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Original Research Article

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To study the prevalence of obstructive sleep apnoea in type 2 diabetes patients in Western Rajasthan, India

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ABSTRACT

Background: Obstructive Sleep Apnoea (OSA) has been too common yet under diagnosed clinical entity. It is associated with the metabolic syndrome, a cluster of cardio-metabolic parameters including central obesity, insulin resistance, hypertension and dyslipidemia. Obesity predisposes to both OSA and disorders in glucose metabolism. There is growing evidence that OSA confers an independent risk of adverse glucose metabolism.

Methods: The present study conducted in the Department of Medicine at MDM Hospital attached to Dr. S.N. Medical College, Jodhpur, Rajasthan, India. Participants after understanding the study protocol and procedure, asked to give their written consent for the study. It was a cross sectional hospital based study in patients, screened at Diabetic clinic and those referred from the periphery. Berlin questionnaires and Epworth score are tools to screen for OSA attending the Medicine OPD and IPD, Dr. S. N. Medical College, Jodhpur. 50 patients with type 2 DM and 20 age and sex-matched controls were studied. Randomly selected T2DM subjects of age 20 to 75 years both sex with obesity, BMI>25 kg/m², clinical history suggestive of OSA, Epworth score>6, Positive Berlin questionnaires were included in the study. Acute and unstable medical condition e.g. CHF, CRF, COPD, Recent stroke, Acute ACS, Pregnant women were excluded.

Results: In the study OSA was prevalent in the diabetic population (54%), Mean age of the study population was 54.96 ± 9.35 years. OSA was found to be increased with increasing age with maximum prevalence in \geq 60 year's age group. OSA was more prevalent in the male population (64.29%), in urban population. Snoring, observed sleep apnoea, restless sleep/insomnia, excessive daytime sleepiness and non-refreshing sleep were significantly associated with OSA. (P<0.001). Prevalence of OSA was more in high BMI group (\geq 35 kg/m²), OSA increased with increase in neck circumference. OSA was more in subjects with uncontrolled diabetes (blood sugar>200 mg/dl), smokers and alcoholics.

Conclusions: This study shows that OSA has a high prevalence in subjects with T2DM and identify several factors that may be associated with its presence in the diabetic population. OSA can be usefully and easily assessed in an outpatient setting by using a portable device such as Apnea Link. Clinicians should increase patients' awareness of the signs and symptoms of OSA and refer for sleep studies when appropriate. Once diagnosed, patients should be encouraged to adhere to CPAP treatment in order to halt progression and prevent complications.

Keywords: Body mass index, Neck circumference, OSA, Type-2 diabetes mellitus

INTRODUCTION

Obstructive Sleep Apnea (OSA) has been too common yet under diagnosed clinical entity. Earlier studies in West have shown prevalence is 4% for men and 2% for women. Prevalence studies from India have shown a prevalence of sleep disordered breathing and OSA syndrome almost similar to those in the West. Udawadia ZF et al in its two-phase cross-sectional prevalence study found that the prevalence of OSA is 7.5% in urban Indian male in the age group 35 to 65 years Sharma SK et al found prevalence is 3.57%. ^{2.3}

OSA is associated with the metabolic syndrome, a cluster of cardiometabolic parameters including central obesity, insulin resistance, hypertension and dyslipidemia. Obesity predisposes to both OSA and disorders in glucose metabolism.4 There is growing evidence that OSA confers an independent risk of adverse glucose metabolism.⁵ Observational data suggest the association of worse glycemic control in patients with diabetes and OSA.6,7 In Hong Kong, DM affects one-tenth of adults aged 25 to 74 years, whereas the age-standardized prevalence of obesity (BMI ≥25kg/m²) is as high as 29% in men and 21% in women in community. 9,10 With the sweeping epidemics of obesity and type 2 DM, there is potential interactions between OSA. Previous crosssectional studies that examined the relationship between OSA and glucose metabolism had focused on the prevalence of DM and impaired glucose metabolism among subjects with OSA compared with subjects without OSA whereas relatively few were published on OSA in established DM. The reported prevalence rates of OSA in diabetic populations ranged from 23% to 86%. 8,11-13 This wide variation in reported prevalence could be accounted by a number of reasons: different study designs ranging from population studies to clinic based studies. Different countries in Europe or in the United States, although the vast majority of subjects were of white ethnicities, and heterogeneous subject characteristics in terms of age, BMI, sex, and the use of antidiabetic medications. Further data will help to clarify the exact scale of the problem of sleep apnoea in diabetic populations. The aim of this study is to evaluate the prevalence of OSA among Western Rajasthan patients with type 2 Diabetes being followed up regularly at a DM clinic and to identify clinical parameters associated with OSA.

