Age Related Effects of Sleep Apnea in Adults: A Polysomnographic Study

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ABSTRACT

Background: Age is a risk factor of obstructive sleep apnea (OSA). It has been shown that OSA progresses over time, full-night polysomnographic sleep study is considered to be the gold standard method for the diagnosis of obstructive sleep apnea syndrome (OSAS).

Objective: This study aims to determine the effect of age on sleep apnea patients.

Patients and Methods: A cross sectional study that included 95 patients conducted at a sleep laboratory in the ENT Department, Sohag University Hospital and in the Clinical Neurophysiology Unit, Cairo University. The study population age range was (20-60). All participants were subjected to personal history taking, detailed sleep history, medical history, general examination, ENT examination, Epworth sleepiness score (ESS), and polysomnography.

Results: Mean age was (43.99 ± 10.96). The male-to-female ratio was 1.7/1.3. Mean BMI was 35.27 ± 7.19. Mallampati score of grade II and III represented the majority of the patients (83.1%). The range of oropharyngeal collapse (tonsil collapse) was ranging from (no collapse-100%), with a mean of (62.31±25.50). The mean value of the Epworth sleep scale score in all patients was 8.09 ± 4.26. The mean of apnea hypopnea index (AHI) was 24.08 ± 28.33. Average O_2 was 82.73 ± 31.61. A statistically significant linear correlation was observed between age and BMI, the circumference of the waist and neck, duration of complaints, and collapse of the tonsils ($p < 0.05$).

Conclusion: Prevalence estimates of OSA increased continuously with age. Obesity could play an important role in elderly patients with OSAS and BMI could be a significant variable in determining the severity of OSAS in elderly patients.

Keywords: Body Mass Index, Obstructive sleep apnea, Polysomnography, Sleep apnea syndromes.

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is thought to affect 3% to 7% of adult men and 2% to 5% of adult women. It is marked by recurring episodes of total or partial upper airway obstruction that occur during sleep **(1)**. The diagnostic criteria of OSAS must have both subjective symptoms and five or more obstructive breathing events per hour night sleep measured by overnight monitoring. Apneas, hypopneas, and respiratory effort-related arousals (RERAs) are all examples of obstructive breathing events **(2)**.

High BMI, male sex, elderly age, supine positioning during sleep, and structural abnormalities in the upper airway are all risk factors for OSAS **(3)**. OSAS prevalence appears to rise consistently with age, peaking in aging individuals before plateauing about 65 years of age. Fat deposits in the pharyngeal area and gradual somatic deposition in adipose tissue mass, lengthening of the upper airway structures, and other age-related anatomic changes could all be contributing to the constant rise in occurrence **(4)**.

For diagnosing OSAS, a full-night polysomnographic sleep study is considered the gold standard. It is consisting of continuous polygraphic recording from surface leads (for electroencephalography, electrooculography, electromyography of the chin/legs, and electrocardiography), thermistors (for nasal/oral airflow), thoracic/abdominal impedance belts (for respiratory effort), and position sensors (for sleep position), as well as pulse oximetry (for SpO_2) and audio recording through a tracheal microphone (for snoring). Polysomnographic recordings were manually evaluated and interpreted according to established guidelines by an experienced sleep physician **(5)**. OSA has been linked to the development of hypertension, myocardial ischemia, congestive heart failure, arrhythmias, stroke, and cerebrovascular illness in many reports **(6)**.

The diagnosis of OSA is based on clinical signs and symptoms that were validated with a thorough sleep evaluation that included a sleep-oriented history, physical examination, and sleep test findings. OSA is defined as symptoms (snoring, observed apneas, gasping/choking episodes, increased sleepiness) occurring in the presence of at least five obstructive respiratory events per hour of sleep (apneas, hypopneas, or arousals due to respiratory effort), according to the International Classification of Sleep Disorders. Because of the stronger link of obstructive frequency with significant outcomes such as increased risk of cardiovascular disease, the occurrence of 15 or more obstructive respiratory episodes per hour of sleep in the absence of sleep-related symptoms is also appropriate for the diagnosis of OSA **(7)**.

