

Obstructive Sleep Apnea in Patients with Central Obesity, in Benha University Hospital

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ABSTRACT

Background: Central obesity is the most prevalent dietary illness in people. It predisposes a person to a complicated health issues, including stroke, type 2 diabetes, hypertension, non-alcoholic fatty liver disease, and obstructive sleep apnea syndrome (OSAS). OSA is a chronic, progressive condition that adversely affects quality of life. Due to lower lung capacity and tension on the pharynx, the degree of upper airway obstruction and hypoxemia owing to OSA seems to correlate more strongly with the amount of visceral fat (abdominal adiposity) than other clinical indicators of obesity.

Objectives: Studying the relationship between central obesity and OSA incidence and severity, as well as the underlying risk factors between their association. **Subjects and Methods:** This cross-sectional study included 364 adult subjects with central obesity who visited the Endocrinology Clinic of Benha University Hospital, Egypt, from February 2023 to May 2023. All participants were subjected to measurement of weight, height and waist circumference (WC), recording of blood pressure, laboratory investigations including: HBA1c, lipid profile, liver and kidney function tests and overnight polysomnography to assess OSA.

Results: OSA was present in 308 (84.6%). OSA severity was closely related to male sex. Moderate and sever OSA was significantly associated with male sex, old age, higher WC, higher BMI, diabetes, hypertension, high cholesterol, triglycerides & LDL. Logistic regression analyses between different variables and OSA showed that age was the most significant predictor for OSA ($p < 0.001$).

Conclusion: Prevalence and severity of OSA among candidates with central obesity was very high. Age, waist circumference, hypertension, disturbed lipid profile and T2DM were significantly associated with increased prevalence and severity of OSA among centrally obese individuals. So, early in the course of their follow-up, healthcare professionals should examine centrally obese patients for OSA.

Keywords: Central obesity, Obstructive sleep apnea (OSA).

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INTRODUCTION

Despite the fact that obesity was formerly believed to be an exclusive problem of high-income countries, recent reports have shown a rapid rise in overweight and obesity in many low- and middle-income countries (LMICs), particularly in several African nations [1]. Even if obesity poses a serious threat to one's health, how much body fat plays a significant role in assessing that hazard. Hence, using the body mass index (BMI) alone as a health indicator would not be acceptable because people with normal weight might occasionally have central obesity [2].

The term "central obesity" refers to a condition in which there is a significant buildup of visceral fat in the abdomen [3]. According to the World Health Organization (WHO), central obesity (also known as abdominal obesity) is defined as having a waist circumference (WC) of more than 94 cm for men and 80 cm for women, respectively [4]. The biggest predictor of a cluster of risk factors for chronic non-communicable diseases (NCD), independent of BMI, is WC as an indicator of central obesity, which is the most prevalent nutritional issue in humans and predisposes the individual to a complicated health problem [5].

Triglycerides (TGS), high-density lipoprotein cholesterol (HDL-C), hypertension, and diabetes all raise the risk of CVD. However, after adjusting for these

significant risk factor. This suggests that the major focus for the primary prevention of CVD is central adiposity. Moreover, central obesity has been connected to a number of health conditions, such as malignancies, stroke [6], type 2 diabetes mellitus, hypertension, nonalcoholic fatty liver disease, and all-cause mortality [7]. The incidence of obstructive sleep apnea (OSA), which affects 9% to 38% of the population generally and is more prevalent in males, is one of the most prevalent and dangerous sleep-related breathing diseases. The incidence of OSA rises with age, and in some senior populations, it has been estimated to be as high as 90% in men and 78% in women [8]. The syndrome is characterized by recurrent upper airway collapse during sleep, which can result in sleep fragmentation, oxygen desaturation, and autonomic dysfunction [9]. Specific symptoms of OSA in sufferers include excessive daily sleepiness, low moods, diminished memory and learning abilities, poor focus and attention span, and cardiovascular diseases, all of which can cause abrupt death. OSA is a chronic, progressive condition that has a severe impact on quality of life since sleep is a brain function that is essential to life and promotes relaxation [10].