Laboratory night polysomnography is the gold standard method for diagnosis of OSA. Polysomnography is a non-invasive technique that involves overnight monitoring of electroencephalography, eye movements, muscle tone, respiratory efforts, and airflow and oxygen saturations. An apnoea was defined as the complete cessation of respiration for at least 10 seconds and hypopnea was defined as at least 30% reduction in thoracoabdominal movement or airflow lasting at least 10 s that was includes a reduction of airflow lasting at least 10 seconds that was associated with at least a 4% drop in

oxygen saturation. OSA is diagnosed when the Apnoea-Hypopnea index (AHI), i.e. the total number of obstructive apneas and hypopneas per hour of sleep, is greater than 5, excluding those with a central apnoea predominance, which was defined by (central apnoea index/AHI) >50%.²⁹ The severity of OSA is graded according to commonly used clinical criteria as mild (AHI>5 but less than 15), moderate (AHI>15 but less than 30), severe (AHI>30). Portable home monitoring as an alternative to laboratory-based PSG for the diagnosis of OSA in selected patients with can be done. Berlin questionnaires and Epworth score are tools to screen for OSA.

METHODS

The present study was conducted in the Department of Medicine at MDM Hospital attached to Dr. S.N. Medical College, Jodhpur, Rajasthan, India. Participants after understanding the study protocol and procedure, asked to give their written consent for the study. Present study was carried out in-group of patients belonging to western Rajasthan.

The design of this study was cross sectional hospital based study and subjects were those screened at diabetic clinic, medical OPD and those referred from the periphery. Berlin questionnaires and Epworth score are tools to screen for OSA.

Inclusion criteria

Randomly selected T2DM subjects of age 20 to 75 years both sex with obesity, BMI>25 kg/mt2, clinical history suggestive of OSA, Epworth score>6, Positive Berlin questionnaires.

Exclusion criteria

Acute and unstable medical condition e.g. CHF, CRF, COPD, Recent stroke, Acute ACS, Pregnant women.

Laboratory investigation

Haemoglobin, blood sugar, serum creatinine, lipid profile, thyroid profile, ECG, chest X-ray PA view, polysomnography.

Number of cases to be studied: 50 Number of control to be studied: 20

Clinical evaluation, anthropometric measures, relevant laboratory investigations and nocturnal polysomnography study test were performed at MDM Hospital Jodhpur, Rajasthan, india. Polysomnography is a non-invasive technique that involves overnight monitoring of electroencephalography, eye movements, muscle tone, respiratory efforts and airflow and O₂ saturations. OSA is diagnosed when the AHI is greater than five.

Data evaluation

In a study, Epi Info statistics and multivariate analysis method used to analyse the data.

RESULTS

In present study group prevalence of OSA in 35-44 years age group was 33.33%, in 45-59 years age group was 44% and in ≥60 years age group was 73.68%. In this study, 28 (56%) subjects were male, out of which 18 male subjects were observed with OSA and 22 (44%) subjects were female, out of which 9 female subjects were observed with OSA with prevalence of OSA, in male was 64.29% and in female was 40.91%. OSA was prevalent in 58.33% urban and in 42.86% of rural subjects. The prevalence of OSA was 71.43% in subjects

related to business, 62.5% in Government employee, 40% in farmers and 33.33% in subjects related to housework.

Table 1: Prevalence of OSA in different age groups.

Age (in years)	Total N=50	OSA subjects N=27	Prevalence %
35-44	6	2	33.33
45-59	25	11	44
≥60	19	14	73.68

Table 2 shows prevalence of symptoms in OSA and without OSA subjects. Prevalence of snoring was 100% in OSA subjects and 34.78% in without OSA subjects. Prevalence of observed sleep apnoea was 66.67% in OSA subjects and 21.74% in without OSA subjects.

Table 2: Prevalence of symptoms in OSA and without OSA subjects.

Presenting Symptoms	With OSA	Prevalence (%)	Without OSA	Prevalence (%)
Snoring	27	100	8	34.78
Observed Sleep Apnoea	18	66.67	5	21.74
Restless sleep/ insomnia	22	81.48	11	47.83
Excessive day time sleepiness	18	66.67	4	17.39
Non-refreshing nocturnal sleep	17	62.96	7	30.43
Fatigue	14	51.85	17	73.91
Impaired concentration	6	22.22	4	17.39

Prevalence of restless sleep/insomnia was 81.48% in OSA subjects and 47.83% in without OSA subjects. Prevalence of excessive daytime sleepiness was 66.67% in OSA subjects and 17.39% in without OSA subjects. The prevalence of non-refreshing nocturnal sleep was 62.96% in OSA subjects and 30.43% in without OSA subjects. Prevalence of fatigue was 51.85% in OSA subjects and 73.91% in without OSA subjects. The prevalence of impaired concentration was 22.22% in OSA subjects and 17.39% in without OSA subjects.

