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Differential Profile of OSA in Obese Kashmiri Patients of Northern India

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ABSTRACT

Obstructive sleep apnea (OSA) and obesity are two interacting global epidemics both with high prevalence and morbidity. Both epidemiologic and clinical studies suggest that majority of patients with obesity also have OSA and untreated OSA in these patients results in significant cardiovascular and metabolic complications.

Objectives: To evaluate the profile of OSA in obese patients of Kashmir.

Methods: We performed polysomnography studies in obese patients that were referred from various sub-specialty clinics from July 2011 to August 2013.

Results: Out of 182 patients who underwent polysomnography (PSG), 11O (60.4%) were obese (BMI > 30 kg/m2). In 110 obese patients, 104 (94.5%) had OSA. Hypertension, diabetes and dyslipidemia were more prevalent among obese ($p<0.05$). The mean neck circumference and mean BMI of obese patients was significantly more than that of non-obese (33.9 kg/m2 vs. 26.8 kg/m2) ($p <0.000$). Presenting symptoms of obese were snoring (97.3%), daytime sleepiness (87.3%) with a mean ESS of 15.3, disturbed nocturnal sleep (70.0%), nocturia (62.7%) and witnessed apneas (45.5%). OSA was significantly ($p=0.002$) more common among obese compared to non-obese (93% vs 76%). Most were clinically suspected to have OSA by internists (29%), cardiologists (20%), endocrinologists (15%) and psychiatrists (13%). Sleep efficiency was significantly less ($p< 0.03$) in obese patients but sleep latency and REM sleep latency did not significantly differ between obese and non-obese. Unlike awake oxygen saturation the average nocturnal oxygen saturation of obese patients was significantly less [$p=0.001$] than that of non-obese patients (84.7% vs. 88.1%). The mean AHI of obese patients was significantly more than non-obese i.e 24.3 vs. 18.0 ($p = 0.001$) and so was the

mean ODI i.e 24.6 vs. 17.2 (p = 0.001). Variables that significantly correlated with presence of OSA include age, gender, BMI, hypertension, diabetes and cardiovascular disease (p< 0.05), however on logistic regression only BMI, hypertension, and nocturia correlated with OSA. CPAP therapy improved snoring, nocturia, nocturnal sleep and daytime sleepiness more in obese than non-obese OSA patients.

Conclusions: OSA which is highly prevalent among obese Kashmiri patients, is largely unrecognized in the primary care setting. It is associated with significant comorbidities and most of these improve with CPAP therapy. ***JMS 2016; 19(2):e1-e13.***

Keywords: Obstructive sleep apnea, Obesity, CPAP.

Introduction:

A substantial proportion of patients with obesity suffer from unrecognized obstructive sleep apnea (OSA) and conversely, obesity is more prevalent among OSA patients. OSA is a common sleep disorder affecting 10–20% of the general population.[1,2] A respiratory sleep-related disease, OSA is characterized by collapse of the pharynx in the face of persistent ineffective breathing efforts leading to repetitive interruptions of ventilation during sleep resulting in sleep fragmentation and arterial hypoxemia. OSA is characterized by upper airway instability during sleep, resulting in markedly reduced (hypopnea) or absent (apnea) airflow. OSA has been commonly associated with obesity, hypertension (HTN), type 2 diabetes mellitus (T2 DM), coronary artery disease (CAD), congestive heart failure (CHF), atrial fibrillation (AF) and stroke.[3-8] OSA not only increases long-term morbidity and mortality but has also been implicated in sudden nocturnal death.[9,10] Few prospective studies have concluded that even habitual snoring independently increases the risk of T2DM[11,12]. A recent study suggested that mild oxyhemoglobin desaturation of less than 4% during sleep may predispose to fasting hyperglycemia.[13] OSA has been reported as a highly prevalent comorbidity of T2DM by various authors [14-16] and among obese patients with T2DM prevalence has recently been estimated at a staggering 86% in the United States[17]. In a recent prospective study of patients admitted with acute myocardial infarction (AMI) evaluated by overnight polysomnography (PSG), OSA was found

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to be significantly underdiagnosed[17]. Compared to stable outpatients, COPD patients who are admitted to the hospital with exacerbations have been found to have increased prevalence of OSA.[18]

Low awareness of OSA in obese patients among varoius admitting services, further compounds this problem. Given the gravity of this common but still under-diagnosed disorder, systematic evaluation and treatment of obese OSA patients is urgently required. Similarly the question whether OSA represents an independent risk for the development of pre-diabetes and T2DM over time also remains to be investigated by large prospective studies. It needs to be established firmly whether Continuous Positive Airway Pressure (CPAP) treatment of OSA improves various metabolic issues including glucose metabolism in obesity. Therefore the effective treatment of OSA with CPAP could potentially improve glucose control in millions of diabetics with OSA world over and thus has major clinical implications. Keeping this in mind we in the present study evaluated the profile of obese Kashmiri patients with OSA.

