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Prevalence of obstructive sleep apnea among patients with chronic obstructive pulmonary disease

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Abstract

Background: The conjunction of chronic obstructive pulmonary disease (COPD) and obstructive sleep apnea (OSA) is known as overlap syndrome (OS). The coexistence of these diseases has cardiovascular morbidity and mortality. The aim of this study is to assess the prevalence of OSA in COPD patients. One hundred COPD patients (obese and non-obese) performed sleep questionnaires and polysomnograms.

Results: OSA prevalence in COPD was 50% and it increases with increasing disease severity ($P < 0.001$). The highest prevalence of OSA was found in obese patients with severe COPD; 90.5% of these patients have OSA. In the OSA group, obese patients were found to have significantly higher STOP-Bang Questionnaire (SBQ), Epworth Sleep Scale (ESS), modified medical research council (mMRC) dyspnea scale, apnea-hypopnea index (AHI), respiratory disturbance index (RDI), and oxygen desaturation index (ODI). Both obese and non-obese COPD patients showed significant positive correlations between AHI and smoking index (SI), SBQ, ESS, mMRC, ODI, and neck circumference (NC).

Conclusions: From this study, it can be concluded that moderate and severe COPD patients had a higher diagnosis of sleep-disordered breathing. Also, obese-COPD patients are more susceptible to develop OSA.

Trial registration: Name of the registry: Benha University Protocol Record Benha U123, Obstructive Sleep Apnea Prevalence in Patients With Chronic Obstructive Pulmonary Diseases. Trial registration number:NCT04903639. Date of registry: 5/22/2021 (retrospective study).

Keywords: COPD, OSA, OS, AHI

Background

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable illness that is characterized by persistent respiratory symptoms and airflow limitation due to airway and additionally alveolar abnormalities ordinarily brought about by critical exposure to harmful particles or gasses [1].

Obstructive sleep apnea (OSA) is a sleep disorder that includes discontinuance or a significant reduction in airflow in the presence of breathing effort bringing about nighttime hypoxemia and arousals from sleep [2]. The coexistence of COPD and OSA, known as overlap syndrome (OS), was first described by David Flenley 30 years ago, and he pointed out that a sleep study should be considered in obese COPD patients, in the individuals who snore, or those who complain of headache following nocturnal oxygen therapy to detect the presence of associated OSA [3]. The Global Burden Disease Study predicted that COPD which was known as the sixth driving reason of death in 1990 will turn out to be the third universal driving reason of death in 2020. A more up-to-date prediction assessed that COPD will be the fourth driving mortality reason by 2030 [4] Patients with COPD

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and OSA have a serious possibility of morbidity and mortality in comparison with those with either disease alone. Overlap syndrome has a prevalence of 14% among patients with mild COPD and 11% among OSA patients [5]. Obviously, sleep has harmful effects on breathing, respiratory drive, and functions of the lung. While these impacts can be ignored in healthy subjects, they can lead to catastrophic consequences especially in at-risk patients with the overlap syndrome. These patients may undergo marked hypoxemia and hypercapnia, diminshed quality of life, and elevated possibilty of death [6]. In this manner, assessing the presence of OSA in patients with advanced COPD appears to be intelligent as the simultaneousness of these diseases may conceivably clarify the high cardiovascular morbidity and mortality in those patients [7].

The aim of this study was to assess the prevalence of OSA in COPD patients.

Methods

Three hundred patients were enrolled in this prospective observational study who attended at chest department, outpatient clinic, Benha University Hospitals for follow-up between August 2018 and October 2020. According to medical history, clinical examination, and pulmonary function tests, only stable COPD patients (100 patients) were included. The research ethics committee at the faculty of medicine has approved the study and all patients provided informed consent before participation.

COPD patients were divided according to their body mass index (BMI) into two groups [8]: group A, 50 obese COPD patients (BMI $\geq 30\,kg/m^2$); and group B, 50 non-obese COPD patients (BMI $\leq 29.9\,kg/m^2$).

Inclusion criteria

COPD patients were diagnosed according to the Global Initiative for Chronic Obstructive Pulmonary Disease on the basis of GOLD, 2017. Included patients who had a chronic cough, sputum production, dyspnea, and/or a history of exposure to risk factors for the disease and confirmed by the presence of a postbronchodilator forced expiratory volume in the 1st second/forced vital capacity (FEV1/FVC) < 70% [9].

