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Original Article

Study of Obstructive Sleep Apnea in patients with Type 2 Diabetes Mellitus in Damietta

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ABSTRACT

Background: Diabetes mellitus is a common condition, that has a bidirectional relationship with sleep disorders. From one side, sleep disorders are associated with increased risk of insulin resistance development and from the other side, diabetes worsens sleep quality. The aim of this study is to estimate the risk to develop obstructive sleep apnea in patients with diabetes mellitus [type 2] and its associated factors.

Methodology: This was a nested case control study [cross sectional descriptive followed by case control study] carried out in Specialized Medical Hospital of Al-Azhar University diabetes mellitus outpatient clinic [OPC] and sleep disordered breathing unit OPC in chest department from January 2024 to September 2024 on 125 patients with type-2 diabetes.

Results: The current study showed that the mean age of the studied patients was 51.53 years. There was a statistically significant difference between obstructive sleep apnea [OSA] and non OSA cases in terms of hypertension and BMI, as OSA cases tended to have higher number of hypertension patients and higher body mass index [BMI] [P=0.001 and P=0.05; respectively]. Using Multivariate logistic regression analysis for independent predictors of OSA, we found that Chronic renal disease patients was the most common associated risk factor [OR=11.3, CI=1.9-57] heart failure [OR=5.4, CI=1.7-21.3] and then hypertension [OR=4.6, CI=1.5-15.5].

Conclusion: Physicians should be particularly cognizant of the likelihood of OSA in patients with type-2 diabetes, especially among individuals with higher BMI, chronic renal disease and hypertension. In addition to known predictors of OSA chronic renal disease and hypertension were identified as risk factors in patients with type 2 diabetes.

Keywords: **Obesity; Diabetes Mellitus; Sleep Disorders; Sleep Disordered Breathing.**

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INTRODUCTION

In the field of sleep medicine, obstructive sleep apnea [OSA] is a common condition. It affects about 3-7% of adult males and 2-5% of adult females. It is associated with significant morbidity and even mortality. More than half of patients with obesity [about 57%] are at higher risk for the development of OSA. Clinically, OSA is defined as repeated episodes of partial or complete upper airway obstruction during sleep time. It is associated with increased respiratory efforts, intermitted arterial oxygen [O_2] desaturation and decreased quality of sleep [sleep fragmentation]. OSA is associated with significant morbidity [e.g., increased risk for the development of cardiovascular and cerebrovascular diseases and congestive heart failure]. The complications of OSA is related mainly to intermitted oxygen desaturation [hypoxia], fragmentation and reduced quality of the sleep, increased activity of the sympathetic system and deoxygenated phenomena [1].

Diabetes Mellitus [DM] is a common condition that affects about 10.5% of adult populations worldwide and associated with higher morbidity and mortality. For instance, about 6.7 million deaths are due to DM each year and type 2 is responsible for up to 90% of all deaths. In addition, DM can lead to development of OSA and OSA in turn can lead to insulin resistance and development of type 2 DM. The intermittent hypoxia and sleep fragmentation are the main clinical features of OSA. Both can lead to alteration of glucose metabolism and neuroendocrine system [e.g., activation of sympathetic nervous system, changes in the hypothalamic pituitary axis [HPA], systemic inflammatory changes, adipokine disturbances and a state of oxidative stress]. All these changes can lead to insulin resistance [IR] and development of type 2 DM. on the other side, changes associated with type 2 DM [e.g., peripheral neuropathy, IR, leptin resistance and oxidative stress], all may affect the neuronal and mechanical control of the upper airway and respiratory muscles, with collapsibility of upper airway muscles during sleep and thus development of OSA [2].

Both diabetes mellitus and OSA share common risk factors [e.g., obesity, old age]. In addition, cardiovascular diseases [e.g., heart failure, arrhythmia, and coronary artery disease [CAD] share the same risk factors with diabetes and OSA. Furthermore, patients with diabetes and OSA had higher blood pressure, poor quality sleep and health related quality of life. Beside there is poor adherence of those patients with diabetes and OSA treatment. About 55% of diabetic patients had one diabetes-related comorbid condition which related to hypertension. On the other side, OSA leads to hypertension, hypertension-related complications and T2DM complications in 73%, 93.3%, and 52.1%, respectively [3].

Unfortunately, the majority of OSA patients are not aware of their disease, the diagnosis is usually accidental. About 83% of patients with diabetes mellitus suffer from undiagnosed OSA. OSA are widely distributed all over the world. However, data [prevalence, clinical manifestations and comorbidities] about the disease is little in developing countries, especially in African countries. Thus, screening is of utmost importance in general populations and in patients with higher risk [e.g., patients with diabetes mellitus]. Screening permits early diagnosis, proper treatment, guarding against complications and reduce health-related costs. A two-stage approach is usually used for OSA screening. The first is a questionnaire followed by a formal sleep study [4].

The overnight polysomnography is the gold-standard diagnostic approach for OSA. However, it is not readily available in all facilities. Thus, a screening questionnaire is a good screening tool to effectively identify patients at risk for OSA development. Many questionnaires are available; the most sensitive one is the STOP-BANG [SBQ]. It is validated, feasible and reliable tool for screening and triaging patients with higher risk to develop OSA. When compared to other available questionnaires [Berlin, Epworth Sleepiness Scale], it is a more acute screening tool for diagnosis of OSA [5].