In the present study 6 (22.2%) subjects were in mild OSA severity group, out of which 4 (66.7%) were male and 2 (33.3%) were female subjects. 10 (37.0%) subjects were in Moderate OSA severity group, out of which 7 (70%) were male and 3 (30%) were female subjects. 11 (40.7%) subjects were in severe OSA severity group, out of which 7 (63.6%) were male and 4 (36.4%) were female subjects.

In this cohort mean BMI was $35.14\pm7.27~kg/m^2$. In the present study, 19 subjects were in $25-29.99~kg/m^2~BMI$ group, out of which 7 were observed with OSA and 13 subjects were in $30-34.99~kg/m^2~BMI$ group, out of which, 7 were observed with OSA and 18 subjects were in $\geq 35~kg/m^2~BMI$ group, out of which, 13 were observed with OSA.

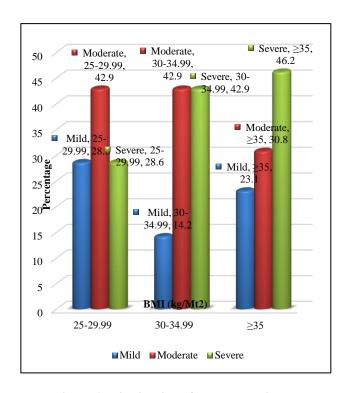


Figure 1: Distribution of BMI according to OSA severity.

So, prevalence of OSA, in 25-29.99 kg/m² BMI group was 36.84%, in 30-34.99 kg/m² BMI group was 53.85% and in \ge 35 kg/m² BMI group was 72.22%. In the present study, 7 OSA subjects were with BMI 25-29.99 kg/m², out of which 2 (28.6%) subjects were in mild OSA severity group, 3 (42.9%) subjects were in moderate OSA severity group, 2 (28.6%) subjects were in severe OSA severity group.7 OSA subjects were with BMI 30-34.99 kg/m², out of which 1 (14.2%) subjects were in mild OSA severity group, 3 (42.9%) subjects were in moderate OSA severity group, 3 (42.9%) subjects were in severe OSA severity group. 13 OSA subjects were with BMI \geq 35 kg/m², out of which 3 (23.1%) subjects were in mild OSA severity group, 4 (30.8%) subjects were in moderate OSA severity group, 6 (46.2%) subjects were in severe OSA severity group.

Table 3: The prevalence of OSA in study subjects based on neck circumference.

Neck circumference (cm)	Total N=50	OSA subjects N=27	Prevalence (%)
30-35	16	1	6.25
36-40	16	9	56.25
>40	18	17	94.44

Table 3 shows the prevalence of OSA, in subjects with NC 30-35cm was 6.25% and in subjects with NC 36-40cm was 56.25%, in subjects with NC>40cm was 94.44%.

Table 4: The prevalence of OSA based on blood sugar level.

Random blood sugar (mg/dl)	Total N=50	OSA subjects N=27	Prevalence (%)
≤150	22	5	22.7
151-200	17	12	70.6
>200	11	10	90.9

Table 4 shows the prevalence of OSA based on blood sugar level of study subjects. In the present study, 22 subjects were with blood sugar $\leq\!150$ mg/dl, out of which 5 subjects were observed with OSA, 17 subjects were with blood sugar 151-200 mg/dl, out of which 12 subjects were observed with OSA and 11 subjects were with blood sugar >200 mg/dl, out of which 10 subjects were observed with OSA Based on the above data, the prevalence of OSA , in subjects with blood sugar $\leq\!150$ mg/dl was 22.7%, in subjects with blood sugar 151-200 mg/dl was 70.6% and in subjects with blood sugar >200 mg/dl was 90.9%.

Table 5 shows relation between OSA severity and Blood sugar level of study subjects. In the present study, 5 subjects were with blood sugar ≤150 mg/dl, out of which 4 (80%) subjects were with mild OSA and 1 (20%)

subject was with moderate OSA severity. 12 subjects were with blood sugar 150-200 mg/dl, out of which 2 (16.7%) subjects were with mild OSA severity and 6 (50%) subject was with moderate OSA severity and 4 (33.3%) subjects were with severe OSA severity. 10 subjects were with blood sugar >200 mg/dl, out of which 3 (30%) subjects were with moderate OSA severity and 7 (70%) subject was with severe OSA severity.