Based on the findings of the polysomnography, OSA is determined. With the aim of non-invasively examining sleep disorders, the technique involves

simultaneously recording parameters such as heart and respiratory rates, respiratory flow and effort, pulse oximetry, the quantification of respiratory events (both obstructive and central ones), snoring, levels of brain electrical activity, eye movement, and muscle activity^[9]. OSA is characterized as having more than five episodes of partial (hypopnea) or total (apnea) blockage of the upper airway each hour despite attempts to breathe^[11]. The severity of OSA is determined by the apnea-hypopnea index (AHI), which categorizes it as mild (5–15 occurrences per hour), moderate (16–30 events per hour), and severe (>30 events per hour)^[12].

Obesity in the central (or abdominal) region is a recognized OSA risk factor. Adipocyte hormones like leptin and acute-phase proteins like CRP, as well as proinflammatory cytokines like IL-6 and TNF- α , are all strongly associated with body mass index in adults^[13]. The metabolically active organ abdominal adipose tissue contains resident macrophages that secrete large amounts of cytokines^[14]. Even after controlling for BMI, elevated levels of CRP and IL-6 have been seen in people with sleep disordered breathing (SDB)^[15]. Moreover, due to lower lung capacity and tension on the pharynx, the degree of upper airway obstruction and hypoxemia owing to OSA appears to correlate more strongly with the quantity of visceral fat than other clinical indicators of obesity^[16].

This study was conducted to examine the relationship between central obesity and the incidence and severity of obstructive sleep apnea as well as the underlying risk factors between them.

SUBJECTS AND METHODS

Study Design:

This is a cross-sectional study of adult patients who visited the endocrinology clinic in department of internal medicine, in Benha University Hospital, Egypt from February 2023 to May 2023.

Data Collection:

A total of 364 patients with established central obesity were enrolled in the study. The WHO defines central obesity as having a waist circumference (WC) larger than 94 cm for men and 80 cm for women, respectively^[4].

Inclusion criteria: Central obesity, defined as a waist circumference (WC) of more than 94 cm in men and 80 cm in women, respectively. Any gender aged 18-65 years old.

Exclusion criteria: Patients with malignancies, renal failure, cardiac ischemia illness, decompensated liver cirrhosis, and liver damage. Patients with chronic respiratory problems. Taking sedatives. Alcoholism or passive smoking within the last three years. Pregnancy.

For all subjects the following were recorded:

- 1) Demographic data about age, sex, height, weight, BMI, and waist circumference.

- 2) Systolic and diastolic blood pressure readings while supine and following a 10-minute rest period. We classified people as having hypertension if their blood pressure was above 140/90 mmHg and as not having hypertension if it was below 130/80 mmHg in accordance with the 2017 ACC/AHA hypertension guideline recommendations^[17].
- 3) Laboratory results of venous blood samples, which were collected after overnight fasting for over 8 hours, for testing of fasting plasma glucose, 2 h postprandial plasma glucose, HBA1c [Diabetes was defined as fasting plasma glucose levels $\geq$ 126 mg/dl or 2 h postprandial plasma glucose $\geq$ 200 mg/dl or HBA1c $\geq$ 6.5%^[18]], lipid profile and liver function and kidney function tests.
- 4) Assessment of obstructive sleep apnea (OSA): We used overnight polysomnography (SOMNO Screen Plus; SOMNO Medics GmbH, Rande sacker, Germany) in our study to assess whether the patients had OSA. The result of the overnight polysomnography was presented as an apnea hypopnea index (AHI), (AHI = apneas + hypopneas / total sleep time in hours). It is considered the gold standard for the diagnosis of OSA^[19]. Based on the AHI values, OSA was divided into three categories which were mild OSA (AHI 5 to 15 events/h), moderate OSA (AHI 15 to 30 events/h), and severe OSA (AHI $\geq$ 30 events/h)^[20].