This work aimed to assess the high-risk patients for OSA and its related factors in patients with type 2 diabetes mellitus, at Al-Azhar University Hospital [Damietta].

PATIENTS AND METHODS

Study design: This was a nested case control study [cross sectional descriptive followed by case control study] carried out in Specialized Medical Hospital of Al-Azhar University diabetes mellitus outpatient clinic [OPC] and sleep disordered breathing unit OPC in chest department from January 2024 to September 2024 on 60 patients with type-2 diabetes.

Inclusion Criteria included: Egyptian diabetic patients with age more than 18 years old.

Exclusion criteria included: Age <18 years old; non-Egyptian nationality; patients with Type 1 Diabetes Mellitus or other endocrinological disorders; patients with central sleep apnea or other sleep disorder.

Ethical Considerations: The study protocol was submitted, investigated and approved by the Institutional review board for research design and ethics. In addition, an informed consent was signed by each participant or his guardian. The collected data were used for the purpose of research and the study was completed according to codes of available research reporting guidelines.

A total number of 60 patients were evenly divided into two groups [30 patients each] according to the state of diabetes [controlled and uncontrolled]. Then patients with sure diagnosis of OSA were categorized according to severity [moderate and severe subgroups].

Methods

Study procedures: All participants underwent the following procedures:

History taking: All patients had a thorough history taking including: Name, age, sex, residence, occupation and co -morbidities

e.g. Hypertension, Diabetes mellitus history e.g. history, treatment, control, comorbidities.

Anthropometric assessment: weight, height, body mass index and neck circumference.

Tonsillar size score [6]*:*

Tonsil size was classified from 0 to 4. It contains 4 classes as the following: Tonsils of size 1 are tonsils that are concealed inside the pillars, Tonsil size 2 indicates a tonsil that extends to the pillars, Tonsil size 3 tonsils are visible behind the pillars, but not to the midline, Tonsil size 4 Tonsils that stretch to the midline are referred to as

Modified Malampati score was of 4 categories as follows: Class 1: fully visible tonsils, uvula, and soft palate. Class 2: the visible parts include hard and soft palate, upper portion of tonsils and uvula. Class 3: Soft and hard palate and base of the uvula are visible. Class 4: Only the hard palate is visible [7].

Friedman OSA score:

Based on palate position [as explained in Friedman Tongue Position] and tonsil scale, it was used as an OSA screening tool. Grade 0 [less than 20 kg/m 2], grade 1 [20–25 kg/m 2], grade 2 [25–30 kg/m 2], grade 3 [30–40 kg/m 2], and grade 4 [more than 40 kg/m2] are the BMI classifications**.** Then, using the following formula, based on the numerical values of these results, we calculate the OSA score [8].

Epworth sleepiness scale [ESS]*:* It is used to assess when you are sleepy during the day by calculating the likelihood of dozing in eight separate active and passive situations. scored 0-3 according to possibility of dozing as the following: **[**0 = would never doze, 1 = Slight chance of dozing**,** 2 = Moderate chance of dozing, 3 = High chance of dozing] [9].

Berlin questionnaire: The Berlin Questionnaire is a brief tool with 10 questions that comprises of 3 domains: [1] habitual snoring, [2] sleepiness, and [3] hypertension or BMI > 30 kg/m^2 [10].

STOP BANG questionnaire*:*

It was used for showing the patients for OSA. It involves of 8 items: **S=** snoring, **T**= tiredness, **O=** observed apnea during sleep, **P**= high blood pressure, **B**= body mass index ≥ 35kg/m^2, **A=** age ≥ 50 years, **N=** neck circumference ≥40 cm, **G=** gender [male gender is positive]. Each positive item gets one point and the sum is interpreted as **High risk of OSA:** ≥3 **[11].**

Full night attended polysomnography*:*

Polysomnography was done for all the patients and was evaluated for the probability of having OSA: In the laboratory, full night polysomnography will be performed using [SONMO screenTM plus, SOMNOmedics, Germany] with AASM standard montage, and studies was interpreted by the most recent manual scoring criteria version [12,13]**.** Diagnosis of OSA was determined by the diagnostic criteria of the international categorizing of the sleep disorders, third edition [ICSD-3] [14]**.**

Fasting and post prandial blood sugar: The American Diabetes Association [ADA] provides guidelines [not mandates] for blood glucose goals for people with diabetes, and the goals vary depending on when you're checking your glucose*:* Fasting [before eating the first meal of the day] and before meals: 80–130 mg/dl [4.4–7.2 mmol/L]. Postprandial [one to two hours after a meal]: Less than 180 mg/dl [10.0 mmol/L].

Statistical Analysis: The statistical tests were performed by the available software computer package known as **SPSS**. The 25th version was used. Data was anonymized by coding and then fed to the program. Data were presented by the following measures [mean and standard deviation for normally distributed quantitative data, frequency and percentages for categorical data]. Groups were compared by independent student "t" and Chi square tests for quantitative and categorical data respectively. Then patients with OSA were classified according to severity and possible risk factors were estimated and finally, multiple logistic regression was calculated to estimate these factors. P value < 0.05 was considered significant.