Methods:

Patients suspected of OSA who were referred for Polysomnography (PSG) from various sub-specialty clinics were recruited at Modern Hospital, Rajbagh, Srinagar between July 2011 to August 2013. Exclusion criteria were: 1) Patients on nocturnal oxygen supplementation; 2) Unstable Cardio-pulmonary, neurological, or psychiatric disease 3) Upper airway surgery 4) Using positive airway pressure therapy or oral appliances.

All participants gave written informed consent before PSG. A detailed history of complaints including snoring, witnessed apneas, nocturia, disturbed nocturnal sleep and morning headaches was taken. Day time sleepiness was assessed by Epworth Sleepiness Scale (ESS)). Height, weight and neck circumference were measured in all patients and body mass index (BMI) was calculated. An overnight laboratory PSG was then performed to diagnose the presence and severity of OSA. PSG recordings were started based on the subject's usual domestic sleeping habits and each patient was recorded for a minimum of 7 hours.

PSG included recordings of airflow by nasal pressure transducer and oronasal thermocouples, chest and abdominal wall motion by piezo electrodes, oxygen saturation by pulse oximeter, electrocardiogram, six electroencephalogram channels, bilateral electro-oculograms, chin and tibialis electromyogram. Recordings were scored visually in 30 seconds in Non-REM sleep stages 1–4 sleep and in REM sleep according to standard criteria[19]. Similarly respiratory events and microarousals were scored according to established criteria[20,21]. Complete cessation of airflow for at least 10 seconds was defined as apnea (obstructive if respiratory efforts were present and central if respiratory efforts were absent) and hypopnea was

identified if there was a discernable reduction in airflow lasting at least 10 seconds and associated with at least 3% oxygen desaturation. The apnea-hypopnea index (AHI) was defined as the total number of obstructive apneas and hypopneas per hour of sleep. Oxygen desaturation index (ODI) was defined as the total number of desaturations of at least 3% per total sleep time in hours. We defined OSA severity categories according to commonly used clinical cutoffs i.e., No OSA (AHI <5); Mild OSA (AHI ≥5 but <15); Moderate OSA (AHI ≥15 but <30); and Severe OSA (AHI ≥30).

Statistical Analysis:

Standard methods of statistical analysis were used for data analysis. After descriptive statistical analysis of the general characteristics of the study participants, the Kolmogorov–Smirnov test was used to examine the distribution of variables, and the Levene test to study the variance. Qualitative variables were analyzed with the x2 test or with Fisher's exact test if at least one cell had an expected count < 5. Student's t-test was applied to compare mean values of quantitative variables when the distribution was normal and the Mann–Whitney U test when it was not. For paired samples the Student's t-test for paired samples and Mc Nemar test were used. Pearson's coefficient was used to test the correlation between quantitative variables; $p \leq 0.05$ was considered significant. SPSS 11.0 was used for data analyses (SPSS Inc., Chicago, IL, USA).

Results:

Out of 182 patients who underwent polysomnography, 11O (60.44%) were obese (BMI > 30 kg/m2) including 50 (45.5%) males and 60 (54.5%) females (p> 0.5). Out of 110 obese patients, 104 (94.5%) had abnormal test and only 6 (5.5%) had normal PSG. The mean age (Table 1) was not significantly different between obese and non-obese subjects (54.9 vs 55.5 years). Comorbidities like hypertension, T2DM and dyslipidemia were more prevalent among obese compared to non-obese patients (p<0.05). However other comorbidities like hypothyroidism, cardiovascular disease and COPD did not differ among the obese and non-obese in the study population (p> 0.05). Mean BMI (Table 2) of obese patients was significantly more than that of non-obese patients (33.9 kg/m2 vs. 26.8 kg/m2) (p <0.000) and so was the mean neck circumference (40.9 vs 39.3 cms) (p<0.002).