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Signs of upper airway stenosis or the presence of other abnormalities that increase the risk of OSA should be investigated. A neck circumference (NC) greater than 17” in men and 16” in women, a BMI of 30 kg/m^2, a modified Mallampati score of 3 or 4, or the presence of retrognathia, lateral peritonsillar narrowing, macroglossia, tonsillar hypertrophy, elongated/enlarged uvula, high arched palate, and nasal abnormalities or overjet all risk factors that may indicate OSA [(8)]**.** This study aims to determine the effect of age on sleep apnea patients.

PATIENTS AND METHODS

This is a cross sectional study that included 95 patients that was conducted at a sleep laboratory in the ENT Department, Sohag University Hospital and in the Clinical Neurophysiology Unit, Cairo University.

Inclusion criteria: Subjects aged 20 years or older complaining from snoring with or without sleep apnea.
Exclusion criteria: Patients younger than 20 years old or > 60 years old.

Ethical consent:

An approval of the study was obtained from Sohag University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of sharing in the study.

This work was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

All participants were subjected to:

- **Personal history taking**: Age, gender, occupation, smoking.
- **Detailed sleep history:** Duration of the complaint of snoring, attacks of sleep apnea (present or not present), duration, number of attacks per night, sleep fragmentation and arousals, unrefreshing sleep, gasping during sleep, depressions, headache, decreased work performance, and dry mouth, excessive daytime fatigue or sleepiness.
- **Medical history**: History of diabetes, history of hypertension, and history of gastroesophageal reflux (GERD).

Physical examination:

- **General examination:** general characteristics of patients with OSA are evident on examination, such as obesity, macroglossia, micrognathia, and maxillary hypoplasia.
- **Body mass index (BMI)** is defined as the body mass divided by the square of the body height and is universally expressed in units of kg/m^2, resulting from mass in kilograms and height in meters.
- **Waist and neck circumference** waist circumference is measured at the midpoint between the lower rib and iliac crest. Neck circumference is measured at the level of the cricoid cartilage.
- **ENT examination: Nasal Examination:** External and internal examination of the nose. Further inspection proceeds with the speculum examination and rigid nasal endoscope.

Oral cavity and oropharynx:

Examination of the oral cavity begins with the inspection of the relative position of the tongue and palate. The oropharynx is described next including the soft palate, tonsils, uvula, and pharyngeal walls. The soft palate can be described as low lying, thick, webbed (redundant posterior pillars), and anteriorly or posteriorly placed. The uvula is described as long (greater than 1 cm) wide, thick, or embedded in the soft palate.

Mallampati score:

The test comprises a visual assessment of the distance from the tongue base to the roof of the mouth. The score is assessed by asking the patient, in a sitting posture, to open his or her mouth and to protrude the tongue as much as possible. The anatomy of the oral cavity is visualized; specifically, the assessor notes whether the base of the uvula, faucial pillars (the arches in front of and behind the tonsils), and soft palate are visible.

Tonsils are described as surgically absent, or 1, 2, 3, or 4 +, dividing the airway into less than 25%, 25% to 50%, 50% to 75%, or greater than 75%, respectively. Patients underwent the Muller maneuver to quantify the percent of airway collapse that occurs. The patient is asked to close the mouth and the examiner holds the patient's nose closed. The patient is asked to suck in against a closed mouth and nose to generate negative pressure in the pharynx.

The percent collapse of the airway at the level of the soft palate is estimated with maximal inspiration. The nasopharyngoscopy proceeds to the level of the oropharynx examining the base of the tongue and tonsils. Expressed as a percent of collapse retropalatal, retrolingual, and collapse at the level of the tonsil.

Epworth sleepiness score (ESS):

ESS is a scale intended to measure daytime sleepiness that is measured by the use of a very short questionnaire. An overnight sleep study in-laboratory polysomnography was done.

Statistical analysis

The collected data were coded, processed and analyzed using the SPSS (Statistical Package for the Social Sciences) version 22 for Windows® (IBM SPSS Inc, Chicago, IL, USA). Data were tested for normal distribution using the Shapiro Wilk test. Quantitative data were expressed as mean ± SD (Standard deviation), median and interquartile range (IQR). P < 0.05 was considered significant.