Table 5: Relation of OSA severity with blood sugar level.

Random blood	OSA severity		
sugar (mg/dl)	Mild	Moderate	Severe
≤150 N=5	4 (80)*	1 (20)*	0 (0)*
151-200 N=12	2 (16.7)	6 (50)	4 (33.3)
>200 N=10	0	3 (30)	7 (70)

*percentage

Table 6 shows the prevalence of OSA based on HbA1c level. According to above data prevalence of OSA in subjects with HbA1c 6.5-6.99% was 56.3%, in subjects with HbA1c 7.0-7.49 % was 47.4% and in subjects with HbA1c >7.5% was 60%.

Table 6: The prevalence of OSA based on HbA1c level.

HbA1c	Total	OSA subjects	Prevalence
(%)	N=50	N=27	(%)
6.5-6.99	16	9	56.3
7.0-7.49	19	9	47.4
>7.5	15	9	60

Table 7: Relationship of HbA1c with OSA severity.

HbA1c	OSA severity		
(%)	Mild	Moderate	Severe
	N=6	N=10	N=11
6.5-6.99 N=9	2 (22.2)*	3 (33.3)*	4 (44.4)*
7.0-7.49 N=9	2 (22.2)	4 (44.4)	3 (33.3)
>7.5 N=9	2 (22.2)	3 (33.3)	4 (44.4)

Table 7 shows relation between OSA severity and HbA1c level of study subjects. In the present study, 9 OSA subjects were with HbA1c 6.5-6.99%, out of which 2 (22.2%) subjects were in mild OSA severity group, 3 (33.3%) subjects were in moderate OSA severity group and 4 (44.4%) subjects were with severe OSA severity group. 9 OSA subjects were with HbA1c 7.0-7.49%, out of which 2 (22.2%) subjects were in mild OSA severity group, 4 (44.4%) subjects were in moderate OSA severity group and 3 (33.3%) subjects were in severe OSA severity group, 9 OSA subjects were with HbA1c>7.5%, out of which 2 (22.2%) subjects were in mild OSA severity group, 3 (33.3%) subjects were in moderate OSA severity group and 4 (44.4%) subjects were in severe OSA severity group and 4 (44.4%) subjects were in severe OSA severity group.

Table 8: The prevalence of OSA in study subjects according to their addiction.

Addiction	Total N=50	OSA subjects N=27	Prevalence (%)
Smoker	16	10	62.5
Non-smoker	34	17	50
Alcoholic	10	8	80
Non-alcoholic	40	19	47.5

Table 8 shows the prevalence of OSA in study subjects according to their addiction. According to the above data, the prevalence of OSA, in smokers was 62.5% and in non-smokers was 50%. The prevalence of OSA in alcoholic subjects was 80% and was 47.5% in non-alcoholic subjects.

Table 9: Prevalence of HTN in different OSA severity groups.

OSA Severity	OSA subjects N=27	With HTN N=15	Prevalence (%)
Mild	6	2	33.33
Moderate	10	4	40
Severe	11	9	81.82

Table 9 shows prevalence of HTN in different OSA severity groups. In the present study, total 27 subjects were observed with OSA, out of which 15 (55.56%) subjects presented with HTN. According to the above data, 6 subjects were observed with mild OSA severity, out of which 2 (33.33%) subjects presented with HTN and 10 subjects were observed with moderate OSA severity, out of which 4 (40%) subjects presented with HTN and 11 subjects were observed with severe OSA severity, out of which 9 (81.82%) subjects presented with HTN.

Table 10: Prevalence of IHD in different OSA severity groups.

OSA Severity	OSA subjects N=27	With IHD N=8	Prevalence (%)
Mild	6	1	16.67
Moderate	10	3	30
Severe	11	4	36.36

^{*}percentage

Table 10 shows the prevalence of IHD in different OSA severity groups. In the present study, total 27 subjects were observed with OSA, out of which 8 (29.63%) subjects presented with IHD. According to the above data, 6 subjects were observed with mild OSA severity, out of which 1 (16.67%) subjects presented with IHD and 10 subjects were observed with moderate OSA severity, out of which 3 (30%) subjects presented with IHD and 11 subjects were observed with severe OSA severity, out of which 4 (36.36%) subjects presented with IHD.

Table 11: Prevalence of OSA in control study subjects.

Control study subjects	With OSA	Prevalence (%)
N=20	1	5

Table 11 shows the prevalence of OSA in control study subjects that is 5%.

DISCUSSION

The present study is a hospital based observational cross-sectional study in Western Rajasthan assessing the prevalence of OSA in T2DM patients.