The main presenting symptoms of obese patients were snoring 107 (97.3%), daytime sleepiness 96 (87.3%) with a mean ESS of 15.3, disturbed nocturnal sleep 77 (70.0%), nocturia 69 (62.7%) and witnessed apneas 50 (45.5%). All these symptoms were more common in obese compared to non-obese patients (Table 3). There was no significant difference in sleepiness between obese and non-obese whose mean ESS was 15.3 and 14.4 respectively (p > 0.5). There were few atypical presentations in our study that included nocturnal enuresis and night terrors for two years in a 38 year obese

male, a 55 year businessman felt asleep while going for a morning walk and slipped into a drain and a near fatal road traffic accident when a retired engineer dozed off while driving his car. OSA was significantly (p=0.002) more common among obese compared to non-obese individuals (93% vs 76%). Clinically most of these patients were initially suspected to have OSA by internists (29%), cardiologists (20%), endocrinologists (15%) and psychiatrists (13%) before referring them for polysomnography (Table 4).

Sleep efficiency (Table 5) was significantly less (p< 0.03) in obese patients (mean 71.0%) as compared to non-obese (mean 74.0%). Mean Sleep latency did not significantly differ (p= 0.41) between obese and non-obese patients (21.6 vs 20.5 minutes). Like sleep latency, mean REM sleep latency also did not significantly differ among obese and non-obese (74.3 vs 75.2 minutes) [p = 0.66]. Mean room air awake oxygen saturation (SpO2) did not significantly differ between obese and non-obese patients (92.7% vs 93.9%) [p=0.053]. Unlike awake oxygen saturation the average nocturnal oxygen saturation of obese patients was significantly less [p=0.001] than that of non-obese patients (84.7% vs. 88.1%). The mean AHI of obese patients was significantly more than non-obese patients i.e 24.3 vs. 18.0 (p = 0.001). The mean ODI of obese patients was significantly more than non-obese i.e 24.6 vs. 17.2 (p = 0.001). Variables that significantly correlated with presence of OSA include age, gender, BMI, hypertension, diabetes and cardiovascular disease (p< 0.05), however on logistic regression only BMI, hypertension, and nocturia correlated with OSA.

CPAP therapy improved snoring in both obese as well as non-obese OSA patients, however there was no significant difference between the two groups (p = 0.663) in terms of response category (Table 6). Like snoring, CPAP therapy also improved nocturia, nocturnal sleep and daytime sleepiness in both obese and non-obese OSA patients, however the overall improvement was more in obese compared to non-obese subjects.

Discussion:

Obstructive sleep apnea (OSA) is underrecognized globally and as obesity relentlessly increases so does OSA. This may be partially explained by the lack of awareness of OSA even in hospitalized settings as is illustrated by the minimal documentation of prior OSA history by resident doctors and limited referrals for OSA evaluation. Recognizing this condition in a timely and efficient manner may lead to fewer readmissions and ultimately reduce costs for at-risk population.[22] Usual risk factors that are independently associated with high risk for OSA include high BMI, male sex, CAD, CHF, and COPD. OSA has significant cardiovascular implications and is associated with sudden death.[23] Although once thought to be independent diseases, the high prevalence of OSA among obese

diabetics [24] and vice versa [25] has raised interesting questions as to how OSA and diabetes interact. Untreated OSA is associated with poor glycemic control resulting in intensification of pharmacotherapy that promotes weight gain and further exacerbates the severity of existing OSA thereby elevating cardiovascular risk. OSA is characterized by recurrent upper airway occlusions during sleep that result in specific physiologic disturbances, including sleep fragmentation and chronic intermittent hypoxia. These disturbances lead to a cascade of events related to the activation of the sympathoadrenal system, oxidative stress, systemic inflammation, and changes in adipokines – all of which are important in increasing the risk of hypertension, cardiovascular disease, metabolic syndrome, and diabetes [26,27]. Enough evidence has been found that supports the hypothesis that OSA and the resultant intermittent hypoxia, elevated sympathetic nervous activity, sleep fragmentation, less amount of slow wave sleep and cumulative sleep loss has adverse effects on glucose tolerance.[28] Chronic sleep loss, a consequence of OSA, is associated with decreased glucose tolerance, decreased leptin, and an increase in evening cortisol levels. Both adipose tissue and diabetes are associated with immune activation and subsequent increase in the circulating pro-inflammatory cytokines, which in turn play a role in the pathogenesis of OSA[29]. In the present study nearly 95% obese patients had OSA indicating that OSA is a highly prevalent comorbidity in obese Kashmiri patients.

Another important observation of our study was that compared to non-obese the average nocturnal oxygen saturation of obese patients was significantly less. Pulse oximetry can be a key to appropriate use of polysomnography by identifying patients most likely to have an uninterpretable test i.e severe insomnia, high oxygen requirement, excruciating pain, altered mental status, frequent anticipated disturbances during the night, and severe restless legs syndrome. Under these circumstances the standardization of pulse oximetry is essential to facilitate agreement with polysomnography and an ODI ≥ 15/h essentially rules out false positive diagnoses and can be a cost-effective tool particularly in resource limited regions like Kashmir.