RESULTS

Demographic data: This study included 95 patients with OSAS with the following demographic criteria. The age range was (20-60) with a mean age of (43.99 ± 10.96). The male-to-female ratio was 1.7/1.3. Nearly half of the patients (47.4%) patients were smokers.

The patients' complaint and its duration:

Snoring was found in 96.3% of patients either alone or combined with OSA. With just 3.1% patients presented with isolated OSA. The mean period of the complaint was 5.07 ± 4.59 year.

Associated OSA comorbidity are included in table 1.

Table (1): Associated comorbidities

	All patients (n= 95)
Hypertension	(n=23) 24.2%
Diabetes mellitus	(n=14) 14.7%
GERD	(n=5) 5.3%

Patients' examination findings:

Patients' body mass index (BMI), and waist and neck circumference are illustrated in table 2.

Table (2): BMI, waist, and NC

	All patients (n= 95)		
	Mean ± SD	**Median**	**IQR**
Body mass index, kg/m^2	35.27 ± 7.19	34	31, 38
Waist circumference,cm	111.33 ± 13.15	110	105, 116
Neck circumference, cm	40.86 ± 3.09	41	39, 43

Examination of the studied subjects:

Oral cavity examination:

Mallampati score of grade II and III represented a majority of the patients (83.1%). More than half of patients had a bulky tongue. Tonsillar hypertrophy grade II and III could be documented in 2/3 of patients (Table 3).

Table (3): Oral cavity examination

All patients (n= 95)		
Mallampati score	**I**	(n= 3) 3.2%
	II	(n= 48) 50.5%
	III	(n= 31) 32.6%
	IV	(n= 13) 13.7%
Bulky Tongue		(n= 53) 55.8%
Uvula	**Normal**	(n= 56) 58.9%
	Bulky	(n= 13) 13.7%
	Elongated	(n= 26) 27.4%
Tonsil grading system	**Tonsillectomy**	(n= 4) 4.2%
	Grade I	(n= 15) 15.8%
	Grade II	(n= 56) 58.9%
	Grade III	(n=16) 16.8%
	Grade IV	(n= 4) 4.2%

Nasal examination: Forty-eight patients had unilateral or bilateral turbinate hypertrophy. The nasal septal deviation was noticed in 85 patients (Table 4).

Table (4): Nasal cavity examination of the studied subjects

All patients (n= 95)		
Turbinate	**Normal**	(n= 47) 49.5%
	Right hypertrophy	(n= 2) 2.1%
	Left hypertrophy	(n= 5) 5.3%
	Bilateral hypertrophy	(n= 41) 43.2%
Nasal septum	**Deviated**	(n= 52) 54.7%
	Spur	(n= 33) 34.7%
	Normal	(n= 10) 10.5%

Muller maneuver:

All patients had a range of retropalatal collapse from (20% to 90%), with a mean of 56.29± 23.53. The range of oropharyngeal collapse (tonsil collapse) in all patients is depicted in figure (1) and it was ranging from (no collapse-100%), with a mean of (62.31±25.50). All patients had varying degrees of retrolingual collapse (no collapse-80%), with a mean of 41.55±23.07.