The sociodemographic features of the study subjects were according to the subjects attending diabetic clinic. The number of subjects studied were 50, out of these 28 (56%) were male and 22 (44%) were female. The mean age of the study population was 54.96±9.35 years, for male 57.07±8.23 yrs, for female 52.27±10.16 yrs. There were a higher number of urban subjects in a study that was 36 (72%) in comparison to, 14 (28%) rural subjects. This shows less awareness and less opportunity to seek medical facilities in rural region. Out of 50 study subjects maximum, 16 (32%) were government employee followed by 15 (30%) subjects related to housework, followed by 14 (28%) related to business and only 5 (10%) were farmer. The mean BMI (body mass index) of study subjects was 33.17±6.41 kg/m² and mean neck circumference was 38.38±4.71 cm.

As per present study prevalence of OSA in T2DM patients was 54%. In male, it was 64.29% and in female it was 40.91% while in control group prevalence of OSA was only 5%. The prevalence of OSA was highest in ≥60 yrs age group which was 73.68% followed by 44% in 45-59 years age group, followed by 33.33% in 35-44 years age group. The result demonstrates a high prevalence of OSA in diabetic men and women of all ages especially in older age.

Various studies in West have shown prevalence in nondiabetic men and women 4% and 2% respectively.1 Udawadia ZF et al found that the prevalence of OSA in the non-diabetic population is 7.5% in urban Indian male in the age group 35 to 65 years in their study.² Sharma SK et al found the prevalence in non-diabetic normal population 3.57%.3 Our finding seems to be consistent with results in the prior studies by Einhorn D et al.¹⁵ Reddy EV et al showed in their study that the prevalence of OSA increases with age, though age is not an independent risk factor for OSA.¹⁶ Katsumata et al also found that the prevalence of OSA was higher in diabetic male population in comparison with a non-diabetic male population in a study of 12,787 subjects. 17 Chasens et al found a very high incidence (65%) of undetected OSA in patients with T2DM.¹⁸ Elmasry et al also found a prevalence rate of OSA of 36% in patients with diabetes

in comparison with 14.5% in normoglycemic control subjects. ¹³ Punjabi NM et al found that recent research demonstrates the likelihood of a relationship between OSA and Type 2DM independent of obesity. ¹⁹

According to the present study OSA was more prevalent in the urban diabetic population as compared to the rural diabetic population as prevalence was 58.33% and 42.86% of urban and rural population respectively.

In present study OSA was more prevalent in subjects related to business (71.43%), followed by govt. Employees (62.5) than farmers (40%) and subjects related to housework (33.33%). This association may be because of lifestyle.

In-a-cross-sectional study, Reichmuth et al found that self-reported diabetes was more prevalent in 1,387 study subjects with an AHI level of \geq 15 events/h than those with an AHI value of <5 events/h, after controlling for shared risk factors such as age, sex, and body habitus.²⁰

In the present study, most of the patients complained of snoring. And snoring was seen in 27 (100%) patients with OSA while it was only 34.78% in without OSA subjects. This shows an association between snoring and OSA. This association was also established by previous studies. The Report of an American academy of sleep medicine task force also favour the study, according to them up to 60% (40 to 60%) patients with OSA reported snoring.²¹ Study of Einhorn D et al also states that snoring and reports of stopping breathing during sleep were also significantly associated with OSA for cut-off levels of ≥10, ≥15, ≥20 events/h. 15 Studies of Elmasry A et al and Al Delaimy WK et al found snoring to be a risk factor for the development of diabetes over 10 years independent of confounding factors.^{22,23} However virtually all patients with OSA snore, but not all snorers have sleep apnoea. This finding was favoured by study of Somers VK et al.²⁴

Observed sleep apnoea was present in 23 (46%) subjects out of which 66.67% were observed with OSA while only 21.74% contribution was from non OSA subjects. According to these data obstructive sleep apnoea is 3 times more prevalent in OSA patient than non OSA patients. According to the study of Resnick HE et al people with diabetes had more episodes of periodic breathing than did those without diabetes. Some other studies also found that autonomic dysfunction that may occur in those with DM could lead to this breathing instability. Act only diabetic patients had more sleep apnoea than non-diabetic but also in the diabetic population it was more associated with subjects with OSA.