CPAP (continuous positive airway pressure) a nonpharmacological intervention which is the gold-standard treatment of OSA, is highly effective in relieving the symptoms of OSA. In one of our recent studies[30] we demonstrated the positive impact of CPAP in the management of blood glucose control as reflected by decline in HbA1c levels in 59% of Kashmiri diabetic patients. A reduction in HbA1c level has been shown to be associated with a reduction in the risk of macrovascular and microvascular complications associated with diabetes.[31,32] In addition to improvement in blood glucose, CPAP therapy also resulted in improvement in blood pressure of as many as 75% of our patients. This dual benefit of positive

impact on blood sugar and blood pressure by CPAP treatment may potentially help globally millions of patients with OSA. One of the earliest studies was that of Brooks et al [33] who investigated the insulin responsiveness before and during CPAP in 10 very obese OSA patients. CPAP significantly improved insulin responsiveness in those patients within 4 months of treatment. In a comparable study, Harsch et al [34] also have reported improvement in insulin sensitivity in obese diabetics with CPAP therapy. The effect, however, was seen after 3 months of CPAP and not immediately like that in non-obese non-diabetics.[35]

Clinician has to carefully examine the associations between OSA severity and metabolic variables. It is also important to obtain polysomnographic recordings and administer subsequent CPAP therapy for longer than the commonly used minimum of 4 hours.

Though CPAP adherence is fundamental to OSA therapy, we should not ignore that the benefits of CPAP may not be the same for cognitive and metabolic outcomes. Our earlier study has demonstrates that CPAP treatment not only improves glycemic control in a significant number of OSA patients but it also improves their cognitive function[30].

Conclusion:

The findings of our study suggest that OSA is highly prevalent among obese Kashmiri patients and is largely unrecognized in the primary care setting. Most of the clinicians do not suspect OSA in the beginning resulting in delayed diagnosis. Thus the role of OSA in the management of obesity is in urgent need of further assessment and current practice approaches should be modified to include systematic evaluation and treatment of OSA.

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TABLE 1: Demographic characteristics of the Study population, obese vs. non-obese

Demographic characteristic		Non Obese (n=72)	Obese (n=110)	Total (n=182)	P value
Age(Years)	Mean ±SD (Range)	55.5 ±12.8 (30 – 85)	54.5 ±13.0 (22 – 90)	54.9 ±12.9 (22 – 90)	0.591
	< 30 y	0, 0.0%	4, 3.6%	4, 2.2%	0.144
	30 – 39	11, 15.3%	7, 6.4%	18, 9.9%	
	40 – 49	13, 18.1%	30, 27.3%	43, 23.6%	
	50 – 59	21, 29.2%	26, 23.6%	47, 25.8%	
	60 – 69	15, 20.8%	24, 21.8%	39, 21.4%	
	≥ 70 y	12, 16.7%	19, 17.3%	31, 17.0%	
Female gender	N, %	35, 48.6%	60, 54.5%	95, 52.2%	0.433
Hypertension	N, %	41, 56.9%	80, 72.7%	121, 66.5%	0.027*
Diabetics	N, %	18, 25.0%	44, 40.0%	62, 34.1%	0.037*
Hypothyroidism	N, %	23, 31.9%	40, 36.4%	63, 34.6%	0.054
CVD	N, %	7, 9.7%	21, 19.1%	28, 15.4%	0.087
Dyslipidemia	N, %	4, 5.6%	22, 20.0%	26, 14.3%	0.006*
COPD/ Asthama	N, %	10, 13.9%	14, 12.7%	24, 13.2%	0.821

*Significant

TABLE 2: Anthropometric characteristics of the Study population, obese vs. non-obese

Variable		Non Obese (n=72)	Obese (n=110)	Total (n=182)	P value
BMI	Mean ±SD (Range)	26.8 ±2.1 (21 - 29)	33.9 ±3.5 (30 - 49)	31.1 ±4.6 (21 - 49)	0.000*
Neck circum	Mean ±SD (Range)	39.3 ±2.9 (34 - 46)	40.9 ±3.6 (33 - 50)	40.3 ±3.4 (33 - 50)	0.002*
	>= 47 cm	0, 0.0%	5, 4.5%	5, 2.7%	0.085
	37 - 47 cm	59, 81.9%	93, 84.5%	152, 83.5%	
	< 37 cm	13, 18.1%	12, 10.9%	25, 13.7%	

