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Original Research Paper

General Medicine

A STUDY OF OBSTRUCTIVE SLEEP APNOEA AND ITS CORELATION WITH OTHER DISEASES

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ABSTRACT

Background To study the anthropometric parameter in patient of obstructive sleep apnoea and to find out its association with associated comorbities.

Methods 30 patients of OSA were previewed by polysomnographic study from the clinically suspected OSA patient by applying STOP BANG criteria with score of >5, and equal number of age and sex match control subjects coming to OPD/IPD were studied.

Results Majority of patients belong to middle age group. Snoring was the most important symptom reported by the patients followed by breathing pause (96.7%), day time tiredness (93.3%) and systemic hypertension (40%).Certain co-morbid condition like hypertension was present in (40%), hypothyroidism (10%) and 3.3% each were having diabetes and epilepsy.

Conclusion Sleep apnea is a common disorder that if not recognized and treated leads to significant morbidity and increased mortality. Early recognition and treatment of OSA should be our aim. Treatment of OSA may represent a novel target to improve health outcome.

KEYWORDS : Obstructive sleep apnoea ; Stop bang criteria ; Snoring

INTRODUCTION

Obstructive sleep apnoea is defined as a syndrome that is characterized by the occurrence of repetitive episode of complete or partial upper airway obstruction during sleep, these episodes usually occur in association with loud snoring and daytime sleepiness. The subtle consequences of OSA include change in cognitive performance viz., difficulty with working memory, slowing of response across the duration of the task, declines in the best effort or fast response, lapses and false responses. OSA is common worldwide and a higher than normal risk or prevalence of cardiovascular diseases has been observed in OSA population.

A number of studies demonstrated the effect of age on the development of OSA, independent of other confounding factors, data addressing the age related absolute change in disease severity are deficient. Some authors claim limited whilst, others suggest marked deterioration in disease severity as much as doubling after 10 years in a given OSA population (5).

Like the increasing age, weight gain has also been shown to be a strong predictor of worsening OSA (6). Increased prevalence of OSA is seen in patients with congestive cardiac failure (40%), stroke (60%) and end stage renal failure (50%) (7).Obese individuals have nearly four time higher risk of having OSA as compared to non obese individual independent of age and gender (1). Syndrome Z is defined as co-occurrence of OSA and metabolic syndrome. The estimated population prevalence of syndrome Z in North India is about 4.5 percent (95% CI 3.7-5.3) and age, male gender, amount of body fat and nocturnal desaturation were found as independent risk factors for it (12).

The combination of male gender, waist-hip ratio and neck circumference has been incorporated in a diagnostic model for predicting OSA and screening subjects for PSG. Males have a 10 fold higher risk of having OSA. Earlier studies have consistently reported a higher prevalence of OSA in men. In fact, ratio as high as 10:1 in favour of men have been reported while studying sleep apnoea in clinical settings (15). There are several reasons for the observed differences in the prevalence between men and women. These include, but not limited to differences in the distribution of adipose tissue, upper airway anatomy and muscle function, control of ventilation, and the effects of sex hormones and leptin (16).

MATERIALS AND METHODS

The present study was carried out in the Department of Medicine Department of General Medicine, Govt. Medical College ; Ratlam on 30 OSA patients and equal controls over a period of 12 months. The objective was to study the anthropometric parameter in patients of obstructive sleep apnoea to find association between anthropometric data and OSA and also to find out association between severity of OSA and left ventricular end systolic/diastolic volume and pulmonary artery pressure.

30 patients of OSA were previewed by polysomnographic study from the clinically suspected OSA patient by applying STOP BANG criteria with score of >5, and equal number of age and sex match control subjects coming to Medicine OPD/IPD were studied. After detailed clinical history and anthropometric examination, they were subjected to biochemical, haematological and echo-cardiological examination.

OBSERVATIONS

The mean age of patients was 52.23 yrs while it was 54.40 in controls which was statistically not significant. Among OSA group, 83.3% were male and 16.7% were female, 63.3% male and 36.7% female were there in control group, showing male predominant. OSA was common in male.

