Original Article

A comparative study on the clinical and polysomnographic pattern of obstructive sleep apnea among obese and non-obese subjects

Rajiv Garg, Abhijeet Singh, Rajendra Prasad, S. Saheer, Jabeed P., Ramkishun Verma

Department of Pulmonary Medicine, CSM Medical University, UP, Lucknow, India

Address for correspondence:
Dr. Rajiv Garg, Department of Pulmonary Medicine, CSM Medical University, Lucknow - 226 003, UP, India.
E-mail: rajivkgmc@gmail.com

Submission: 04-06-11
Accepted: 14-08-11

Abstract:

OBJECTIVE: This study was designed to compare the pattern of obstructive sleep apnea (OSA) among obese and nonobese subjects regarding clinical and polysomnographic data obtained for a polysomnographic study.

METHODS: A cross-sectional retrospective descriptive study was conducted by analyzing polysomnographic data in 112 consecutive patients underwent a sleep study at our sleep laboratory from January 2009 to July 2010. Out of them, 81 were diagnosed to have OSA (apnea-hypopnoea Index ≥ 5). These patients were classified in two groups with body mass index (BMI) < 27.5 kg/m^2 as nonobese and BMI $\geq$ 27.5 kg/m^2 as obese. Clinical as well as polysomnographic data were evaluated and compared between the two groups. Patients were also evaluated for other risk factors such as smoking, alcoholism, and use of sedatives. Data were subjected to statistical analysis (χ^2-test, *P* value <0.05 considered to be significant). The Fisher Exact test was applied wherever the expected frequency for a variable was ≤ 5.

RESULTS: Of 81 patients with OSA, 36 (44.4%) were nonobese with a mean BMI of 26.62 ± 2.29 kg/m^2 and 45 (55.6%) were obese with a mean BMI of 35.14 ± 3.74 kg/m^2. Mean AHI per hour was significantly more in the obese than in the nonobese group (50.09 ± 29.49 vs. 24.36 ± 12.17, *P*<0.001). The use of one or more sedatives was more in nonobese as compared to obese (58.3% vs. 24.4%, *P*=0.002). The obese group had significantly higher desaturation and arousal index (*P*<0.001). The minimal oxygen saturation was lower in the obese than the nonobese group (68.5 ± 13.00 vs. 80.3 ± 7.40, *P*<0.001) and was well below 90% in both groups. Overall, the OSA in nonobese patients was mild-to-moderate as compared to that of the obese and no significant differences were observed between them as regard to age, gender, mean neck circumference, excessive daytime sleepiness, adenoid or tonsillar enlargement, smoking, and remaining polysomnographic parameters.

CONCLUSION: Obstructive sleep apnea can occur in nonobese persons though with less severity as compared to obese leading to a concept that OSA is not restricted to obese persons only and there is a high demand of its awareness regarding evaluation, diagnosis, and management in such individuals.

Key words:

Body mass index, obesity, obstructive sleep apnea

Access this article online

Quick Response Code:

Website: www.thoracicmedicine.org

DOI: 10.4103/1817-1737.91561

Obstructive sleep apnea (OSA) is a disease that is characterized by disruptive snoring, repeated episodes of complete or partial pharyngeal obstruction during sleep resulting in nocturnal hypoxemia, frequent arousals during sleep, and excessive daytime sleepiness (EDS).[1] It is a serious and potentially life-threatening disorder that is far more common than generally believed. Several risk factors, including obesity, male sex, age, and heritable factors, have been associated with an increased prevalence of obstructive sleep apnea in the general population. Obesity has been considered to be one of the classical risk factor for OSA.[2] It has become a major health problem worldwide with an increasing prevalence and associated with high comorbidities such as diabetes mellitus, hypertension, cardiovascular disease, dyslipidemia, malignancies, and overall mortality. Results of various studies indicate that approximately 60–70% of patients with OSA are obese.[3-16] The data regarding association of OSA among nonobese patients is scarce and needs great effort as fewer studies have reported magnitude of association.[17,18] The occurrence of OSA in nonobese patients may exhibit different characteristics than obese patients with OSA. Therefore, this study was designed to study the pattern of OSA among nonobese Indian subjects coming for the polysomnographic study.