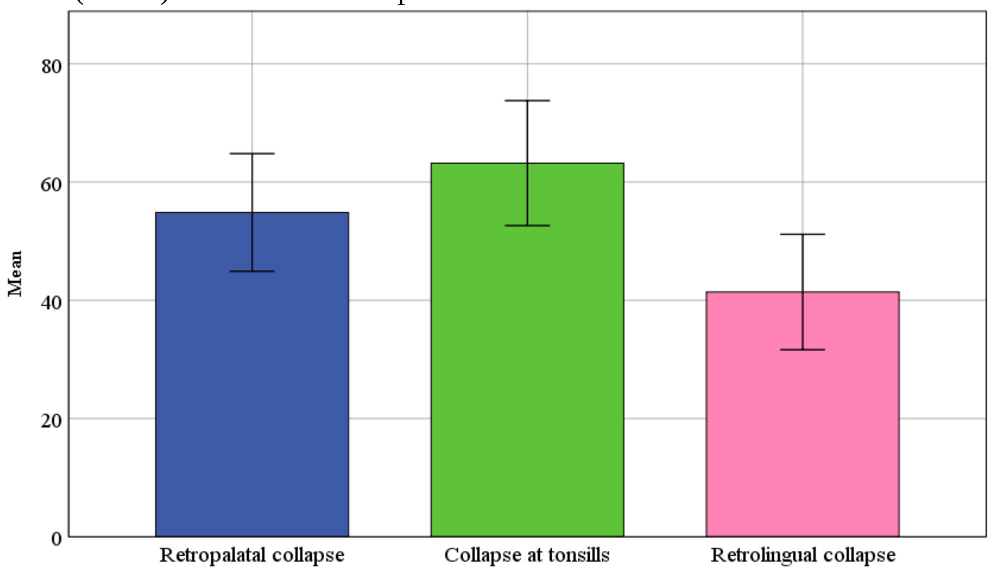

Fig. (1): Retropalatal collapse, collapse at tonsils, and retrolingual collapse

Epworth sleep scale (ESS): Twenty-nine patients had a score >10. The range of the score in all patients was (2-21). The mean value of the score in all patients was 8.09 ± 4.26 (Tables 5).

Table (5): Epworth sleep scale (ESS) value

All patients (n= 95)			
Epworth sleep scale value	No. of patients		
(0-8)	N=58		
(9-16)	N=35		
(17-24)	N=2		
Epworth sleep scale	**Mean ± SD**	**Median**	**IQR**
	8.09 ± 4.26	8	5, 12

Polysomnography data:

Polysomnography data included sleep efficiency calculation, recording duration, average O_2, and lowest O_2, total sleep time (TST), and AHI. Data of all patients are illustrated in table 6. Thirty patients had no OSA, only one had central sleep apnea, 64 patients had mild, moderate, or severe OSA.

Table (6): Polysomnography data

All patients (n= 95)			
	Mean ± SD	**Median**	**IQR**
Sleep efficiency ,%	74.30 ± 12.57	76.30	67, 83
Average O_2, %	82.73 ± 19.61	96	93, 97
Lowest O_2 ,%	70.35 ± 15.64	79	60, 89
Recording duration, hour.	7.81 ± 0.64	8	7.90, 8
TST, hour.	5.90 ± 1.08	6	5.30, 6 .60
AHI, hour. night sleep	24.08 ± 5.33	11.60	2.90, 3 6.40

TST: Total sleep time, AHI: Apnea hypopnea index

A statistically significant linear correlation was observed between age and BMI, the circumference of the waist and neck, duration of complaints, and collapse of the tonsils (Table 7).

Table (7): Correlation in the analysis between age and quantitative variables

	Correlation Coefficient	**95% CI**	**P**
BMI	**0.404**	**0.220, 0.560**	**< 0.001**
WC	**0.382**	**0.196, 0.542**	**< 0.001**
NC	**0.208**	**0.006, 0.393**	**0.043**
Duration of complaints	**0.221**	**0.021, 0.405**	**0.031**
Retropalatal collapse	0.111	- 0.273, 0.465	0.574
Collapse at tonsils	**0.483**	**0.118, 0.733**	**0.012**
Retro lingual collapse	-0.028	- 0.390, 0.342	0.886

DISCUSSION

Our study included 95 patients with OSA. The age range was (20-60) with a mean age of (43.99 ± 10.96). The male to female ratio was 1.7-1.3. This reflects the findings present in the literature that report OSAS as mostly affecting men. In studying the sex-specific prevalence of OSA by **Huang *et al.*** [(9)] out of 143326 females 9111 (6.4%) reported a diagnosis of OSA, whereas 3156 (13.8%) of 22896 males reported the diagnosis. The increased susceptibility to OSA in males compared to females may be due to anatomical variations in the pharyngeal and upper airway structures. Female sex hormones (e.g., estrogen and progesterone) may have beneficial effects on upper airway collapsibility, resulting in sex variations in the pathophysiology of OSA [(10)].