Table 1 - Anthropometric measurements of OSA patients and controls

Anthropometric Measure	Case (n=30)	Control (n=30)	P value
Height [cm] Mean (SD)	161.43±7.21	157.47±7.48	0.04
Weight [kg] Mean (SD)	84.87±12.08	63.43±7.82	<0.001
BMI [kg/m^2] Mean (SD)	32.07±4.16	25.23±3.39	<0.001

Table shows that OSA patients had higher BMI, they were taller and heavier as compared to controls. The weight and BMI was significantly higher than controls (p=0.001). The mean height of cases was 161.43 cm and controls was 157.47 cm the difference was significant (P<0.05).

Table 2 - Laboratory parameters of OSA patients and controls

Laboratory Parameter	Case (n=30)	Control (n=30)	P
Hb [g/dl] Mean (SD)	11.53±1.27	11.12±1.00	0.17
TLC [/cu mm] Mean (SD)	7050.03±1363.72	7198.87±1183.74	0.65
MCV [fl] Mean (SD)	83.82±5.89	86.12±4.76	0.10
PCV [%] Mean (SD)	44.76±15.66	38.74±5.22	0.05
MCH [pg] Mean (SD)	31.91±9.35	29.41±1.93	0.15
MCHC [%] Mean (SD)	32.36±2.19	33.34±1.83	0.06
Random blood sugar [mg%] Mean (SD)	104.9±24.04	95.80±8.31	0.05

Table shows various laboratory values in OSA patients and controls. Both the groups were comparable with regards to laboratory parameters. The mean values of Hb and Total leukocyte count in our study was 11.53 g/dl and 7050 /cumm in cases which was comparable with the controls. Mean MCV in cases was 83.82 fl while that in control was 86.12 fl. Values of PCV, MCH and MCHC in cases were 44.76 %, 31.91 pg and 32.36 %, while that of in controls were 38.74%, 29.41 pg and 33.34% respectively. Except PCV which was significantly higher in OSA group (P=0.05), other lab parameters were not significant. This significance could be due to hemo-concentration or polycythaemia which might be due to secondary hypoxemia. The random blood sugar was on the higher side could be due to the patient who were obese and were having higher BMI.

Table 3 - Stop Bang Criteria for diagnosis of OSA patients and controls

Criteria	CASE (n=30)	CONTROL (n=30)	Statistical value	P
Snoring reported by [%]	30 (100%)	23 (76.7%)	7.92	0.005
Day time tiredness reported by [%]	28 (93.3%)	6 (20%)	32.85	<0.001
Breathing pause observed in [%]	29 (96.7%)	6 (20%)	36.27	<0.001
Systemic hypertension present in [%]	12 (40%)	2 (6.7%)	9.31	0.002
BMI [kg/m2]	32.07	25.23	6.97	<0.001
Age [years]	52.23	54.50	-0.78	0.43
Neck Circumference [cm]	42.40	35.10	8.96	<0.001
Gender Male	25(83.3%)	19(63.3%)	3.06	0.08

Table shows that OSA patients had statistically significant difference as compared to controls in snoring, day time tiredness, breathing pauses and BMI (P=<0.05). They were taller, heavier and had larger neck circumference as compared to controls.

Table 4- Comorbid illness in OSA patients and controls (n=30)

Comorbidity	Case present (%)	Control present (%)	Chi-square test	P value
Cardiac (Hypertension)	12 (40%)	2 (6.7%)	9.3	0.002
Endocrine (Hypothyroidism/ Diabetes)	4 (13.3%)	4 (13.3%)	5.0	0.28
Neurology (Epilepsy)	1 (3.5%)	0	1.01	0.31

Table shows that 40% were hypertensive in OSA patients and 6.7% in controls, thus hypertension was significantly higher in OSA patients than control. Hypothyroidism was found in 10% of OSA patients and in 3% of controls. Diabetes mellitus was found in 3% of OSA patients and in 10% of control. Only one patient of OSA had epilepsy.