Methods

Study population

This cross-sectional retrospective study was done by reviewing data from 81 OSA patients consecutively referred to the sleep laboratory of Department of Pulmonary Medicine, Chhatrapati

Shahuji Maharaj Medical University, Lucknow, between August 2009 and July 2010. This study was conducted after approval by the local research ethics committee. OSA was diagnosed if there was apnea hypopnoea index (AHI)>5 events/h combined with habitual snoring or EDS. The patients were classified into nonobese (n=36) and obese (n=45) groups if their body mass index (BMI) was <27.5 and ≥27.5 kg/m^2, respectively, as per the WHO criteria of obesity for Asians.[19] Clinical and polysomnographic data were evaluated and compared between the two groups. Clinical evaluation was done with the help of sleep questionnaire by recording age, sex, co-morbid illnesses, EDS by Epworth sleepiness scale (ESS), and anthropometric measurements (BMI, neck circumference, thyromental distance, and Mallampati's score). Patients were also evaluated for other risk factors such as smoking, alcohol, and the use of sedative or tranquilizers. Neck circumference was measured with a tape measure at the level of cricothyroid membrane. Thyromental distance was measured horizontally from the thyroid prominence to a perpendicular dropped from the soft tissue mentum. Mallampati's score was measured with mouth wide open, tongue maximally protruded without phonation, and classified into Grades I–IV. However, subjects having age >80 years, tuberculosis, chronic obstructive pulmonary disease (COPD), pregnancy, uncontrolled congestive heart failure (CHF), chronic renal failure (CRF), and neurological disorders were excluded from the study.

Polysomnography

All patients with OSA underwent a sleep study consisting of an overnight polysomnographic examination, which included an EEG (C3-A2, C4-A1, O2-A1, and O3-A2), bilateral electrooculogram, chin and lower leg electromyogram, nasal and mouth airflow, thoracic and abdominal respiratory movements, ECG, oxygen saturation measured by finger oximetry, and body position. For this study, S-7000 computerized polysomnography machine (manufactured in the year 2007 by Cogent Technologies EMBLA System Inc., UK) with 20 channel inputs was used. Sleep staging was done with the help of Somnologica Studio software and classified into Awake, nonrapid eye movement (NREM) sleep with Stages I, II, III and IV, and rapid eye movement (REM) sleep. The episodes of apnea were defined as complete cessation of airflow for ≥10 s, and hypopnea consisted of a ≥50% reduction in oronasal airflow accompanied by a reduction in oxygen saturation measured by pulse oximetry of at least 4%. Apnea events were classified as obstructive, mixed, or central, according to the presence or absence of breathing efforts with thoracoabdominal paradox. AHI was determined by the frequency of these events per hour during sleep time based on the results of the overnight polysomnography. Polysomnographic data, including respiratory arousal index, minimal oxygen saturation, total sleep time, and desaturation index, were also collected. Sleep data recorded by the computer was cross checked manually for scoring of sleep stages apneas and hypopnoeas regarding each subject.

Statistical analysis

Numerical and categorical data were compared between groups using the Student t-test and χ^2-test as appropriate. All statistical analyses were performed using statistical software SPSS version 10.0 (SPSS Inc, Chicago, USA). A P value of <0.05 was considered significant.

Results

There were 81 patients of OSA, among them 36 (44.4%) were non-obese and 45 (55.6%) were obese with a mean BMI of 26.62 ± 2.29 and 35.14 ± 3.74 kg/m^2, respectively. No significant difference was found in mean age, gender, mean neck circumference, Mallampati grading, ESS, adenoid or tonsillar enlargement, smoking, and other co-morbid conditions. More patients with smoking, alcohol use, snoring, diabetes, and hypertension were found in the obese group [Table 1]. The hypertension was significantly higher in the obese (57.8% vs. 33.3%, P=0.03) than the non-obese patients. The use of sedatives was more common in the non-obese patients than the obese patients (58.3% vs. 24.4%, P=0.002). The nonobese group were also having significantly shorter thyromental distance than the obese group (48.41 ± 10.61 mm vs. 60.54 ± 12.24 mm, P<0.001). Mean AHI was significantly less in the non-obese than in the obese (24.36 ± 12.17/h vs. 50.09 ± 29.49/h, P<0.001). Most of the non-obese patients were having mild-to-moderate severity OSA, as 26 (72.2%) of them were having AHI ≤30/h as compared to only 14 (31.1%) obese patients. The obese group had a significance with regard to lower minimal oxygen saturation (68.47 ± 13.00 vs. 80.25 ± 7.40, P<0.001), higher average desaturation index (48.32 ± 13.08 vs. 30.63 ± 15.63, P<0.001), and higher arousal index (28.42 ± 4.99 mm vs. 17.84 ± 5.07 mm, P<0.001). Although there were more number of obese patients than nonobese (25/45 vs. 14/36) having minimum oxygen saturation <90%, but the percentage of nonobese patients showing similar findings were not less (55.6 vs. 38.9, P=0.37). Rest of the polysomnographic parameters were comparable. The data are summarized in Table 2.