In present study, other symptoms which were prevalent in OSA subjects than non OSA subjects were restless sleep/insomnia (81.48% in OSA, 47.83% in without OSA), excessive daytime sleepiness (66.67% in OSA, 21.74% in without OSA), non-refreshing nocturnal sleep (62.96% in

OSA, 30.43% in without OSA and impaired concentration (22.22% in OSA, 17.39% in without OSA). These findings were also supported by study of Einhorn D et al and study of Vgontzas AN et al.^{15,28}

A diagnosis of OSA syndrome is accepted when a patient has an Apnoea-Hypopnea Index (AHI; number of apneas and hypopneas per hour of sleep) >5 and symptoms of excessive daytime sleepiness (Sleep-related breathing disorders in adults. In the present study the prevalence of OSA was 54% in subjects with AHI \geq 5 (66.7% male, 33.3% female) and 42% in subjects with AHI \geq 15 (66.7% male, 33.3% female). Above findings were also supported by study of Einhorn D et al and study of Foster GD et al. In 1.15

In the present study OSA seems to be associated with higher BMI. The prevalence of OSA in subjects with BMI≥35 kg/mt² was highest (72.22%), followed by 53.85% in subjects with BMI 30-34.99 kg/mt² followed by 36.84% in subjects with BMI 25-29.99 kg/mt². Not only prevalence but severity also increased with an increase in BMI. According to Einhorn D et al OSA is more common in a population with higher BMI.¹⁵ This association is also supported by Young T et al and Grunstein R et al.^{29,30} Foster GD et al also did a study on obstructive sleep apnoea among obese patients with type 2 diabetes in which they found that in participants with AHI ≥5, BMI was the only significant predictor of severe OSA (OR 1.1; 95% CI 1.0–1.2; P<0.03). 11 Independent of other variables, a 1-unit increase in BMI was associated with a 10% increase in the predicted odds of severe OSA.

In our observations, we found a significant increase in prevalence of OSA with an increase in neck circumference but no significant increase in severity was found to increase in neck circumference. Pineda E et al correlated NC with severity of OSA. They concluded NC can be used to differentiate mild from moderate to severe OSA but NC does not show a significant correlation with AHI. Cross sectional analyses of the Sleep Heart Health Study data show that, in middle-aged and older adults, moderate to severe obstructive sleep apnoea, as defined as an AHI greater than or equal to 15 events per hour, is independently associated with BMI and neck circumference.²⁹

In the present study, we tried to show the relation between blood sugar level and prevalence of OSA. We found that there were more cases of OSA with high level of blood sugar. With blood sugar >200 the prevalence was 90.9%, in comparison to 22.7% in subjects with blood sugar level ≤150 mg/dl. We also found that with the increasing level of blood sugar, severity of OSA was also increasing, as in subjects with blood sugar level >200 mg/dl, 70% were associated with severe OSA while in subjects with blood sugar level ≤150 mg/dl, 80% were associated with mild OSA. There is growing evidence in support of an independent association between OSA and impaired glucose metabolism by study of Pamidi S et

al.³² These findings were also supported by a study of McNicholas WT et al.³³ Evidence from a recently published randomized, double-blind, placebo-controlled trial supports the hypothesis of a deleterious effect of OSA on glucose metabolism. OSA is associated with impaired fasting glucose, glucose intolerance, and type-2 diabetes, even after accounting age, sex, waist circumference, and obesity.³⁴ Although our data do not provide evidence for a causal link between increasing blood glucose and OSA.

In present study, however we found that the prevalence of OSA increased with the increased HbA1c but there was not any significant causal relationship. Severity of OSA also not increased significantly with increased HbA1c according to Aronsohn RS et al.³⁵ Compared to those without OSA, the adjusted mean HbA1c was 1.5% higher in those with mild OSA, 1.9% higher in those with moderate OSA, and 3.69% higher in severe OSA patients. Some recent studies also show no significant association between HbA1c and OSA.¹⁵

In studies of Wetter DW et al and Khoo SM et al cigarette smoking and alcohol have been suggested as possible risk factors for obstructive sleep apnoea. Findemiologic investigations show that current smoking is associated with a higher prevalence of snoring and obstructive sleep apnoea. Franklin KA et al in their study shows that even exposure to second-hand smoke has been independently linked with habitual snoring. Findemiological study shows that even exposure to second-hand smoke has been independently linked with habitual snoring.

In present study also, OSA was more prevalent in smokers and alcoholic subjects. Wetter DW et al stated that smokers are 2.5 times more likely to have OSA than non-smokers.³⁶ Ingestion of alcohol before sleep has been shown to increase upper airway collapsibility and the precipitate obstructive apnoeas and hypopnoea during sleep.

