRITERIA

- Patient not giving consent for study.
- Underlying respiratory disease, M.I patients or any underlying cardiac disease, cerebrovascular insufficiency.

ETHICAL APPROVAL

Detailed information about the study was provided for participants, and written, informed consent was obtained from each one.

PROCEDURE:

- Fifty patients of age group 20-60 years with the signs and symptoms suggestive of upper airway obstruction were included in the study.
- A detailed history was taken, and patients fulfilling the inclusion criteria of upper airway obstruction were taken in the study.
- The patient's history was followed by general physical, clinical otorhinolaryngological examination, carefully assessing the possible symptoms of upper airway obstruction.
- After observing required symptoms , patients were evaluated for risk factors.
- Diagnostic nasal endoscopy and Video laryngoscopy was done.
- Polysomnography was done.

RESULTS

During the study period, 50 patients were recruited to the study.

Present study mean age of the cases was 46.40±9.27 years. Out of 50 cases. 37(74.0%) cases were male and 13(26.0%) cases were female.

Difficulty in breathing at night was found to be the most common factor as seen in 56% of the study population. This increased the excessive day time sleepiness (67%) and poor concentration (47%). Snoring was found in 65% of the total study population.

56% of patients with deviated nasal septum and 42% patients with IT hypertrophy had symptoms of OSA. These were the most common clinical signs as seen on routine examination of the study population.

Abnormally large uvula was seen in 56% of the total patients and 96% of them had symptomatic OSA. Macroglossia was seen in 52% of the patients, and all of them had symptomatic OSA.

The most commonly observed risk factor as seen in our study is BMI, wherein 64% of our study population was above 30kg/m2 and had OSA. Our study also shows that patients with WHR>0.9 (88%) and neck circumference >17 inch (87%) had OSA.

In this study, 68% population were hypertensive and 62% population were diabetic. Among them 79.4% of the total hypertensive patients and 90% of the diabetic population had OSA. 16% of the study population with severe OSA had sedentary lifestyle.

DISCUSSION

In the present study mean age of the cases was 46.40±9.276 years. Minimum 20 years and range extends to a maximum of 59 years. It was observed in an earlier study by **Bixler et al**[11]that the occurrence of OSA increases around 55 years of age.

Out of 50 cases. 37 (74.0%) cases were male and 13 (26.0%) cases were female. Male to female ratio was 3:1.

Amongst 13 severe OSA, snoring was found in 24% (n=12) cases, excessive day time sleepiness in 22% (n=11), difficulty in breathing at night in 20% (n=10), poor concentration in 18% (n=9), mouth breathing in 20% (n=10). This was similar to studies done in the past. **Maimon et al**[12], who demonstrated an increased AHI severity as the frequency of snoring increased that ranged from 46.3 +/- 3.6 db in patients with AHI < 5 to 60.5 +/- 6.4 dB in those with AHI > 50.

On comparing the occurrence of mouth breathing with the occurrence of OSA in the subjects studied, it was observed that around 52% percent of the patients complained of mouth breathing while sleeping. It has been seen that oral breathing can induce obstructive sleep apnea (OSA) or make it worse by increasing airway collapse and nasal resistance. This increased incidence was also reported by **Gleeson et al**[13]

Nasal valve collapse was found in 4%(n=2) of the study population, deviated nasal septum was found in 72%(n=36), inferior turbinate hypertrophy found in 56% (n=28, nasal polyp was reported in 28%(n=14), nasal discharge in 26%(n=13), abnormal uvula in 68%(n=34), retrognathia 28%(n=14) , 52%(n=26) showed macroglossia and only 10% (n=5) showed tonsillar hypertrophy. It has been understood that major septal deviation (SD) can lead to severe nasal congestion, which, in turn, can