RESULTS

The present study was a prospective study conducted on 60 patients with diabetes mellitus as evaluated by history, fasting and postprandial blood sugar tests. The current study showed that the mean age was 53.1±0.53years, 32 patients were male [53.3%] and 28 [46.7%]were female. Regarding co-morbidities among the studied group, Hypertension was the most common co-morbidity between them 25[41.6%], then ischemic heart disease 16[26.6%], heart failure disease 11[18.3%], bronchial asthma 2 [3.3%], and renal diseases 6[10%]. Among the studied groups, the mean weight was 111.36±9.261kg, mean body mass index was 39.88±3.42 kg/m2,mean neck circumference was 44.75±6.28 cm, mean fasting blood sugar was 146.23±39.01, mean post prandial blood sugar was 255.62±51.94, mean glycosylated hemoglobin was 8.02±1.04, mean peripheral capillary oxygen saturation was 94.01±2.55, mean systolic blood pressure was 122.00±8.57 mmHg, mean diastolic blood pressure was 81.52±8.03 mmHg, 19 patients [31.7%] were on insulin and 41 patients [68.3%] were on oral hypoglycemic. Regarding the tonsil size, 16 patients were grade 0 [26.7%], 23 patients were grade 1 [38.3%], 11 patients were grade 2 [18.3%], 5 patients were grade 3 [8.3%] and 5 patients were grade 4 [8.3%]. Patients according to Friedman tongue position 5 patients [8.3%] grade I, 18 patients [20%] grade IIa, 18 patients [30%] were grade IIb, 19 patients [31.7%] grade III and 6 patient [10%] grade IV.

We classified the studied patients clinically by using: [1] OSA questionnaire: 5 patients were negative [8.3%], 42 patients were borderline [70%] and 13 patients were positive [21.7%]. [2] 3

patients were negative [5%] and 57 patients were positive [95%]. [3] Epworth scale: less than 10 in 42 patients [70%] and more than 10 in 18 patients [30%]. [4] Berlin questionnaire: less than 10 in 42 patients [70%] and more than 10 in 18 patients [30%]. [5] Polysomnography was normal in 48 patients [80%] and 12 patients [20%] had OSA divide into 3 were moderate [25%] and 9 patients were severe [75%].

The comparison between cases with controlled diabetes and cases with uncontrolled diabetes regarding demographic data and comorbidities revealed that patient who has uncontrolled diabetes has significant association with hypertension 18 [60%] Vs. 7 patients [23.3%] in controlled diabetes patients [p=0.004] and also a significant higher numbers of heart failure patients among uncontrolled patients 15 [50%] Vs. 2 patients [6.6] among the controlled diabetes patients [p<0.001]. On the other hand, there was no significant difference between the two groups regarding: ischemic heart disease, renal diseases, and liver diseases [p=0.2, p=0.39, p=0.285; respectively].

The comparison between cases with controlled diabetes and cases with uncontrolled diabetes regarding clinical data which revealed that there was a significant difference between the two groups only in the BMI [P=0.031] and Glycosylated hemoglobin [p= 0.0560], with no significant difference between controlled and uncontrolled diabetes patients regarding other clinical data [p=0.767, p=0.410, p=0.556, respectively]. the comparison between cases with controlled and cases with uncontrolled diabetes regarding clinical examination revealed no statistically significant differences in tonsil size and Friedman tongue position. [p= 0.531, p=0.314]. The comparison between cases with controlled and cases with uncontrolled diabetes regarding scores show significant in mean apnea hypopnea index which is 48.33 in patients with uncontrolled diabetes and 6.063 in controlled diabetes patients [p = 0.607, p=0.378, p= 0.87, p=0.247, p=0.328, p=0.13].

the comparison between moderate and sever OSA regarding clinical data that reveal significant difference in glycosylated hemoglobin, and NC [p=0.018, p= 0.05; respectively]. There was no other significant difference among the other clinical data [p p=.549, p=0.599, p= 0.329, p= 0.093]. There was no significant difference between the two groups regarding the tonsil size and FTP. In terms of the scores differences, there was only significant difference in apnea hypopnea index [p=0.014], no other significant difference [p=0.731, p=0.392, p=0.341, p=0.106, p=0.603, p=0.712].

Using Multivariate logistic regression analysis for independent predictors of OSA, we found that Chronic renal disease patients was the most common associated risk factor [OR=11.3, CI=1.9-57] heart failure [OR=5.4, CI=1.7-21.3] and then hypertension [OR=4.6, CI=1.5-15.5].

The comparison between controlled and uncontrolled diabetics revealed a significant difference in the overall OSA prevalence [p<0.001]. There was also a negative correlation between OSA prevalence and poor control in diabetics [P<0.001].