Ethical consent: The Academic and Ethical Committee of Benha University approved the project (Rc 13-1-2023). Each patient signed a written informed consent form to agree to participate in the study. The study was conducted out in line with the Helsinki Declaration.

Statistical analysis

The SPSS software (for Windows, version 26.0) was employed. Mean $\pm$ SD were used to summarise quantitative data. Quantitative factors were compared between two groups using the Student t-test and ANOVA test. The Fisher exact test and the chi-square test were used, if necessary, to compare proportions between the various research groups. The threshold for statistical significance was $P \leq 0.05$.

RESULTS

364 patients with central obesity were enrolled in the study, 148 (40.6%) were males and 216 (59.4%) were females. OSA was present in 308 (84.6%). The detailed baseline characteristics were shown in table (1), which showed that the incidence of OSA was significantly higher in females and older age. OSA was also associated with significantly higher blood pressure, blood sugar and disturbed lipid profile. OSA was non-significantly associated with higher waist circumference and BMI than those with no OSA.

Table (1): Clinical characteristics of the studied population according to presence or absence of OSA

Variables	OSA	Non OSA	Total	Statistical test (St t)	P value
	N=308	N=56	N=364		
Age (years) mean $\pm$ sd	52.73 $\pm$ 9.85	46.57 $\pm$ 8.36	51.78 $\pm$ 9.88	4.4	<0.001**
Sex n (%)				X ² = 19.08	<0.001**
Male 148(40.61)	140(45.5)	8(14.3)	148(40.7)		
Female 216(59.39)	168(54.5)	48(85.7)	216(59.3)		
BMI (kg/m ²) mean $\pm$ sd	42.49 $\pm$ 9.71	41.07 $\pm$ 8.59	42.27 $\pm$ 9.55	1.03	0.306
WC (cm) mean $\pm$ sd	121.14 $\pm$ 24.92	120.79 $\pm$ 20.65	121.09 $\pm$ 24.28	0.101	0.92
SBP (mmHg) mean $\pm$ sd	140.39 $\pm$ 13.08	132.86 $\pm$ 13.98	139.23 $\pm$ 13.48	3.92	<0.001**
DBP (mmHg) mean $\pm$ sd	86.49 $\pm$ 8.03	82.14 $\pm$ 9.48	85.82 $\pm$ 8.41	3.62	<0.001**
HbA1c mean $\pm$ sd	6.08 $\pm$ 1.34	5.57 $\pm$ 1.25	6.0 $\pm$ 1.34	2.63	0.009**
Cholesterol (mg/dl) mean $\pm$ sd	209.77 $\pm$ 36.83	174.29 $\pm$ 24.66	204.31 $\pm$ 37.47	6.93	<0.001**
TG (mg/dl) mean $\pm$ sd	147.3 $\pm$ 22.57	137.64 $\pm$ 18.6	145.81 $\pm$ 22.26	3.02	0.003**
HDL (mg/dl) mean $\pm$ sd	42.42 $\pm$ 3.92	48.71 $\pm$ 2.62	43.38 $\pm$ 3.9	3.16	0.002**
LDL (mg/dl) mean $\pm$ sd	138.73 $\pm$ 42.8	97.71 $\pm$ 27.27	132.42 $\pm$ 43.38	6.92	<0.001**

Table (2) showed the relation between the studied clinical parameters and OSA severity. According to different AHI categories, moderate and sever OSA was significantly associated with male sex, old age, higher WC, higher BMI, diabetes, hypertension, high cholesterol, triglycerides & LDL. Median (IQR) of OSA severity for Male =30.0 (15.0-56.0) while that of female =16.5 (6.0-43.0) (Figure 1).