RESULTS

1. Out of 30 patients 25 were male and 5 were female. The mean age of OSA patients was 52.23±10.1 years and control 54.50±12.1 years. The youngest patient was 36 year old and oldest 72 year. Majority of patients belong to middle age group.
2. Majority of patients (87%) belonged to middle socio-economic group, and a negligible number to the higher socio-economic group.
3. Snoring was the most important symptom reported by the patients followed by breathing pause (96.7%), day time tiredness (93.3%) and systemic hypertension (40%).
4. Certain co-morbid condition like hypertension was present in (40%), hypothyroidism (10%) and 3.3% each were having diabetes and epilepsy.
5. The mean height of OSA patients (161.43±7.21cm) as compared to control (157.47±7.48 cm), mean weight was significant (p=0.001), (84.87±12.08 kg) as compared to control (63.43±7.82 kg), mean BMI (32.07±4.16 kg/m^2) significant (p=0.001) more than control (25.23±3.39kg/m^2) and mean neck circumference (42.40±3.09 cm) was also significant (p=0.001) more than the control (35.10±2.49cm) respectively.
6. There was no significant difference in haematological, biochemical and serum lipid level between study patients and control, except VLDL level which was significantly (51.70±24.55 mg/dl) more in OSA patients.
7. Above observation of OSA patients shows that there are significant cardiovascular functional abnormality making them prone to cardiac morbidity and mortality as compared to normal healthy controls of same age and sex. Thus appropriate measures are required at all level not only to identify such high risk patients of OSA but also to take appropriate action.

STATISTICAL ANALYSIS

Data collected from both the groups will be cleared, sorted and entered in to Microsoft EXCEL. After data entry, various statistical analyses will be done.SPSS (version 17.0, Chicago, IL, USA) was used for statistical analysis and continuous variables were noted as mean ± standard deviation, VAS as mean ± standard error and analyzed using ANOVA. Categorical variables were noted in number of patients (%) and analyzed using chi-squared and Fisher's exact test. A P value of < 0.05 was considered statistically significant and P value of < 0.01 was highly significant were taken

DISCUSSION

This study had shown that subjects with severe OSA were heavier, had higher body mass index and a larger neck circumference as compared to controls. A male predominance was seen in the OSA patients (83.3%). This finding has been supported by a number of earlier studies that had shown that OSA was more prevalent among males. The male predominance could be related to a number of factors. It has been hypothesized that males have larger tongue, longer soft palate, and higher abdominal girth as compared to females. All these are known risk factors for OSA. Such anatomical factors contribute to reduction in the upper airway space and compliance of chest. Hence, these factors predispose the males for the OSA.

Higher BMI suggests more fat in the body and this fat gets deposited in a number of areas in the body. These areas include abdominal cavity in males, gluteal and sub-cutaneus tissue in females and also the parapharyngeal fat pads. Amount of fat in parapharyngeal region is related to the neck circumference and hence, larger neck is associated with high risk for OSA. However, contradictory studies are also available that did not find any difference between the OSA and controls on these measures . This is worth mentioning here that OSA depends upon a number of other factors that regulate the pharyngeal airway patency. Besides anatomical factors mentioned so far, that influence the patency of upper airway, other physiological factors like central chemo-sensitivity, tone of the pharyngeal dilator muscles, chest wall compliance, tracheal tug also affect the chances of development of OSA. In addition, craniofacial anatomy plays a major role in development of OSA. Position of maxilla and mandible relative to the posterior pharyngeal wall, nasal airway structure also decide the calibre of upper airway and any deviation from normal anatomy may cause OSA. Since, occurrence of these factors is independent of BMI, OSA can be found in subjects with low BMI.

In present study, we found that serum creatinine was higher in OSA patients. In present study, the blood sample was drawn in early morning after the overnight study. Since these patients had sleep apnea in the previous night, a possibility of relative dehydration could not be ruled out as many of these patients are mouth breathers. However, values in both the groups were within reference range as shown in earlier studie. Fasting blood sugar was comparable between two groups. Similar findings have been reported earlier.