Table [1]: Demographic data among the studied groups

Demographic data		Study group [n=60]
Age/ years	Mean ± SD Min-Max	53.1±0.533 20-64
Sex	Male Female	32 [53.3%] 28 [46.7%]
Comorbidities	Hypertension. Ischemic heart disease Heart failure disease Bronchial asthma Liver disease Hypothyroidism Chronic renal disease	19 [31.7%] 12 [20%] 8 [13.3%] 10 [16.7%] 2 [3.3%] 4 [6.7%] 6 [10%]
Weight [kg]		111.36±9.26
Body mass index [kg/m^2]		39.88±3.42
Neck Circumference [cm]		44.75±6.28
Fasting Blood Sugar [mg/dL]		146.23±39.01
Post Prandial blood sugar [mg/dL]		255.62±51.94
Glycosylated hemoglobin%		8.02±1.04
peripheral capillary oxygen saturation[SPO2]		94.01±2.55
Systolic Blood Pressure [mm Hg]		122.00±8.57
Diastolic Blood Pressure [mmHg]		81.52±8.03
Insulin use	Yes No	19 [31.7%] 41 [68.3%]
Tonsil size	Median [Min-Max]	1 [0-4]
Tonsil size grade	G0 G1 G2 G3 G4	16 [26.7%] 23 [38.3%] 11 [18.3%] 5 [8.3%] 5 [8.3%]
FTP	GI GIIa GIIb GIII GIV	5 [8.3%] 12 [20%] 18 [30%] 19 [31.7%] 6 [10%]
OSA questionnaire [Mean ± SD]		7.21±0.92
OSA class	Negative Borderline Positive	5 [8.3%] 42 [70%] 13 [21.7%]
STOP BANG [Mean ± SD]		5.88±1.46
STOP BANG class	Negative Positive	3 [5%] 57 [95%]
Epworth	Normal Borderline Sleepiness	11 [5-24] 42 [70%] 18 [30%]
Berlin [Mean ± SD]		2.01±0.81
Berlin class	Low risk High risk	3 [5%] 57 [95%]
Apnea Hypopnea Index[Median [Min-Max]]		2.23 [0-110]
Polyomnography	Normal OSA: • Mild [5-14.9] • Moderate [15-29.9] • Sever [>30]	48 [80 %] 12 [20%] 0 [0%] 3 [25%] 9 [75%]

G: grade; FTP: Friedman tongue position; OSAH: Obstructive sleep apnea hypopnea, OSA: Obstructive sleep apnea.

Table [2]: Comparison between cases with OSA and cases without OSA:

	Variables	Cases with controlled diabetes [n=30]	Cases with uncontrolled diabetes [n=30]	p-value
Age/ years	Mean ± SD	52.07±8.311	54.916±2.906	0.313
Sex [n,%]	Male	12 [40%]	16 [53.33%]	0.369
	Female	18 [60%]	4 [47.67%]	
Hypertension		18 [60%]	7 [23.3%]	0.004*
Ischemic Heart Disease		9 [30%]	8 [26%]	0.2
Heart failure		15 [50%]	2 [6.6%]	<0.001*
Liver disease		2 [6.6%]	1 [1.3%]	0.285
Chronic renal disease		4 [13.2%]	4 [13.%]	8.922
Glycosylated hemoglobin		8.46±1.03	7.04±1.04	0.056
Insulin use	Yes	15 [50%]	9 [30%]	0.130
	No	15 [50%]	21 [70%]	
Weight		112.625±8.744	113.416±5.648	0.767
Body Mass Index		41.1667±2.918	40.208±3.155	0.031
Neck Circumference		46.583±5.054	44.833±6.727	0.410
peripheral capillary oxygen saturation		94.583±2.75	94.083±2.55	0.556
Tonsil size [Median [Min-Max]		1.00 [0.00-3.00]	1.00 [0.00-4.00]	0.486
Tonsil size	0	5 [16.6%]	7 [23.3%]	0.531
Grade	1	15 [50%]	10 [33.3%]	
	2	3 [10%]	7 [23.3%]	
	3	4 [13.3%]	3 [10 %]	
	4	3 [10%]	4 [13.3%]	
FTP	GI	5 [16.6%]	7 [23.3%]	0.314
	GIIa	15 [50%]	10 [33.3%]	
	GIIb	3 [10%]	7 [23.3%]	
	GIII	4 [13.3%]	3 [10 %]	
	GIV	3 [10%]	4 [13.3%]	
OSA [Mean ± SD]		7.3±1.018	7.5±0.905	0.607
OSA class	Negative	0 [0%]	3 [10 %]	0.378
	Borderline	23 [76.6%]	7 [23.3%]	
	Positive	7 [23.3%]	20 [66.66%]	
STOP BANG [Mean ± SD]		6.438±6	6.5±6.5	0.870
STOP BANG	Negative	5 [16.6%]	0 [0%]	0.247
	Positive	25 [83.3%]	30 [100%]	
Epworth	<10	22 [73.3%]	20 [66.6%]	0.328
	≥10	8 [26.7%]	10 [43.5%]	
Berlin [Mean ± SD]		2±2	2.4±0.9	0.13
Berlin class	Low risk	9 [30%]	0 [0%]	<0.001
	High risk	21 [70%]	30 [100%]	
AHI [Median[Min-Max]]		6.063±0.618	6.063±0.618	<0.001

t: student t-test, χ^2: chi square test, FET: Fischer exact test, * significant p <0.05