Nearly half of the patients (47.4%) were smokers. This comes in agreement with the literature. The results of an overnight polysomnography (PSG) PSG and health interviews with 811 individuals were presented by the University of Wisconsin Sleep Cohort Study, one of the largest cohort studies to research OSA in the United States. When compared to nonsmokers, current smokers had a higher risk of moderate or severe OSA (OR, 4.4; 95% CI, 1.5-13) [(11)].

The study population showed increased prevalence with age. This is in line with the findings of **Fietze *et al.*** [(12)], who conducted a large-scale PSG research in a general German population with a wide age range (20–81 years) and found that OSA prevalence estimates grew steadily with age. In adults, the occurrence of OSA rises with age [(4)]. This increase in prevalence with age may be due to parapharyngeal fat deposition, soft palate lengthening, and changes in other anatomic parapharyngeal components [(13)].

On studying the medical history and associated comorbidities of OSA patients, we found that hypertension (HTN) is the most significant associated comorbidity (24.2%). This is similar to **Natsios *et al.*** [(14)] findings, which showed that 37.6% of patients had HTN, and these findings support the current understanding that hypertension and OSA shared risk factors such as age and BMI.

Following HTN, diabetes mellitus (DM) was the second comorbidity associated with OSA (14.7%). There was a statistically significant linear correlation between age and DM and these findings are consistent with the findings of other researchers [(13)].

In our study, the mean of ESS was (8.09 ± 4.26). There was no correlation between it and age. These findings are consistent with prior research that has demonstrated that elderly persons have less sleepiness. Elderly adults had lower mean ESS scores than middle-aged adults, according to **Gottlieb *et al.***[(15)] (7.5 vs. 7.9).

In the examination of the study population, the mean BMI was (35.27 ± 7.19). The mean value of BMI steadily increased with age. There was a statistically significant linear regression between age and BMI. On studying the correlation between AHI and BMI, there was no statistically significant correlation. This comes in agreement with literature as the presence of increased body weight in more than 60% of patients referred for a diagnostic sleep evaluation is a common clinical observation [16]. Increases in body weight can change normal upper airway mechanics during sleep through different mechanisms including increased parapharyngeal fat deposition resulting in a reduced upper airway, changes in brain compensatory mechanisms that preserve airway patency, respiratory control system instability, and a reduction in functional residual capacity, resulting in a decrease in the upper airway stabilizing caudal traction [17].

There was a statistically significant linear correlation, which was observed between age and the circumference of the waist and neck, also there was a statistically significant linear regression between age and the circumference of the waist and neck. These findings are similar to **Soylu *et al.*** [18] results when he looked back on 535 patients who had been diagnosed with OSAS and had full-night polysomnography, he found that they all had OSAS. The OSAS group's average BMI, WC, and NC were statistically higher than the control group ($p < 0.001$).

As regards the correlation between AHI and the circumference of the waist and neck there was a statistically significant linear correlation between AHI and NC. In other studies from the literature, NC was found statistically significant much more related to the development of OSAS than BMI and WC [19].

There was no significant linear correlation between age and polysomnographic parameters (AHI, average O_2, and lowest O_2) this is similar to findings in previous studies [20, 21]. Our results oppose the findings made by **Peppard *et al.*** [3], AHI increases with age, although this increase was strongly related to weight gain.

CONCLUSION

OSA prevalence estimates have risen steadily with age. This increase in prevalence with age may be due to parapharyngeal fat deposition, soft palate lengthening, and alterations in other anatomic parapharyngeal structures. Obesity may have a significant role in defining the severity of OSAS in old patients, and BMI may be a significant determinant in determining the severity of OSAS in elderly patients.