Table (2): Clinical characteristics of the studied population in relation to AHI categories

Variables	AHI quartiles				Statistical test (ANOVA)	P value
	Normal	Mild 5.1-	Moderate	Severe $\geq$ 30		
	0-5.0	14.9	15.0-29.9			
	N=56	N= 76	N= 84	N=148		
Age (years) mean $\pm$ sd	46.57 $\pm$ 8.36	44.11 $\pm$ 12.31	56.95 $\pm$ 5.95	54.76 $\pm$ 7.23	44.36	<0.001**
Sex n (%)					X ² = 27.84	<0.001**
Male 148(40.61)	8(14.3)	28(36.8)	32(38.1)	80(54.1)		
Female 216(59.39)	48(85.7)	48(63.2)	52(61.9)	68(45.9)		
BMI (kg/m ²) mean $\pm$ sd	41.07 $\pm$ 8.59	39.79 $\pm$ 7.24	43.86 $\pm$ 12.96	43.11 $\pm$ 8.38	3.21	0.023*
WC (cm) mean $\pm$ sd	120.79 $\pm$ 20.65	126.89 $\pm$ 23.12	132.62 $\pm$ 29.17	111.68 $\pm$ 19.0	17.21	<0.001**
Cholesterol (mg/dl) mean $\pm$ sd	174.29 $\pm$ 24.66	181.68 $\pm$ 26.33	194.95 $\pm$ 20.97	232.59 $\pm$ 34.25	87.69	<0.001**
TG (mg/dl) mean $\pm$ sd	137.64 $\pm$ 18.6	132.26 $\pm$ 28.17	143.24 $\pm$ 23.8	157.32 $\pm$ 10.56	32.0	<0.001**
HDL (mg/dl) mean $\pm$ sd	48.71 $\pm$ 12.62	49.79 $\pm$ 17.01	47.86 $\pm$ 14.34	35.54 $\pm$ 6.94	34.05	<0.001**
LDL (mg/dl) mean $\pm$ sd	97.71 $\pm$ 27.27	105.16 $\pm$ 36.88	121.95 $\pm$ 22.10	165.49 $\pm$ 37.3	91.22	<0.001**
Diabetes Mellitus n (%)					X ² = 52.19	<0.001**
Yes	12(21.4)	16(21.1)	20(23.8)	88(59.5)		
No	44(78.6)	60(78.9)	64(76.2)	60(40.5)		
Hypertension n(%)					X ² = 44.77	<0.001**
Yes	16(28.6)	24(31.6)	44(52.4)	104(70.3)		
No	40(71.4)	52(68.4)	40(47.6)	44(29.7)		

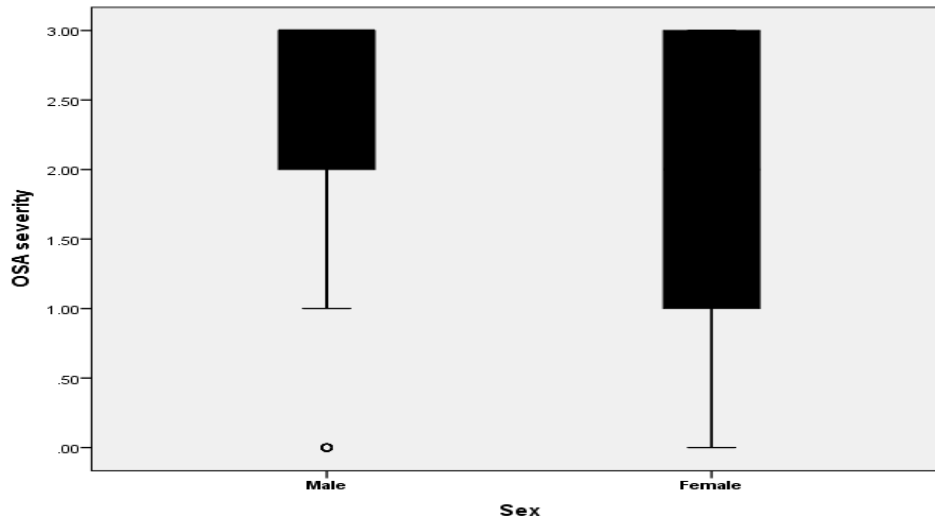


Figure (1): Pox plot of OSA severity difference between males and females

Table (3) showed that, the link between the AHI and age, BMI, waist circumference, systolic and diastolic blood pressure, HbA1c, TGs, and LDL ($p < 0.001$) was highly statistically significant. AHI score and HDL had a strong negative connection that was statistically significant ($p < 0.001$).