Table [3]: Comparison between moderate and sever OSA:

Clinical data	Moderate OSA [n=5]	Sever OSA [n=10]	p-value
Glycosylated hemoglobin	7.133±0.231	8.567±0.838	0.018*
Insulin use **Yes** **No**	 2 [66.66%] 1 [33.33%]	 4 [44.44%] 5 [55.55%]	0.549
Weight	114±4.183	115.6±8.51	0.599
Body Mass Index kg/m2	42.333±2.082	44.22±2.906	0.329
Neck Circumference cm	51.33±8.083	45±2.739	0.05*
Peripheral oxygen saturation po2	97±1	93.778±2.863	0.093
Tonsil size **Median [Min-Max]**	1.00 [0.00-3.00]	1.00 [0.00-3.00]	^{Z}0.241
FTP **GI** **GIIa** **GIIb** **GIII** **GIV** **GV**	 1 [33.33%] 2 [66.66%] 0 [0%] 0 [0%] 0 [0%] 0 [0%]	 0 [0%] 3 [33.33%] 3 [33.33%] 2 [22.22%] 1 [11.11%] 0 [0%]	^{MC}0.192
OSA Mean ± SD	7.44±1.014	7.66±0.577	0.731
OSA class Borderline Positive	 3 [60%] 2 [40%]	 8 [80%] 2 [20%]	0.392
STOP BANG Mean ± SD	6.667±1.118	6±0.1	0.341
Epworth Mean ± SD	12.556±2.068	11.667±0.577	0.106
Berlin Mean ± SD	2.33±1	2.667±0.577	0.603
Berlin class Low risk High risk	1 [20%] 4 [80%]	2 [20%] 8 [80%]	0.712
AHI Median [Min-Max]	55 [25-90]	30 [20-35]	0.014*

t: student t-test, FET: Fischer exact test, Z: Mann Whitney test; * Significant P < 0.05

Table [4]: Multivariate logistic regression analysis for independent predictors of OSA

Independent predictors	β	Std. Error	P value	OR [95% CI]
Hypertension	1.674	0.778	0.017	4.6 [1.5-15.5]
Heart failure	1.941	0.923	0.021	5.4 [1.7-21.3]
Chronic renal disease	2.315	0.825	0.04	11.3 [1.9-57]

OR: Odds ratio, CI: confidence interval

DISCUSSION

OSA is a highly prevalent comorbid condition in patients with diabetes mellitus. Generally, the prevalence of OSA witnessed

progressive increase due to increasing old aged populations and obesity [15]. This study aimed to examine the prevalence of OSA in Egyptian patients with T2DM. Moreover, we explored the associations between some basic and physiological measures and the risk of OSA among this population.

In our study a total of f 60 patients [30 with controlled diabetes and 30 with uncontrolled diabetics] were reported with mean age [53.1±0.533]. We found that 53.3% of total patients were males [32 patients] compared to 46.7% female patients [28 patients] with type 2 diabetes mellitus. Furthermore, there were 19 patients [31.7%] were on insulin and 41 patients [68.3%] were on oral hypoglycemic. Among the included patients, 48 patients [80%] were normal, while 12 patients [25%] were diagnosed as OSA using polysomnography. Old age is a known risk factor for the development of OSA. Adding diabetes mellitus and its comorbid conditions [e.g., obesity and hypertension] increasing the risk of OSA [16].

Previous studies tried to define the causes for age-related effect on OSA and its pathophysiological mechanisms. The suggested mechanisms for OSA increase in old age include changes in structures around the pharynx, increased length of soft palate and increased fat deposition in the para-pharyngeal area [1]. However, our study was not able to find a s significant differences between groups regarding patient age or gender. The lack of a significant linear relationship between OSA and age in patients with diabetes is interesting. The average age of diabetic patients was approximately 52 years old. However, previous studies reported a mean age of 65 and patients who developed OSA are usually younger than 65 years old [17]. However, a United Kingdom [UK]-based study showed a non-significant relationship between age and OSA [18]. But, there are epidemiological studies linked advanced age in diabetic patients with obstructive sleep apnea and sleep disordered breathing [SDB] [16,19,20].

In addition, previous study linked the male gender to the development of OSA. This was explained by elevated mass in the neck and torso[4]. However, others reported no significant relationship between male gender and development of OSA [21,22]. Our results did not reflect such relation. In addition, and similar to the current work, a previous study used polysomnography to assess sexes differences among diabetic patients found no significant association between gender and development of OSA [1].

Interestingly, another study reported higher incidence among female than males, suggesting higher susceptibility of women with type 2 diabetes than men to adverse effects of diabetes [18].

Obesity is by far the most reliable risk factor for OSA and diabetes mellitus. Every one-standard-deviation increase in BMI resulted in a fourfold increase in OSA prevalence, according to the Wisconsin sleep study.

Furthermore, the initiation of OSA or the worsening of pre-existing OSA is reliably predicted by weight gain[4].

Our study supports some of these plausible mechanisms by showing that obesity reflected by the mean BMI was significantly higher among OSA group than the normal group [p=0.05]. This is similar to the results reported by **Saad *et al.*** [1] where they find a higher level of BMI among OSA group than the non-OSA group [P=0.03].