lead to sleep apnea. The study by **Yeom SW et al[14]**, reflect this association but is has not always been a consistent finding.
Moreover, it appears that IT hypertrophy is more of a contributing factor to the occurrence of OSA rather than the primary causes itself.
Macroglossia is also considered as one of the factors of the occurrence of the disease. Here, 30 percent subjects were found to have an enlarged tongue which showed a significant association. Also, it is worth mentioning the frequency of the occurrence of the condition increased with an increase in the disease severity. **Dahlqvist et al[15]** mentioned that a high tongue is more frequently associated with OSA and **Passos et al[16]**.
In this present study, another important finding was that the presence abnormal uvula was significantly associated with the occurrence of OSA. Around 32 percent of the patients with OSA also had abnormal uvula and the number of which increased with increasing severity of the disease. **Dahlqvist et al[15]** reported that a macroglossia, and a wide uvula were independent factors associated with OSA.
BMI of more than 30 was found in 72% (n=36) of the study population and BMI of less than 30 was found in 28% (n=14) of the population. While studying the occurrence of BMI in OSA patients, many studies in the past have demonstrated a positive correlation between the two. **Gabbay et al [17]** reported that the severity of OSA rose linearly in patients as their BMI increased.
26 out of 50 patients with WHR >0.9 had moderate to severe symptoms of OSA. This study also compared other anthropometrical parameters like waist hip ratio to the occurrence of OSA. **Seidell et al[18]** reported that WHR is probably a better indicator of the risk of OSA as opposed to obesity.
Neck circumference of more than 17 inches was found in 48% (n=24) of the study population, 87% of them had symptoms of OSA. While 52% (n=26) of the study population had neck circumference of less than 17 inches. **Ahbab et al[19]** reported that the mean neck circumference was higher in patients with severe OSA.
In this study, 68% population were hypertensive. Among them 79.4% of the total hypertensive patients had OSA. The link between OSA and systemic hypertension has been studied since a long time. In 1980, **Lugaresi et al[20]** associated systemic hypertension with snoring in the general population.
In this study, 62% patients had diabetes and 34% of them were put in moderate to severe OSAH. In the study by **Gontzas et al[21]** 31/2000 14 OSA patients and 11 obese control subjects Higher fasting glucose and insulin levels in OSA compared to obese control subjects. In **Meslier et al[22]**22/2003 cross-sectional study men with age range, 21–78 yr High prevalence of type 2 diabetes and impaired glucose tolerance in OSA.
This study also shows that as the sedentary life style increased the patients had severe symptoms of OSAH.
56% of people with deviated nasal septum and 42% patients with IT hypertrophy had symptoms of OSA. These were most common clinical signs as seen on routine examination of the study population. Abnormally large uvula was seen in 56% patients of total patients and 96% of them had symptoms of OSA. Macroglossia was seen in 52% of the patients and all of them had symptoms of OSA.52% of the patients were found to have lateral pharyngeal wall narrowing and 96.4% among them had OSA.

CONCLUSION

Obstructive sleep apnea with its rising prevalence is believed to be a condition that is easy to detect and diagnose, but this is not true for a significant portion of the population, who exhibit OSA related signs and symptoms.
Obstruction at certain anatomical levels contributes towards obstructive sleep apnea severity. Multi-level obstruction is more prevalent in obstructive sleep apnea and is associated with increased severity. Risk factors increases the index of suspicion and encourage physicians to embark on a diagnostic path to investigate possible OSA.
Physicians should follow a step-by-step approach to come to a clinical judgement for polysomnography in an individual patient. OSA was found to be more common in males above 40

years of age. The results indicates that snoring, difficulty in breathing at night were the most commonly found symptom in the study population.

Deviated nasal septum and inferior turbinate hypertrophy were the most commonly found signs. The addition of diagnostic nasal endoscopy and video laryngoscopy led to the diagnostic pathway and provided a valuable adjunct to the clinical assessment of upper airway collapse. Nasal endoscopy and video laryngoscopy offered better illumination, higher magnification, and the ability to move straight to diseased areas in addition to better visibility.

With these diagnostic advancement, we can better assess the soft palate, uvula, tonsils, adenoids, lateral pharyngeal wall, to understand the pathology behind OSA. Our study concludes that large uvula and lateral pharyngeal wall narrowing, macroglossia and polyps have a significant role in severity of OSA.

TABLES

TABLE :1 AGE DISTRIBUTION OF THE STUDY POPULATION

Age groups	Frequency	Percent
< = 40 years	15	30.0
> 40 years	35	70.0
Total	50	100.0

TABLE 2: GENDER DISTRIBUTION OF THE STUDY POPULATION

Gender	Frequency	Percent
Female	13	26.0
Male	37	74.0
Total	50	100.0

TABLE 3:CLINICAL SYMPTOMS IN THE STUDY POPULATION

SYMPTOMS		Non OSAH		OSA					
				Mild OSAH		Moderate OSAH		Severe OSAH	
		N	%	N	%	N	%	N	%
Snoring	Yes	2	4.0	8	16.0	11	22.0	12	24.0
	No	10	20.0	4	8.0	2	4.0	1	2.0
Mouth Breathing	Yes	3	6.0	10	20.0	9	18.0	10	20.0
	No	9	18.0	2	4.0	4	8.0	3	6.0
Recurrent sore throat	Yes	0	0.0	2	4.0	2	4.0	2	4.0
	No	12	24.0	10	20.0	11	22.0	11	22.0
Excessive Day time sleepiness	Yes	3	6.0	8	16.0	10	20.0	11	22.0
	No	9	18.0	4	8.0	3	6.0	2	4.0
Difficulty in breathing at night	Yes	9	18.0	9	18.0	11	22.0	10	20.0
	No	3	6.0	3	6.0	2	4.0	3	6.0
Poor concentration	Yes	5	10.0	2	4.0	10	20.0	9	18.0
	No	7	14.0	10	20.0	3	6.0	4	8.0
Nasal discharge/nasal obstruction	Yes	8	16.0	9	18.0	5	10.0	7	14.0
	No	4	8.0	3	6.0	8	16.0	6	12.0
Difficulty in swallowing	Yes	1	2.0	0	0.0	1	2.0	0	0.0
	No	11	22.0	12	24.0	12	24.0	13	26.0
	Yes	8	16.0	7	14.0	4	8.0	7	14.0