In present study, we found that OSA patients had low HDL and increased VLDL as compared to controls. Low HDL in OSA patients could be related to metabolic syndrome that is an integral part of

the OSA pathology. The repetitive episodes of upper airway obstruction that are characteristic of OSA, result in intermittent hypoxia and large swings in intra-thoracic pressure that in turn trigger autonomic responses, and sympathetic over activity in patients with OSA. There is a direct link between the adrenergic system and lipid levels. The chronic elevated sympathetic activity in OSA patients may lower HDL and increase serum TG levels. Borgel et al demonstrated an influence of OSA on HDL levels.

In their study, an independent association was found between the change in AHI and the change in HDL and triglycerides respectively. Can et al found that OSA was associated with increased lipid levels. Total cholesterol, LDL and TGs values were increased in patients with OSA compared with controls. Iesato et al reported that circulating lipoprotein lipase concentrations were lower in OSA patients compared with those in non-OSA patients. Tan et al demonstrated that OSA subjects had greater degree of HDL dysfunction and increased oxidized LDL levels compared with controls. These studies found that AHI was the main determinant of HDL dysfunction in OSA patients.

In present study there was no difference of haematological parameter such as haemoglobin, total leukocyte count, MCV, MCH, MCHC between OSA patients and healthy control. PCV was significantly higher in OSA patients. This significance could be due to hemo-concentration or polycythaemia which might be due to secondary hypoxemia.

In this study, all the OSA subjects complained of snoring, 93% reported day time tiredness, breathing pauses were observed by bed partners of 97% subjects and 40% OSA patients were hypertensive. However, the daytime systolic and diastolic blood pressure was comparable between groups. Usui Y et al demonstrated that systolic blood pressure and diastolic blood pressure was not different between normal subjects and severe OSA patients. Lee et al demonstrated that increased systolic and diastolic blood pressure in OSA patients compared with control, but in this study the number of healthy control will be less as compared with OSA patients.

CONCLUTION

We have demonstrated a very high prevalence of cardiovascular risk factor such as obesity, HTN, DM and hyperlipidemia in OSA patients. The available evidence supports higher incidence of heart failure, CAD, hypertension, and stroke in OSA subjects. OSA also causes cardiac remodeling i.e. LVH and LA enlargement, which may have arrhythmic potential such as high incidence of AF in OSA.

Our observation in OSA patients shows that there are significant cardiovascular functional abnormality making them prone to cardiac morbidity and mortality as compared to normal healthy controls of same age and sex. Thus appropriate measures are required at all levels not only to identify such high risk patients of OSA but also to take appropriate action.

Sleep apnea is a common disorder that if not recognized and treated leads to significant morbidity and increased mortality. Early recognition and treatment of OSA may improve cardiovascular function. Treatment of OSA may represent a novel target to improve cardiovascular health outcome.