REFERENCES

1. **Lam J, Mak J, Ip M (2012):** Obesity, obstructive sleep apnoea and metabolic syndrome. Respirology, 17(2): 223-236.
2. **Mokhlesi B, Finn L, Hagen E *et al.* (2014):** Obstructive sleep apnea during REM sleep and hypertension. Results of the Wisconsin Sleep Cohort. American journal of Respiratory and Critical Care Medicine, 190: 1158-1167.
3. **Peppard P, Young T, Barnet J *et al.* (2013):** Increased prevalence of sleep-disordered breathing in adults. American Journal of Epidemiology, 177(9): 1006-1014.
4. **Kapur V (2010):** Obstructive sleep apnea: diagnosis, epidemiology, and economics. Respiratory Care, 55(9): 1155-1167.
5. **Iber C, Chesson A, Quan S *et al.* (2007):** The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications. https://www.researchgate.net/ publication/ 285881487_The_AASM_Manual_for_the_Scoring_of_Sleep_and_Associated_Events_Rules_Terminology_and_Technical_Specifications
6. **Shamsuzzaman A, Somers V (2003):** Obstructive sleep apnea: implications for cardiac and vascular disease. JAMA., 290(14): 1906-1914.
7. **Patil S, Schneider H, Schwartz A *et al.* (2007):** Adult obstructive sleep apnea: pathophysiology and diagnosis. Chest, 132(1): 325-337.
8. **Epstein L, Kristo D, Strollo P *et al.* (2009):** Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med., 5(3): 263-276.
9. **Huang T, Lin B, Redline S *et al.* (2018):** Type of menopause, age at menopause, and risk of developing obstructive sleep apnea in postmenopausal women. American Journal of Epidemiology, 187(7): 1370-1379.
10. **Pillar G, Malhotra A, Fogel R *et al.* (2000):** Airway mechanics and ventilation in response to resistive loading during sleep: influence of gender. American Journal of Respiratory and Critical Care Medicine, 162(5): 1627-1632.
11. **Wetter D, Young T, Bidwell T *et al.* (1994):** Smoking as a risk factor for sleep-disordered breathing. Archives of Internal Medicine, 154(19): 2219-2224.
12. **Fietze I, Laharnar N, Obst A *et al.* (2019):** Prevalence and association analysis of obstructive sleep apnea with gender and age differences–Results of SHIP-Trend. Journal of Sleep Research, 28(5): 12770-75.
13. **Garvey J, Pengo M, Drakatos P *et al.* (2015):** Epidemiological aspects of obstructive sleep apnea. Journal of Thoracic Disease, 7(5): 920-25.
14. **Natsios G, Pastaka C, Vavougios G *et al.* (2016):** Age, body mass index, and daytime and nocturnal hypoxia as predictors of hypertension in patients with obstructive sleep apnea. The Journal of Clinical Hypertension, 18(2): 146-152.
15. **Gottlieb D, Whitney C, Bonekat W *et al.* (1999):** Relation of sleepiness to respiratory disturbance index: the Sleep Heart Health Study. American Journal of Respiratory and Critical Care Medicine, 159(2): 502-507.
16. **Strohl K, Redline S (1996):** Recognition of obstructive sleep apnea. American Journal of Respiratory and Critical Care Medicine, 154(2): 279-289.
17. **Fogel R, Malhotra A, White D (2004):** Sleep· 2: Pathophysiology of obstructive sleep apnoea/hypopnoea syndrome. Thorax, 59(2): 159-163.
18. **Soylu A, Levent E, Sarıman N *et al.* (2012):** Obstructive sleep apnea syndrome and anthropometric obesity indexes. Sleep and Breathing, 16(4): 1151-1158.
19. **Dancey D, Hanly P, Soong C *et al.* (2003):** Gender differences in sleep apnea: the role of neck circumference. Chest, 123(5): 1544-1550.
20. **Chung S, Yoon I, Lee C *et al.* (2009):** Effects of age on the clinical features of men with obstructive sleep apnea syndrome. Respiration, 78(1): 23-29.
21. **Vicini C, De Vito A, Iannella G *et al.* (2018):** The aging effect on upper airways collapse of patients with obstructive sleep apnea syndrome. European Archives of Oto-Rhino-Laryngology, 275(12): 2983-2990.