OSA is likely to exacerbate glycemic regulation in type 2 diabetes patients due to its links to obesity, insulin resistance, and B-cell dysfunction. Obesity is a major confounder in OSA research, as it is in most fields. Some studies found that OSA and OSA strength are related to poorer glycemic regulation [both HbA1c and fasting plasma glucose] and glycemic variability and total sleep time after adjusting for age, sex, ethnicity, BMI, number of diabetes medications, amount of exercise, and years of diabetes [23].

OSA can also exacerbate DM regulation and lead to DM-related complications, according to research. Previous studies found that a higher severity of OSA was associated with impaired glycemic control in diabetic patients, as measured by HbA1c levels; this result was independent of confounders like BMI, race, age, gender, and the number of years with DM. The severity of hypoxemia in patients with OSA correlates with HbA1C levels ranging from normal to pre-diabetes and diabetes [24].

In contrast to this, our study found that there were no significant results between OSA and normal patients. This is supported by the previously reported results by **Saad *et al.*** [1] who did not find also a statistically significant difference between the two groups in their HbA1c.

Obese patients with SDB have a statistically significant higher neck circumference than obese patients without SDB, according to a survey of patients with class three obesity [25].

Also, **Reis *at al.*** reported that significant increase in neck circumference in patients with OSA versus those without OSA. Unlike our study that revealed no significant difference between OSA group and Non-OSA group in neck circumference [26]. OSA risk factors and comorbidities vary significantly across studies, owing to differences in the patients surveyed, research designs, and the procedure and criteria used to diagnose OSA [25]. Patients with Type 2 diabetes have a high prevalence of OSA, according to epidemiological evidence [27].

According to an epidemiological study previously done by **Tahrani *et al***, OSA is very common among patients with type 2 diabetes [8.5–85 %, 23.8–70 % for moderate to severe OSA] [28]. However, according to our study the prevalence of OSA in T2DM using polysomnograhy is 12%. It revealed that 110 [86%] was normal and only 15 patients [4%] had OSA, 5 patients [33.3%] were moderate OSA and 10 patients [66.7%] were severe OSA.

Our result is close to the results reported by a previous studies conducted in Nigeria and Jordan which reported high risk prevalence of OSA using berlin questionnaire among type 2 DM was 27% and 31% respectively [1,29].

On the other hand, the current finding was lower than a study done in Ethiopia, which showed that the prevalence of high risk OSA was 45.5% among their study participants [21].

This result was in line with findings from Saudi Arabia and India using SBQ which reported the prevalence of high risk OSA 48.6%, 45.8% and 47.3% respectively [22,30]. Moreover, the current finding was much lower than a study done in the USA 86%, and the UK 57% [31,32].

In addition, by completing a multi-center analysis involving secondary and tertiary hospitals in six Chinese regions, **Zhang *et al.*** found that among the 880 hospitalized patients with T2DM the frequency of overall OSA [AHI ≥ 5] 60.0% [56.8, 63.2%] [33]. The potential causes for high prevalence in these studies may be attributed to the different ethnic groups, different settings of the study and the use of different tools to recognize OSA. For example, we used polysomnography [the gold standard tool] whereas the UK-based study used the Berlin questionnaire for a large sample of studied population. Furthermore, the USA-based study was a multicenter study confined for obese subjects.

In the present work, OSA prevalence was much higher in comorbid hypertensive than normotensive subjects with the identification of hypertension as an independent predictor for OSA among type-2 diabetic patients [OR=4.6, CI=1.5-15.5]. These results are comparable to previous studies. **Subramanian *et al.*** preformed a retrospective study to estimate OSA incidence in adult subjects with or without type 2 DM matched for age, sex, and BMI. In a multivariate regression analysis, hypertension was identified as a significant predictor of OSA [1.32 [1.23–1.43]; $P < 0.001$]. Moreover, they also reported that diabetic patients with heart failure was 1.4 more susceptible to develop OSA [1.41 [1.18–1.70]; $P < 0.001$], and this comes in line with our finding, as we found that diabetic patients with heart failure was 5.4 times susceptible to develop OSA [OR=5.4, CI=1.7-21.3] [18].

Similarly, **Siwasaranond *et al.*** performed a prospective cohort study to explore the relationship between OSA and diabetes-related comorbid conditions and if this was mediated by hypertension. They found that subjects with moderate-to-severe OSA were 3.05 times more likely to have hypertension [34].

These results are also supported by **Nowakowska *et al.*** who studied the comorbidity burden of type 2 DM in England between 2007 and 2017. They reported that hypertension was identified as the most common risky condition among all patients, with higher prevalence in women than men [35]. The possible explanation for these results is that, OSA induces chronic hypoxia of intermittent nature, that leads to different systematic changes [e.g., exaggerated sympathetic activity, systemic inflammation, endothelial dysfunction and oxidative stress], which promote high blood pressure development [36].

Conclusion: The results of the current work adds to the available evidence supporting likelihood of OSA in patients with type 2 DM. Thus, physicians should be aware for this relationship, especially among obese individuals, subjects with chronic renal disease and hypertension. However, due to small sample size, the results must be treated with caution and future studies are highly recommended.