*Significant

TABLE 3: OSA features of the Study population, obese vs. non-obese

	Obese (BMI>30)		Total	P value
	No	Yes		
Co-Morbidity	10	16	26	0.901
	13.9%	14.5%	14.3%	
Snoring	66	107	173	0.088
	91.7%	97.3%	95.1%	
Witnessed Apneas	23	50	73	0.069
	31.9%	45.5%	40.1%	
Nocturia	39	69	108	0.250
	54.2%	62.7%	59.3%	
Disturbed sleep	44	77	121	0.214
	61.1%	70.0%	66.5%	
Daytime Sleepiness	57	96	153	0.144
	79.2%	87.3%	84.1%	
OSA	55	102	157	0.002*
	76.4%	92.7%	86.3%	
OSA Severity	16	45	61	0.115
o Severe	16	45	61	
	29.1%	44.1%	38.9%	
o Moderate	26	43	69	
	47.3%	42.2%	43.9%	
o Mild	13	14	27	
	23.6%	13.7%	17.2%	

TABLE 4 : Clinical suspicion of OSA specialty-wise, obese vs. non-obese

Referred by	Obese (BMI>30)		Total
	No	Yes	
Pulmonologist	04, 5.6%	05, 4.5%	09, 4.9%
Psychiatrist	12, 16.7%	14, 12.7%	26, 14.3%
Neurologist	05, 6.9%	06, 5.5%	11, 6.0%
Nephrologist	04, 5.6%	04, 3.6%	08, 4.4%
Internist	20, 27.8%	32, 29.1%	52, 28.6%
Gen. surgeon	03, 4.2%	03, 2.7%	06, 3.3%
ENT surgeon	04, 5.6%	06, 5.5%	10, 5.5%
Endocrinologist	10, 13.9%	17, 15.5%	27, 14.8%
Cardiologist	10, 3.9%	22, 20.0%	32, 17.6%
Anaesthetist	0, 0.0%	01, 0.9%	01, 0.5%
P = 0.963			

TABLE 5 : PSG characteristics of the Study population, obese vs. non-obese

		Non Obese (n=72)	Obese (n=110)	Total (n=182)	P value
E. S. Score	Mean ±SD	14.4 ±4.2	15.3 ±3.7	14.9 ±3.9	0.156
	Range	7 - 22	7 - 22	7 - 22	
Sleep Efficiency	Mean ±SD	74.0 ±10.7	71.0 ±8.1	72.2 ±9.3	0.034*
	Range	52 - 93	50 - 91	50 - 93	
Sleep latency	Mean ±SD	20.5 ±10.1	21.6 ±8.3	21.2 ±9.1	0.419
	Range	8 - 56	8 - 51	8 - 56	
REM latency	Mean ±SD	75.2 ±15.3	74.3 ±13.7	74.6 ±14.3	0.663
	Range	41 - 114	40 - 112	40 - 114	
Awake SpO2	Mean ±SD	93.9 ±3.6	92.7 ±4.2	93.2 ±4.0	0.053
	Range	80 - 98	80 - 98	80 - 98	
Nocturnal SpO2	Mean ±SD	88.1 ±5.6	84.7 ±6.9	86.0 ±6.6	0.001*
	Range	64 - 95	64 - 95	64 - 95	
AHI	Mean ±SD	18.0 ±12.3	24.3 ±11.3	21.8 ±12.0	0.001*
	Range	1 - 46	2 - 54	1 - 54	
ODI	Mean ±SD	17.2 ±13.9	24.6 ±15.0	21.7 ±15.0	0.001*
	Range	0 - 61	1 - 61	0 - 61	
*Significant					

TABLE 6 : Effect of CPAP therapy, obese vs. non-obese

	Obese (BMI>30)		Total	P value
	No	Yes		
Treatment				
o CPAP / BiPAP	47 65.3%	96 87.3%	143 78.6%	
o Refused treatment	2 2.8%	4 3.6%	6 3.3%	0.001*
o NA	17 23.6%	8 7.3%	25 13.7%	
o Medical Management	6 8.3%	2 1.8%	8 4.4%	
Snoring effect				
Significant improvement / Recovery	34 73.9%	77 81.9%	111 79.3%	
Moderate improvement	7 15.2%	10 10.6%	17 12.1%	0.663
Mild improvement	3 6.5%	3 3.2%	6 4.3%	
Worsening / No improvement	2 4.3%	4 4.3%	6 4.3%	
Nocturia effect				
Significant improvement/ Recovery	7 24.1%	23 36.5%	30 32.6%	
Moderate improvement	11 37.9%	27 42.9%	38 41.3%	0.266
Mild improvement	6 20.7%	9 14.3%	15 16.3%	
Worsoning/ No improvement	5 17.2%	4 6.3%	9 9.8%	
*Significant				