Discussion

Obese patients with OSA have been observed to show significant severity than the nonobese OSA patients in terms of various

Table 1: Demographic and clinical characteristics in the non-obese and obese patients with OSA

	Non-obese (n=36) (%)	Obese (n=45) (%)	P value
Mean age (years)	52.88 ± 7.19	49.76 ± 10.06	0.12
Sex (male/female)	27/18	23/13	0.37
Mean BMI (kg/m^2)	26.62 ± 2.29	35.14 ± 3.74	0.12
Mean neck circumference (Inches)	15.81 ± 1.71	16.10 ± 1.78	0.46
Mean ESS score	11.50 ± 3.27	13.07 ± 5.60	0.45
Thyromental distance (mm)	48.41 ± 10.61	60.54 ± 12.24	<0.001
Mallampati score			0.44
Class I	10 (27.8)	8 (17.8)	
Class II	12 (33.3)	13 (28.9)	
Class III	8 (22.2)	10 (22.2)	
Class IV	6 (16.7)	14 (31.1)	
Tonsillar enlargement	9 (25.0)	12 (26.7)	0.87
Habitual snoring	33 (91.7)	41 (91.1)	0.93
Smoking	9 (25.0)	16 (35.6)	0.31
Alcohol	13 (36.1)	18 (40.0)	0.72
Sedatives	21 (58.3)	11 (24.4)	0.002
Hypertension	12 (33.3)	26 (57.8)	0.03
Diabetes mellitus	7 (19.4)	13 (28.9)	0.33

OSA = Obstructive sleep apnea, BMI = Body mass index; ESS = Epworth sleepiness scale

Table 2: Polysomnographic characteristics in the non-obese and obese patients with OSA

	Non-obese (*n*=36) (%)	Obese (*n*=45) (%)	*P* value
Total sleep period	539.46 ± 54.78	542.59 ± 42.12	0.77
TST	333.59 ± 78.36	313.45 ± 58.83	0.19
Total wake time	212.12 ± 92.46	229.14 ± 73.86	0.36
AHI/h	24.36 ± 12.17	50.09 ± 29.49	<0.001
Sleep stage 1 (%TST)	23.23 ± 20.61	23.37 ± 16.73	0.97
Sleep stage 2 (%TST)	20.87 ± 20.57	19.18 ± 16.20	0.68
Sleep stage 3 (%TST)	13.96 ± 10.13	17.58 ± 11.31	0.14
Sleep stage 4 (%TST)	21.44 ± 20.74	22.44 ± 15.39	0.80
REM stage (%TST)	20.37 ± 18.57	17.35 ± 19.20	0.48
Average oxygen saturation (%)	94.59 ± 2.40	89.68 ± 6.06	<0.001
Minimum oxygen saturation (%)	80.25 ± 7.40	68.47 ± 13.00	<0.001
Minimal oxygen saturation <90%	14 (38.9)	25 (55.6)	0.37
Arousal index (events/h)	17.84 ± 5.07	28.42 ± 4.99	<0.001
Desaturation index (events/h)	30.63 ± 15.63	48.32 ± 13.08	<0.001

OSA = Obstructive sleep apnea, TST = Total sleep time, REM = Rapid eye movement, AHI = Apnea hypopnea index

parameters such as AHI, average as well as minimal oxygen saturation, arousal and desaturation indices.[20-24] The present study has also shown similar findings and strikingly the minimal oxygen saturation was well below 90% in both the groups. The etiology of OSA in obese has been linked with anatomic alterations that predisposes to upper airway obstruction during sleep. Obesity seems to have two distinct mechanical influences on the pharyngeal airway collapsibility.[25] First, it increases soft tissue surrounding the pharyngeal airway within limited maxillo-mandibular closure occupying and narrowing its space (pharyngeal anatomical imbalance). It can be considered that elevations in neck circumference and increased deposition of peripharyngeal fat could narrow and compress the upper airway.[26,27] Furthermore, increased peripharyngeal fat has been correlated with increased sleep apnea severity.[28,29] Second, it increases visceral fat volume that decreases lung volumes such as functional residual capacity (FRC) and expiratory reserve volume (ERV) leading to increased pharyngeal wall collapsibility possibly through decreased longitudinal tracheal retraction. Thus, obesity imposes mechanical loads on both the upper airway and respiratory system that predispose to upper airway narrowing, collapse, and airflow obstruction during sleep. Neural compensation for functioning structural abnormalities operating during wakefulness is also lost during sleep. These effects may be mediated by circulating adipokines, which influence body fat distribution and central nervous system activity.[30]