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REFERENCES

1. Saad AMJ, Hiyasat D, Jaddou H, Obeidat N. The prevalence of high risk obstructive sleep apnoea among patients with type 2 diabetes in Jordan. Diabetes Res Clin Pract. 2019 Jun; 152:16-22. doi: 10.1016/j.diabres.2019.04.035.

2.Aurora RN, Gaynanova I, Patel P, Punjabi NM. Glucose profiles in obstructive sleep apnea and type 2 diabetes mellitus. Sleep Med. 2022 Jul 1; 95:105–11. doi: 10.1016/j.sleep.2022.04.007.

3. Bouloukaki I, Schiza S, Tsiligianni I. Obstructive sleep apnea as an additive or even synergistic risk factor for cardiovascular disease in patients with type 2 Diabetes: A call for action in primary care? Diabetes Res Clin Pract. 2021 Aug; 178:108940. doi: 10.1016/j.diabres.2021.108940.

4.Agha M, Shehab-Eldin W, Helwa M. Obstructive sleep apnea in patients with type 2 diabetes mellitus. Egypt J Chest Dis Tuberc. 2019;68[4]:560. DOI: 10.4103/ejcdt.ejcdt_17_19

5.Althoff MD, Ghincea A, Wood LG, Holguin F, Sharma S. Asthma and Three Colinear Comorbidities: Obesity, OSA, and GERD. J Allergy Clin Immunol Pract. 2021;9[11]:3877–84. doi: 10.1016/j.jaip. 2021.09.003.

6. Friedman M, Ibrahim H, Bass L. Clinical staging for sleep-disordered breathing. Otolaryngol Head Neck Surg. 2002 Jul;127[1]:13-21. doi: 10.1067/mhn.2002.126477.

7. Mallampati SR, Gatt SP, Gugino LD, Desai SP, Waraksa B, Freiberger D, Liu PL. A clinical sign to predict difficult tracheal intubation: a prospective study. Can Anaesth Soc J. 1985 Jul;32[4]:429-34. doi: 10.1007/BF03011357.

8. Friedman M, Tanyeri H, La Rosa M, Landsberg R, Vaidyanathan K, Pieri S, Caldarelli D. Clinical predictors of obstructive sleep apnea. Laryngoscope. 1999 Dec;109[12]:1901-7. doi: 10.1097/00005537-199912000-00002.

9. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991 Dec;14[6]:540-5. doi: 10.1093/sleep/14.6.540.

10. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med. 1999 Oct 5;131[7]:485-91. doi: 10.7326/0003-4819-131-7-199910050-00002.

11. Chung F, Abdullah HR, Liao P. STOP-Bang Questionnaire: A Practical Approach to Screen for Obstructive Sleep Apnea. Chest. 2016 Mar;149[3]:631-8. doi: 10.1378/chest.15-0903. Epub 2016 Jan 12. PMID: 26378880.

12.Iber C, Ancoli-Israel S, Chesson A, Quan S. The AASM manual for the

scoring of sleep and associated events: Rules, terminology and technical specifications. 1st Edition 2007, American Academy of Sleep medicine. Westchester.

13. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, et al; American Academy of Sleep Medicine. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med. 2012 Oct 15;8[5]:597-619. doi: 10.5664/jcsm.2172.

14.AASM. AASM | Clinical Resources | International Classification of Sleep Disorders [Internet]. 2023 [cited 2024 Feb 23]. Available from: https://aasm.org/clinical-resources/international-classification-sleep-disorders/

15. Fallahi A, Jamil DI, Karimi EB, Baghi V, Gheshlagh RG. Prevalence of obstructive sleep apnea in patients with type 2 diabetes: A systematic review and meta-analysis. Diabetes Metab Syndr. 2019 Jul-Aug; 13 [4]:2463-2468. doi: 10.1016/j.dsx.2019.06.030.

16. Thompson C, Legault J, Moullec G, Baltzan M, Cross N, Dang-Vu TT, et al. A portrait of obstructive sleep apnea risk factors in 27,210 middle-aged and older adults in the Canadian Longitudinal Study on Aging. Sci Rep. 2022 Mar 24;12[1]:5127. doi: 10.1038/s41598-022-08164-6.

17. Farrell PC, Richards G. Recognition and treatment of sleep-disordered breathing: an important component of chronic disease management. J Transl Med. 2017;15[1]:114. doi: 10.1186/s12967-017-1211-y.

18. Subramanian A, Adderley NJ, Tracy A, Taverner T, Hanif W, Toulis KA, et al. Risk of Incident Obstructive Sleep Apnea Among Patients With Type 2 Diabetes. Diabetes Care. 2019 May;42[5]:954-963. doi: 10.2337/dc18-2004.

19.Chang JL, Goldberg AN, Alt JA, Mohammed A, Ashbrook L, Auckley D, et al. International Consensus Statement on Obstructive Sleep Apnea. Int Forum Allergy Rhinol. 2023 Jul 1;13[7]:1061–482. doi: 10.1002/alr.23079.