Table (3): Correlation between AHI and clinical and laboratory variables among patients with central obesity ($n=364$)

Variables & AHI	Correlation coefficient	P value
Age (years)	0.254	$<0.001^{**}$
BMI (kg/m^2)	0.193	$<0.001^{**}$
WC (cm)	0.251	$<0.001^{**}$
SBP (mmHg)	0.275	$<0.001^{**}$
DBP (mmHg)	0.248	$<0.001^{**}$
HbA1c	0.432	$<0.001^{**}$
Cholesterol (mg/dl)	0.714	$<0.001^{**}$
TG (mg/dl)	0.428	$<0.001^{**}$
HDL (mg/dl)	-0.443	$<0.001^{**}$
LDL (mg/dl)	0.712	$<0.001^{**}$

In table (4) we studied logistic regression analyses between different variables and OSA and we found that age was the most significant predictor for OSA ($p < 0.001$).

Table (4): Multivariate logistic regression analysis of factors associated with OSA

Variables & AHI	Exp (b)	P value	LL 95%CI	UL 95%CI
Age (years)	1.08	$<0.001^{**}$	1.04	1.118
SBP (mmHg)	0.999	0.963	0.963	1.037
DBP (mmHg)	1.04	0.193	0.98	1.107
HDL (mg/dl)	1.05	0.272	0.963	1.142
LDL (mg/dl)	1.07	0.064	0.996	1.165
Cholesterol (mg/dl)	0.97	0.467	0.894	1.053
TG (mg/dl)	0.99	0.575	0.954	1.026

DISCUSSION

Obstructive sleep apnea is characterized by repeated partial or total closure of the upper airway during sleeping. Despite the fact that patients with central obesity frequently have obstructive sleep apnea, the majority of these patients go undetected since the test is so expensive and there are so few sleep clinics, especially in poor countries. This study aimed to assess the relationship between central obesity and the incidence of obstructive sleep apnea and its associated factors among patients at Benha University Hospital.

The findings of our study revealed a very high prevalence of OSA in patients with central obesity as 84.6% of the study participants were diagnosed as having OSA. This is nearly similar to the results obtained by **Modena et al.** [21] who reported the prevalence of high risk OSA was 80.34% when they assessed OSA in Brazilian patients undergoing bariatric surgery in a comprehensive preoperative program. There was also high prevalence of OSA in the study of **Ma et al.** [22] who studied the relationship between the distribution of abdominal fat tissue and obstructive sleep apnea in obese Chinese individuals as it was 51.06%. In contrast to our results, the incidence of high risk OSA was only 7.8% in the study by **Wosu et al.** [23] who examined the connection between Chilean college students' overall and central obesity and their elevated risk for obstructive sleep apnea, as their study participants were young and healthy college students.

Although most of our study participants were females, OSA severity was closely related to male sex, which was coincident with many other studies [24]. Another study done previously in sleep clinic of Benha University Hospital also proofed that OSA severity was highly associated with male sex [25]. **Gaines et al.** [25] reported that OSA severity was associated with male sex when they assessed the relation between inflammation and visceral adiposity and obstructive sleep apnea in USA adolescents. A more recent study

conducted by **Lee et al.** [26] confirmed the association between male sex and high risk OSA when they evaluated the risk of sleep apnea among Asian Americans with abdominal obesity. Some studies had different opinion as they found that sex had no significant relation to OSA severity in their studies [21, 23]. The hormonal profile, morphological, physiological, and craniofacial alterations, as well as structural variations in the upper airways in men and women during sleep, have all been linked to connections between OSA and the male gender. This may happen as a result of the way that male body fat is distributed, which suggests that there is an accumulation of fat tissue in the upper portion of the male body, encouraging a larger collapse of the upper airways [27, 28].