In contrast, Sakakibara *et al*. observed that the etiology of OSA in nonobese patients appears to be somewhat different which includes bony structure discrepancies.[17] In accordance with this study, we observed shorter thyromental distance in the nonobese patients which signifies that the position of the chin is relatively low with reference to the thyroid cartilage and shorter length of the anterior cranial base.[31] A retrospective study from Thailand also reported similar findings in 71 nonobese patients.[18] Nonobese OSA patients tend to present the following anatomical craniofacial characteristics such as caudal hyoid, increased soft palate dimensions, and consequent anteroposterior reductions of the airways at the soft palate level, reduction of anteroposterior region of nasopharynx, and oral pharynx. Obese OSA patients can present with these findings but in addition they have increased volume of tongue and anterior hyoid bone. Lower and anterior position of hyoid bone in obese patients seems to be related to increased fat deposition on the tongue, which increases its volume.[32] It has been suggested that the discrepancy in these cephalometric measurements may also depend on sex, age, and race.[33-37] OSA in Asian men has been found more frequently in the nonobese patients, despite the presence of severe illness, when compared with white male patients with OSAS.[35]

The present nonobese subjects were more likely in habit of taking sedatives for sleeping as compared to obese counterpart which is in concordance with study conducted by Ghanem and Mahmood on 102 patients with OSA.[21] The incidence of hypertension was observed to be more in the obese OSA patients than the non-obese patients. This finding has been supported by several cross-sectional, longitudinal, and treatment studies.[38-42] Obesity and OSA are each very strongly associated with hypertension.[43] OSA is independently associated with hypertension, independent of obesity. Furthermore, obesity and OSA often co-exist and, in fact, one may be conducive to the other. It is therefore plausible that at least part of the association between obesity and hypertension is related to the presence of OSA and perhaps *vice versa*. The presence of OSA in the obese patients may further contribute to adverse cardiovascular outcomes including hypertension when compared with each condition in isolation.[44] OSA probably contributes to or exacerbates the obesity-related hypertension. OSA should be strongly suspected in obese individuals with resistant hypertension, those with the absence of a nocturnal decrease in blood pressure, those with unexplained weight gain or difficulty losing weight, and in those with symptoms suggestive of OSA. The diagnosis of OSA in this context is therefore of considerable clinical importance.

There are some potential limitations to this study. One limitation of the study was that we have considered parameters such asthyromental distance and Mallampati grading for cephalometric analysis as they are easy and cheap to perform in clinical practice and can correlate with severity of OSA. There are multiple imaging techniques to evaluate the upper airway in patients with OSA such as cephalometric radiography, CT, MRI, fluoroscopy, and somnofluoroscopy but these methods are cumbersome and expensive. Cephalometry is a diagnostic procedure to collect information on skeleton abnormalities and soft tissues of patients with OSA, providing support for indication of surgery. This should be based on disease severity and the presence of anatomical alterations of upper airway and of craniofacial skeleton. Another limitation is that the findings of this study need to be confirmed in a large number of samples.

It can be concluded that obstructive sleep apnea is not uncommon in the nonobese persons contrary to earlier concept that OSA is confined to the obese persons. Therefore, whenever a nonobese patient presents with a clinical picture suggestive of

OSA, the diagnostic possibility should not be underestimated and should be evaluated thoroughly.

Acknowledgments

The authors are grateful to all the patients for their co-operation and to Mr. Ravi Verma for his technical help.

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How to cite this article: Garg R, Singh A, Prasad R, Saheer S, Jabeed P, Verma R. A comparative study on the clinical and polysomnographic pattern of obstructive sleep apnea among obese and non-obese subjects. Ann Thorac Med 2012;7:26-30.

Source of Support: Nil, **Conflict of Interest:** None declared.