20. Bonsignore MR, Randerath W, Schiza S, Verbraecken J, Elliott MW, Riha R, et al. European Respiratory Society statement on sleep apnoea, sleepiness and driving risk. Eur Respir J. 2021 Feb 25;57 [2]:2001272. doi: 10.1183/13993003.01272-2020.

21. Abdissa D. Prevalence of obstructive sleep apnea risk and associated factors among patients with type 2 diabetes mellitus on follow up at Jimma Medical Center, Southwest Ethiopia. J Clin Transl Endocrinol. 2020 Aug 18; 21:100234. doi: 10.1016/j.jcte.2020.100234.

22.Alkhodaidi ST, Alobaylan MM, Alharthy LM, Almalki DM, Alswat K. Obstructive Sleep Apnea Risk Assessment among Type 2 Diabetes and its Relation to Neck Circumference. Int J Clin Ski. 2020;14 [1]:295–300.

23. Hu ST, Tsao LI, Yu CC, Liu CY. Development and validation of a Chinese version of the Sleep Apnea Quality of Life Index. Sleep Breath. 2020 Jun;24[2]:591-597. doi: 10.1007/s11325-020-02012-5.

24. Chang AC, Fox TP, Wang S, Wu AY. Relationship between obstructive sleep apnea and the presence and severity of diabetic retinopathy. Retina. 2018 Nov;38[11]:2197-2206. doi: 10.1097/IAE. 0000000000001848.

25. Morsy NE, Farrag NS, Zaki NFW, Badawy AY, Abdelhafez SA, El-Gilany AH, et al. Obstructive sleep apnea: personal, societal, public health, and legal implications. Rev Environ Health. 2019 Jun 26;34[2]:153-169. doi: 10.1515/reveh-2018-0068.

26. Reis R, Teixeira F, Martins V, Sousa L, Batata L, Santos C, Moutinho J. Validation of a Portuguese version of the STOP-Bang questionnaire as a screening tool for obstructive sleep apnea: Analysis in a sleep clinic. Rev Port Pneumol [2006]. 2015 Mar-Apr;21[2]:61-8. doi: 10.1016/j.rppnen.2014.04.009.

27.Yazdi MS, Samadi A, Akrami R. Prevalence of Obesity and Risk of Obstructive Sleep Apnea among People with Type II Diabetes Mellitus. J Sleep Sci [Internet]. 2019; 3 [1-2] SE-Original Article[s]]:10–6. Available from: https://jss.tums.ac.ir/index.php/jss/article/view/101

28. Tahrani A. Obstructive Sleep Apnoea and Vascular Disease in Patients with Type 2 Diabetes. Eur Endocrinol. 2015 Aug;11[2]:81-89. doi: 10.17925/EE.2015.11.02.81.

29. Obaseki DO, Kolawole BA, Gomerep SS, Obaseki JE, Abidoye IA, Ikem RT, Erhabor GE. Prevalence and predictors of obstructive sleep apnea syndrome in a sample of patients with type 2 Diabetes Mellitus in Nigeria. Niger Med J. 2014 Jan;55[1]:24-8. doi: 10.4103/0300-1652.128154.

30.Bamanikar A, Duggal S, Sharma S, Rana S. Assessment of risk for obstructive sleep apnea by using STOP-BANG questionnaire in type 2 diabetes mellitus. Int J Diabetes Dev Ctries. 2020 Apr 1;40[2]:173–7. doi: 10.1007/s13410-019-00768-5

31. West SD, Nicoll DJ, Stradling JR. Prevalence of obstructive sleep apnoea in men with type 2 diabetes. Thorax. 2006 Nov;61[11]:945-50. doi: 10.1136/thx.2005.057745.

32. Foster GD, Sanders MH, Millman R, Zammit G, Borradaile KE, Newman AB, et al.; Sleep AHEAD Research Group. Obstructive sleep apnea among obese patients with type 2 diabetes. Diabetes Care. 2009 Jun;32[6]:1017-9. doi: 10.2337/dc08-1776.

33. Zhang P, Zhang R, Zhao F, Heeley E, Chai-Coetzer CL, Liu J, et al. The prevalence and characteristics of obstructive sleep apnea in hospitalized patients with type 2 diabetes in China. J Sleep Res. 2016 Feb; 25[1]:39-46. doi: 10.1111/jsr.12334.

34. Siwasaranond N, Nimitphong H, Manodpitipong A, Saetung S, Chirakalwasan N, Thakkinstian A, Reutrakul S. The Relationship between Diabetes-Related Complications and Obstructive Sleep Apnea in Type 2 Diabetes. J Diabetes Res. 2018 Mar 7;2018: 9269170. doi: 10.1155/2018/9269170.

35. Nowakowska M, Zghebi SS, Ashcroft DM, Buchan I, Holt T, et al. The comorbidity burden of type 2 diabetes mellitus: patterns, clusters and predictions from a large English primary care cohort. BMC Med. 2019;17[1]:145. doi: 10.1186/s12916-019-1373-y.

36. Baguet JP, Barone-Rochette G, Tamisier R, Levy P, Pépin JL. Mechanisms of cardiac dysfunction in obstructive sleep apnea. Nat Rev Cardiol. 2012;9[12]:679-88. doi: 10.1038/nrcardio. 2012.141.

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