We also discovered that OSA prevalence and severity was significantly associated with higher BMI, WC, older age, higher levels of glycated hemoglobin, blood lipids and blood pressure. This agrees with many other studies [21-26]. **Wondie et al.** [29] who evaluate type 2 diabetes mellitus patients' risk for obstructive sleep apnea and its related variables at Wolkite University in Southern Ethiopia, also found that BMI, WC, blood lipids and blood pressure were associated with OSA independent of T2DM.

Visceral obesity increases the risk of OSA because it decreases lung capacity and thoracic compliance, which requires more effort during inspiration and results in pharyngeal blockage [30]. Due to hormone dysregulation and physical inactivity brought on by daytime drowsiness, OSA causes weight gain primarily [31]. The two hallmarks of OSA, intermittent hypoxia and sleep fragmentation, impair the neuroendocrine system, which disrupts glucose metabolism. Insulin resistance and T2DM are brought on by sympathetic nervous system activation, hypothalamic-pituitary axis changes, adipokine abnormalities, systemic inflammation, and oxidative stress [32]. Because of alterations in salt and water metabolism and reduced baroreceptor sensitivity, OSA patients may have higher systemic arterial blood pressure. As a result of the carotid chemoreceptors being stimulated by the apnea, there is a drop in oxygen saturation and a rise in carbon dioxide levels, which raises systemic blood pressure and peripheral vascular resistance [33]. An additional factor in intermittent hypoxia is dyslipidemia. Continuous positive airway pressure (CPAP) therapy for OSA decreases blood pressure, enhances lipid profile, and improves glucose metabolism [34].

The overall age-related reduction in upper airway lumen size may be the connection between the severity of OSA, central obesity, and advanced age. The extension of the pharyngeal airway and the fall of the hyoid bone, which increases pharyngeal resistance, are structural alterations to the upper airway's dimensions. Increased comorbidities and a higher percentage of body fat with age may possibly be responsible for an increased risk of OSA and greater collapsibility. Old age is also accompanied with muscular and neurological

loss of upper airway muscle tone [35].

STUDY LIMITATION

First, due to the cross-sectional character of our study, cause-and-effect correlations between the variables were not examined. We were unable to tell whether central obesity came before OSA or vice versa.

Secondly, we were unable to assess the effects of changes in central adiposity during follow-up since the obtained anthropometric data were only evaluated at baseline. Furthermore, the study was limited in its ability to evaluate anthropometric traits with the risk of developing OSA due to the use of a single population sample profile, central obesity. However we were able to determine that differences in BMI and waist circumference may change the risk of OSA: the higher these values, the higher the risk of OSA.

Third, we did not test whether a decrease in waist size and abdominal fat would result in an improvement in OSA.

Fourth, although several other studies have demonstrated that inflammation mostly accounts for the link between visceral adiposity and OSA in adolescents, we did not assess inflammatory markers such as CRP, IL6, TNF, and leptin.

Finally, Only Egyptians residing in and around Benha area were included in the research. Thus, other Egyptian regions may have different results from ours.

CONCLUSION

This study came to the conclusion that individuals with central obesity had a very high incidence and severity of OSA, which had not been identified before during regular hospital visits. Age, waist circumference, hypertension, a disturbed lipid profile, and type 2 diabetes (T2DM) were all substantially linked to a higher prevalence and severity of OSA in those who were centrally obese. As a result, the findings of the study point to the necessity for early OSA screening by healthcare professionals in the case of central obesity. Health promotion and disease prevention methods on the significance of keeping a healthy weight, making dietary adjustments, altering one's lifestyle, and exercising should be part of the medical treatment provided to people with central obesity.