REFERENCES

1. Guilleminault C. Obstructive Sleep Apnoea: The Clinical Syndrome and Historical Perspective. Med Clin N America. 1985; 69: 1187-1203.
2. Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. Lancet. 2009; 373: 82-93.
3. Sharma SK, Kumpawat S, Banga A, Goel A. Prevalence and risk factors of obstructive sleep apnoea syndrome in a population of Delhi, India. Chest. 2006; 130: 149-56.
4. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med. 1993; 328: 1230-35.
5. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. N Engl J Med. 2005; 353: 2034-41.
6. Udwadia ZF, Doshi AV, L onkar SG, Singh CI. Prevalence of sleep disordered breathing and sleep apnoea in middle-aged urban Indian men. Am J Respire Crit Care Med. 2004; 169: 168-73.
7. Ip MS, Lam B, Chan L-Y. Circulating nitric oxide is suppressed in obstructive sleep apnea and is reversed by nasal continuous positive airway pressure. Am. J. Respire. Crit. Care Med. 2000; 162: 2166-71.
8. Phillips BG, Narkiewicz K, Pesek CA, Haynes WG, Dyken ME, Somers VK. Effects of obstructive sleep apnea on endothelin-1 and blood pressure. J Hypertension. 1999; 17: 61-6.
9. Kapsimalis F, Kryger MH. Gender and obstructive sleep apnea syndrome: part 2 Mechanism of Sleep. 2002; 25: 499-506.
10. Young T, Palta M, Dempsey J. The occurrence of sleep disordered breathing among middle aged adults. N Engl J Med. 1993; 328: 1230-35.
11. Shanoudy H, Soliman A, Raggi P. Prevalence of patent foramen ovale and its contribution to hypoxemia in patients with obstructive sleep Fapnea. Chest.1998; 113: 91-6.
12. Barbe F, Pericas J, Munoz A. Automobile accidents in patients with sleep apnea syndrome. Am J Respire Crit Med. 1998; 158: 18-22.
13. Teran SJ, Jimenez Gomez A, Cordero J. The association between sleep apnea and the risk of traffic accidents. Cooperative Group Burgos Santander. N Engl J Med. 1999; 340: 847-51.
14. Otilia N, Graur IL, Popescu SD, Popa DA, Mihalache L. Anthropometric predictors of high risk of obstructive sleep apnea syndrome in a rural population. Rom J Diabetes Nut Dis. 19(1): 07-16.
15. Hoffstein V, Mateika S. Differences in abdominal and neck circumferences in patients with and without obstructive sleep apnea. Eur Respire J. 1992; 5: 377-81.
16. Otilia N, Graur IL, Popescu SD, Popa DA, Mihalache L. Anthropometric predictors of high risk of obstructive sleep apnea syndrome in a rural population. Rom J Diabetes Nutr Dis. 19(1): 07-16.
17. Vgontzas AN, Bixler EO, Chrousos GP. Metabolic disturbances in obesity versus sleep apnea: the importance of visceral obesity in insulin resistance. J Intern Med. 2003; 254: 32-44.
18. Friedman M, Tanyeri H, La RM. Clinical predictors of obstructive sleep apnea. Laryngoscope. 1999; 109: 1901-07.
19. Deegan PC, McNicholas WT. Predictive value of clinical feature for the obstructive sleep apnea syndrome. Eur Respire J. 1996; 9: 117-24.
20. Zonato AI, Bittencourt LRA, Martinho FL. Association of systematic head and neck physical examination with severity of obstructive sleep apnea-hypopnea syndrome. Laryngoscope. 2003; 113: 973-80.
21. Choi HJ, Miyazaki S, Okawa M, Kim JE, Lee BJ, Shin C, Lee HS. Clinical implications of mandible and neck measurements in non obese Asian snorers. Clinical and Experimental Otorhinolaryngology. 2011; 4: 40-3.
22. Patwardhan AA, Larson MG, Levy D, Benjamin EJ, Leip EP, Keyes MJ. Obstructive sleep apnea and plasma natriuretic peptide level in a community based sample. Sleep. 2006; 29: 1301-06.
23. Vgontzas AN, Tan TL, Bixler EO, Martin LF, Shubert K, Kales A. Sleep apnea and sleep disruption in obese patients. Arch Intern Med. 1994; 154: 1705-11.
24. Becker HF, Piper AJ, Flynn WE. Breathing during sleep in patients with nocturnal desaturation. Am J Respire Crit Care Med. 1999; 159(1): 112–18.
25. Chaouat A, Weitzenblum E, Krieger J, et al. Association of chronic obstructive pulmonary disease and sleep apnea syndrome. Am J Respire Crit Care Med. 1995; 151(1): 82–6.
26. Chung F, Subramanyam R, Liao P, Sasaki E, Shapiro C, Sun Y. High STOP-Bang score indicates a high probability of obstructive sleep apnoea. Br J Anaesth. 2012 May; 108(5): 768–775.
27. Borgel J, Sanner BM, Bittlinsky A, et al. Obstructive sleep apnoea and its therapy influence high-density lipoprotein cholesterol serum Levels. Eur Respire J. 2006; 27: 121-7.
28. Iesato K, Tatsumi K, Saibara T, et al. Decreased lipoprotein lipase in obstructive sleep apnea syndrome. Circ J. 2007; 71: 1293-8.