Morning headache	No	4	8.0	5	10.0	9	18.0	6	12.0

TABLE 4: CLINICAL SIGNS ON EXAMINATION IN THE STUDY POPULATION

SIGNS		Non OSAH		OSA					
				Mild OSAH		Moderate OSAH		Severe OSAH	
		N	%	N	%	N	%	N	%
Nasal valve collapse	Yes	0	0.0	1	2.0	0	0.0	1	2.0
	No	12	24.0	11	22.0	13	26.0	12	24.0
Deviated nasal Septum	Yes	8	16.0	10	20.0	8	16.0	10	20.0
	No	4	8.0	2	4.0	5	10.0	3	6.0
Inferior Turbinate Hypertrophy	Yes	7	14.0	9	18.0	8	16.0	4	8.0
	No	5	10.0	3	6.0	5	10.0	9	18.0
Ethmoidal polyp	Yes	2	4.0	6	12.0	9	18.0	11	22.0
	No	10	20.0	6	12.0	4	8.0	2	4.0
Nasal Discharge	Yes	2	4.0	3	6.0	6	12.0	2	4.0
	No	10	20.0	9	18.0	7	14.0	11	22.0
Abnormal Uvula	Yes	1	2.0	9	18.0	9	18.0	10	20.0
	No	11	22.0	3	6.0	4	8.0	3	6.0
Retrognathia	Yes	1	2.0	11	22.0	0	0.0	2	4.0
	No	11	22.0	1	2.0	13	26.0	11	22.0
Macroglossia	Yes	0	0.0	6	12.0	9	18.0	11	22.0
	No	12	24.0	6	12.0	4	8.0	2	4.0
Tonsillar hypertrophy grade 3 grade 4	Yes	0	0.0	2	4.0	1	2.0	2	4.0
	No	12	24.0	10	20.0	12	24.0	11	22.0

TABLE 5: RISK FACTORS PREVALENT IN THE STUDY POPULATION

RISK FACTOR		Non OSA		OSA					
				Mild OSAH		Moderate OSAH		Severe OSAH	
		N	%	N	%	N	%	N	%
BMI (30kg/m2)	<=30kg/m2	8	16.0	3	6.0	2	4.0	1	2.0
	> 30kg/m2	4	8.0	9	18.0	11	22.0	12	24.0
Waist to hip Ratio	<=0.9	9	18.0	9	18.0	3	6.0	3	6.0
	> 0.9	3	6.0	3	6.0	10	20.0	10	20.0
Neck	<= 17 inch	9	18.0	11	22.0	3	6.0	3	6.0
	> 17 inch	3	6.0	1	2.0	10	20.0	10	20.0
HTN	Yes	7	14.0	7	14.0	10	20.0	10	20.0
	No	5	10.0	5	10.0	3	6.0	3	6.0
DM	Yes	3	6.0	9	18.0	9	18.0	10	20.0
	No	9	18.0	3	6.0	4	8.0	3	6.0
Sedentary Life style	Yes	1	2.0	8	16.0	9	18.0	8	16.0
	No	11	22.0	4	8.0	4	8.0	5	10.0

Table 6: DNE and VLS findings in the study population

		Non OSAH		OSA					
				Mild OSAH		Moderate OSAH		Severe OSAH	
		N	%	N	%	N	%	N	%
Deviated nasal Septum	Yes	8	16.0	10	20.0	8	16.0	10	20.0
	No	4	8.0	2	4.0	5	10.0	3	6.0
Inferior Turbinate Hypertrophy	Yes	7	14.0	9	18.0	8	16.0	4	8.0
	No	5	10.0	3	6.0	5	10.0	9	18.0
Ethmoidal polyp	Yes	2	4.0	6	12.0	9	18.0	11	22.0
	No	10	20.0	6	12.0	4	8.0	2	4.0
Adenoids Enlargement	Yes	0	0.0	1	2.0	0	0.0	1	2.0
	No	12	24.0	11	22.0	13	26.0	12	24.0
Macroglossia	Yes	0	0.0	6	12.0	9	18.0	11	22.0
	No	12	24.0	6	12.0	4	8.0	2	4.0
Low lying soft palate	Yes	0	0.0	0	0.0	2	4.0	0	0.0
	No	12	24.0	12	24.0	11	22.0	13	26.0
Large Uvula	Yes	1	2.0	9	18.0	9	18.0	10	20.0
	No	11	22.0	3	6.0	4	8.0	3	6.0
Tonsillar Hypertrophy	Yes	1	2.0	2	4.0	1	2.0	1	2.0
	No	11	22.0	10	20.0	12	24.0	12	24.0
Lateral Pharyngeal wall narrowing	Yes	1	2.0	5	10.0	10	20.0	10	20.0
	No	11	22.0	7	14.0	3	6.0	3	6.0

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