To help patients reduce belly fat and prevent central obesity, guidelines should include recommendations and intervention programs. Health education modules that may be utilized to increase information about sleep disorders should also be included in the strategies. In order to give fuller information and higher generalizability, future research on this topic should employ objective measures to explore the relationships between sleep apnea risk, WC, abdominal obesity, and subsequent medical issues among Egyptians throughout diverse locations.

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REFERENCES

- Ford N, Patel S, Narayan K (2017):** Obesity in low-and middle-income countries: burden, drivers, and emerging challenges. *Annu Rev Public Health*, 38: 145–64.
- Owolabi E, Goon D, Ter Adeniyi O (2017):** Central obesity and normal-weight central obesity among adults attending healthcare facilities in Buffalo City Metropolitan Municipality, South Africa: a cross-sectional study. *J Heal Popul Nutr.*, 36: 1–10.
- Molla M, Wolde H, Atnafu A (2020):** Magnitude of central obesity and its associated factors among adults in urban areas of Northwest Ethiopia. *Diabetes Metab Syndr Obes.*, 13: 4169–4178.
- WHO (2011):** Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation, Geneva, Pp. 8–11. http://www.who.int/entity/nutrition/publications/obesity/WHO_report_waist_circumference_and_waist_hip_ratio/en/
- Reis J, Macera C, Araneta M et al. (2009):** Comparison of overall obesity and body fat distribution in predicting risk of mortality. *Obesity*, 17 (6): 1232–9.
- Seo M, Kim Y, Han K et al. (2018):** Prevalence of obesity and incidence of obesity-related comorbidities in Koreans based on national health insurance service health checkup data 2006–2015. *J Obes Metab Syndrome*, 27 (1): 46. doi: 10.7570/jomes.2018.27.1.46
- Schetz M, De Jong A, Deane A et al. (2019):** Obesity in the Critically Ill: A Narrative Review. *Intensive Care Med.*, 45 (6): 757–69.
- Senaratna C, Perret J, Lodge C et al. (2017):** Prevalence of Obstructive Sleep Apnea in the General Population: A Systematic Review. *Sleep Med Rev.*, 34: 70–81.
- Benjafield A, Ayas N, Eastwood P et al. (2019):** Estimation of the global prevalence and burden of obstructive sleep apnea: a literature-based analysis. *Lancet Respir Med.*, 7 (8): 687–698.
- Faber J, Faber C, Faber A (2019):** Obstructive sleep apnea in adults. *Dental Press J Orthod.*, 24 (3): 99–109.
- Turnbull C, Wang S, Manuel A et al. (2018):** Relationships Between MRI Fat Distributions and Sleep Apnea and Obesity Hypoventilation Syndrome in Very Obese Patients. *Sleep Breath*, 22 (3): 673–81.
- Young T, Finn L, Peppard P et al. (2008):** Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep*, 31 (8): 1071–8.
- Gami A, Olson E, Shen W et al. (2013):** Obstructive sleep apnea and the risk of sudden cardiac death: a longitudinal study of 10,701 adults. *J Am Coll Cardiol.*, 62 (7): 610–6.
- Fain J (2006):** Release of interleukins and other inflammatory cytokines by human adipose tissue is enhanced in obesity and primarily due to the non-fat cells. *Vitam Horm.*, 74: 443–477.
- Tauman R, O'Brien L, Gozal D (2007):** Hypoxemia and obesity modulate plasma C-reactive protein and interleukin-6 levels in sleep-disordered breathing. *Sleep Breath*, 11: 77–84.
- Owens R, Malhotra A, Eckert D et al. (2010):** The Influence of End-Expiratory Lung Volume on Measurements of Pharyngeal Collapsibility. *J Appl Physiol.*, 108 (2): 445–51.
- Whelton P, Carey R, Aronow W et al. (2018):** 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol.*, 71: 127–248.