According to Peppard PE et al the consequences of OSA include hypertension, coronary artery disease.³⁹ The present study also shows that prevalence of HTN was 33.33%, 40% and 81.82% in mild moderate and severe OSA respectively and prevalence of IHD was 16.67%, 30% and 36.36% in mild, moderate, severe OSA respectively. However, hypertension did not have an independent association with OSA, though it was significantly more common in patients with OSA. This was also shown in the study of Reddy EV et al.³¹ With the advent of large epidemiologic studies and well-controlled clinical trials, there is now substantial proof that obstructive sleep apnoea does increases the risk for various cardiovascular endpoints, most notably hypertension

CONCLUSION

This study shows that OSA has a high prevalence in subjects with T2DM and identifies several factors that may be associated with its presence in the diabetic

population. Current guidelines for the management of patient with T2DM do not include evaluation for possible OSA, despite clear evidence from many studies that OSA can adversely affect many aspects of management and that treatment of OSA can yield considerable improvements in glycaemic control. OSA can be usefully and easily assessed in an outpatient setting by using a portable device such as Apnea link. Because treatment of OSA has the potential both to decrease BP and to improve glycaemic control, clinicians should increase patients' awareness of the signs and symptoms of OSA and refer for sleep studies when appropriate. Once diagnosed, patients should be encouraged to adhere to CPAP treatment in order to halt progression and prevent complications.

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REFERENCES

- 1. 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;328:1230-5.
- Udwadia ZF, Doshi AV, Lonkar SG, Singh CI. Prevalence of sleep-disordered breathing and sleep apnea in middle-aged urban Indian men. Am J Respir Crit Care Mede. 2004;169(2):168-73.
- 3. Sharma SK, Kumpawat S, Bangra A, Goeal A. prevalence and risk factor of OSA in population of Delhi. Chest. 2006;130(1):149-56.
- 4. Shah N, Roux F. The relationship of obesity and obstructive sleep apnoea. Clin Chest Med. 2009;30(3):455-65.
- 5. Levy P, Bonsignore MR, Eckel J. Sleep. Sleep-disordered breathing and metabolic consequences. Eur Respir J. 2009;34(1):243-60.
- 6. Aronsohn RS, Whitmore H, Van Cauter E, Tasali E. Impact of untreated obstructive sleep apnoea on glucose control in type 2 diabetes. Am J Respir Crit Care Med. 2010;181(5):507-13.
- Drager LF, Queiroz EL, Lopes HF, Genta PR, Krieger EM, Lorenzi-Filho G. Obstructive sleep apnoea is highly prevalent and correlates with impaired glycaemic control in consecutive patients with metabolic syndrome. J Cardiometab Syndr. 2009;4(2):89-95.

- 8. Tasali E, Mokhlesi B, Van Cauterv E. Obstructive sleep apnoea and type 2 diabetes: interacting epidemics. Chest. 2008;133(2):496-506.
- Janus ED, Watt NM, Lam KS, et al. Hong Kong Cardiovascular Risk factors Steering Committee. American Diabetes Association. The prevalence of diabetes, association with cardiovascular risk factors and implications of diagnostic criteria (ADA 1997 and WHO 1998) in a 1996 community based population study in Hong Kong Chinese. Diabetes Med. 2000;17(10):741-5.
- Ko GT, Tang JS. Prevalence of obesity, overweight and underweight in a Hong Kong community: the United Christian Nethersole Community Health Service (UCNCHS) primary health care program 1996-1997. Asia Pac J Clin Nutr. 2006;15(2):236-41.
- 11. Foster GD, Sanders MH, Millman R. Obstructive sleep apnoea among obese patients with type 2 diabetes. Diabetes Care. 2009;32(6):1017-9.
- 12. Ronksley PE, Hemmelgran BR, Heitman SJ. Obstructive sleep apnoea is associated with diabetes in slept subjects. Thorax. 2009;64(10):834-9.
- 13. Elmasry A, Lindberg E, Berne C. Sleep disordered breathing and glucose metabolism in hypertensive men: a population based study. J Intern Med. 2001;249(2):153-61.
- Garcia-Touchard A, Somers VK, Olson LJ, Caples SM. Central Sleep Apnoea: implication for congestive heart failure. Chest. 2008;133(6):1495-504.
- 15. Einhorn D, Stewart DA. Prevalence of sleep apnea in a population of adults with type 2 diabetes mellitus. Endocr Pract. 2007;13(4):355-62.
- Reddy EV, Kadhivaran T, Mishra HK, Sree Niwas V, Handa KK, Sinha S, et al. Prevalence and risk factor of OSA among middle aged urban Indian. A community based study. Sleep Med. 2009;10:913-8.
- 17. Katsumata K, Okadu T, Miyao M, Katsumata Y. High incidence of sleep apnoea syndrome in a male diabetic population. Diabetes Res Clin Pract. 1991;13:45-51.
- 18. Chasens ER, Weaver TE, Umlauf MG. Insulin resistance and obstructive sleep apnea: is increased sympathetic stimulation the link? Biol Res Nors. 2003;5:87-96.
- Punjabi NM, Shahar E, Redline S. Sleep disordered breathing, glucose intolerance and insulin resistance: The sleep heart health study, Am J Epidemiol. 2004;160;521-30.
- 20. Reichmuth KJ, Austin D, Skatrud JB, Young T. Association of sleep apnoea and type 2 diabetes: a population based study. Am J Respir Crit Care Med. 2005;172:1590-5.
- 21. Sleep related breathing disorders in adults; recommendations for syndrome definition and measurements techniques in clinical research. The report of an American Academy of Sleep Medicine Task force. Sleep. 1999;22:667-89.