- Classification and Diagnosis of Diabetes (2022):** Standards of Medical Care in Diabetes—2022. *Diabetes Care*, 45 (1): 17–38.
- Kapur V, Auckley D, Chowdhuri S et al. (2017):** Clinical Practice Guideline for Diagnostic Testing for Adult Obstructive Sleep Apnea: An American Academy of Sleep Medicine Clinical Practice Guideline. *J Clin Sleep Med.*, 13 (3): 479–504.
- American Academy of Sleep Medicine (2008):** Obstructive sleep Apnea. Illinois: American Academy of Sleep Medicine, Pp. 1–4. <https://aasm.org/resources/factsheets/sleepapnea.pdf>
- Modena D, Cazzo E, Cândido E et al. (2017):** Obstructive sleep apnea syndrome among obese individuals: A cross-sectional study. *Rev Assoc Med Bras.*, 63 (10): 862–68.
- Ma B, Li Y, Wang X et al. (2022):** Association between abdominal adipose tissue distribution and obstructive sleep apnea in Chinese obese patients. *Front Endocrinol.*, 13: 847324. doi: 10.3389/fendo.2022.847324
- Wosu A, Vélez J, Barbosa C et al. (2014):** The Relationship between High Risk for Obstructive Sleep Apnea and General and Central Obesity: Findings from a Sample of Chilean College Students. *International Scholarly Research Notices*, 14: 871681. <http://dx.doi.org/10.1155/2014/871681>
- Negm M, Kamel M, Ebrahim W et al. (2022):** Evaluation of Obstructive Sleep Apnea in Metabolic Syndrome. *The Egyptian Journal of Hospital Medicine*, 88: 3417–3422.
- Gaines J, Vgontzas A, Fernandez-Mendoza J et al. (2016):** Inflammation mediates the association between visceral adiposity and obstructive sleep apnea in adolescents *Am J Physiol Endocrinol Metab.*, 311: 851–858.
- Lee S, Soomin R, Lee G et al. (2023):** Risk of Sleep Apnea Is Associated with Abdominal Obesity Among Asian Americans: Comparing Waist-to-Hip Ratio and Body Mass Index. *Journal of Racial and Ethnic Health Disparities*, 9: 1–11.
- Daltro C, Fontes F, Santos-Jesus R et al. (2006):** Obstructive sleep apnea and hypopnea syndrome (OSAHS): association with obesity, gender and age. *Arq Bras Endocrinol Metab.*, 50 (1): 74–81.
- Young T, Peppard P, Gottlieb D (2002):** Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med.*, 165 (9): 1217–39.
- Wondie A, Taderegew M, Girma B et al. (2022):** Obstructive sleep apnea risk and its associated factors among type 2 diabetes mellitus patients at wolkite university specialized hospital, Wolkite, Southern Ethiopia 2021. A comparative cross-sectional study. *Diabetology & Metabolic Syndrome*, 14:157. <https://doi.org/10.1186/s13098-022-00931-9>
- Mesarwi O, Sharma E, Jun J et al. (2015):** Metabolic dysfunction in obstructive sleep apnea: a critical examination of underlying mechanisms. *Sleep and Biological Rhythms*, 13: 2–17.
- Romero-Corral S, Caples F, Somers V (2010):** Interactions between obesity and obstructive sleep apnea: implications for treatment. *Chest*, 137: 711–719.
- Malik J, Masoodi S, Shuib S (2017):** Obstructive sleep apnea in type 2 diabetes and impact of continuous positive airway pressure therapy on glycemic control. *Indian J Endocrinol Metab.*, 21 (1): 106–12.
- Lima A, Franco C, Castro C et al. (2008):** Obstructive sleep apnea contribution to oxidative stress in obesity. *Arq Bras Endocrinol Metabol.*, 52 (4): 668–76.
- Mayer G, Arzt M, Braumann B et al. (2017):** German S3 guideline nonrestorative sleep/sleep disorders, chapter sleep-related breathing disorders in adults, short version. *Somnologie*, 21 (4): 290–301.
- Glasser M, Bailey N, McMillan A et al. (2011):** Sleep apnoea in older people. *Breathe*, 7: 248–256.