- 22. Elmasry A, Janson C, Lindberg E. The role of habitual snoring and obesity in the development of diabetes, a 10-year-follow-up-study in a male population. J Intern Med. 2000;248:13-20.
- 23. Al-Delaimy WK, Manson JE, Willett WC. Snoring as a risk factor for type 2 DM: a prospective study, Am J Epidemiol. 2002;155:387-93.
- 24. Somers VK, White DP, Amin R, Abraham WT. Sleep Apnea and Cardiovascular Disease. J Am Coll Cardiol. 2008;52(8):686-717.
- 25. Resnick HE, Redline S, Shahar E, Gilpin A, Newman A, Walter R, et al. Diabetes and sleep disturbances finding from the Sleep Heart Health study. Diabetes Care. 2003;26:702-9.
- 26. Catterall JR, Calverley PM, Ewing DJ, Shapiro CM, Clarke BF, Douglas NJ. Breathing, sleep and diabetic autonomic neuropathy. Diabetes. 1984;33:1025-7.
- 27. Ficker JH, Dertinger SH, Siegfried W, Konig HJ, Peutz M, Sailer D, et al. Obstructive sleep apnea and diabetes mellitus the role of cardiovascular autonomic neuropathy. Eur Respir J. 1998;11:14-9.
- 28. Vgontzas AN, Papanicolaou DA, Bixler EO. Sleep apnoea and day time sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypocytokinemia. J Clin Endocrinol Metab. 2000;85:1151-8.
- 29. Young T, Shahar E, Nieto FJ. Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. Arch Intern Med. 2002;162(8):893-900.
- 30. Grunstein R, Wilcox I, Yang TS, Gould Y, Hedner J. Snoring and sleep apnea in men: association with central obesity and hypertension. Int J Obes Relat Metab Disord. 1993:17:533-40.
- 31. Pineda E, Fitelson D, Rahill J, O'donnell A, Kuru T, Waldhorn R, Ruse T. Assessing the Adjusted Neck Circumference Sleep Apnea Screening Score in Patients Undergoing Polysomnography for Suspected Sleep Apnea. CHEST Journal. 2011 Oct 1;140(4_MeetingAbstracts):812A.
- 32. Pamidi S, Aronsohn RS, Tasali E. Obstructive sleep apnoea: role in the risk and severity of diabetes. Best Pract Res Clin Endocrinol Metab. 2010;24:703-15.
- 33. McNicholas WT, Bonsigore MR. Sleep apnoea as an independent risk factor for cardiovascular disease: current evidence, basic mechanisms and research priorities. Eur Respir J. 2007;29:156-78.
- 34. Punjabi NM. The Epidemiology of adult obstructive sleep apnoea. Proc Am Thorac Soc, 2008;5:136-43.
- 35. Aronsohn RS, Whitmore H, Van Cauter E, Tasali E. Impact of untreated obstructive sleep apnoea on glucose control in type 2 diabetes. Am J Respir Crit Care Med. 2010:181:507-13.
- 36. Wetter DW, Young TB, Bidwell TR, Badr MS, Palta M. Smoking as a risk factor for sleep-disordered breathing. Arch Intern Med. 1994;154:2219-24.

- 37. Khoo SM, Tan WC, Ng TP, Ho CH. Risk factors associated with habitual snoring and sleep-disordered breathing in a multi-ethnic Asian population: a population-based study. Respir Med. 2004;98:557-66.
- 38. Franklin KA, Gislason T, Omenaas E, Jogi R, Jensen EJ, Lindberg E, et al. The influence of active and passive smoking on habitual snoring. Am J Respir Crit Care Med. 2004;170:799-803.
- 39. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-

disordered breathing and hypertension. N Engl J Med. 2000;342(19):1378-84.

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