Exclusion criteria

Exclusion criteria included patients with COPD exacerbation; very severe COPD patients (postbronchodilator FEV1 < 30% anticipated or < 50% anticipated + chronic respiratory failure); patients with decompensated heart failure; thyroid dysfunction; ears, nose, and throat (ENT) causes of OSA; or patients with impaired hepatic and renal function.

COPD patients were characterized by their postbronchodilator FEV1 into mild (FEV1 ≥ 80% anticipated), moderate (50% ≤ FEV1 < 80% anticipated), and severe (30% ≤ FEV1 < 50% anticipated).

All patients included in this study were subjected to full history taking, physical examination (general including BMI and neck circumference (NC), local chest examination, oral, and ENT), questionnaires [The Epworth Sleepiness Scale (ESS) which translated to Arabic according to Anwar et al. [10], STOP-Bang Questionnaire (SBQ) [11], modified medical research council (mMRC) breathlessness scale which was translated ta Arabic according to Alyamani et al.] [12], spirometry which was done using JAEGER carefusion Germany 234 GmbH Lelbnizstr .7, 97,204 Hoechberg, Germany. Spirometry was done during stability of the disease. Echocardiography, liver, kidney, and thyroid function tests [triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH)] were done (overnight polysomnography (PSG) (SOMNO Screen Plus; SOMNO Medics GmbH, Randersacker, Germany)). The polysomnography consists of electroencephalogram (EEG), electrooculogram (EOG), electrocardiogram (ECG), electromyogram (EMG), pulse oximetry, thoracic and abdominal straps, body posture sensor, nasal thermistor, and nasal cannula to assess respiratory flow and pressure and bipolar channel limb movements (tibialis anterior). Electrodes and sensors were attached to patients by sleep physiologists. Data collection was obtained following signal perception by preprocessed computer (DOMINO Software, ver. 2.6.0; SOMNO Medics GmbH).

Patients considered having apnea if they had cessation of respiration for at least 10 s, hypopnea defined as an event that lasts for ≥10 s and is characterized by a decrease from baseline in the amplitude of breathing during sleep that either reaches > 30% with an oxygen desaturation of 4% or an arousal. The apnea-hypopnea index (AHI) is gotten from the absolute number of apneas and hypopneas separated by the total sleep time. Cutoff levels on AHI incorporate 5–15 episodes per hour for mild, 15–30 episodes per hour for moderate, and more than 30 episodes per hour for severe OSA [13].

Statistical analysis [14]

The gathered data were computerized and statistically analyzed utilizing IBM SPSS (Statistical Package for Social Science) statistic for Windows, version 24.0 Armonk, NY: IBM Corp. Qualitative data were addressed as frequencies and relative percentages. Chi-square test (χ^2) and Fisher's exact test were utilized to the figure differentiation between qualitative variables. Quantitative data were expressed as median and range for being non-parametric data (not normally distributed).

Mann-Whitney test was utilized to detect dissimilarity between quantitative variables in two groups for non-parametric variables. Spearman's correlation test was utilized for correlating non-parametric variables. Multiple linear regression analysis was performed to show the relationship of AHI with different variables.

Results

Smoking index, BMI, and neck circumference were significantly higher in the obese group. However, there was no statistically significant difference between both groups concerning age or sex distribution. Male gender was higher in both groups (Table 1). Obese and non-obese groups differed significantly regarding Pre FEV1, Pre FVC, and post FEV1% predicted as they were higher in non-obese groups. Also, both groups differed significantly as regard SBQ, ESS, mMRC questionnaires, AHI, respiratory disturbance index (RDI), and oxygen desaturation index (ODI) as they were statistically higher in obese patients (Table 2). OSA prevalence in COPD was 50%, and it increases with increasing disease severity (Table 3). The highest prevalence of OSA was found in obese patients with severe COPD (90.5%) (Table 4). In the OSA group, obese patients were found to have

Table 1 Comparison between the studied groups regarding clinico-demographic data

Parameter		Group		MW test	P
		Non-obese *N* = 50	**Obese *N* = 50**		
		Median (range)	**Median (range)**		
Age(years)		64 (42–75)	64 (46–72)	− 0.8	0.409
SI (pack/year)		40 (10–140)	65 (14–150)	− 2.2	0.02*
BMI (kg/m^2)		26.7 (20.4–28.5)	43 (31–64)	− 7.5	< 0.001**
NC (cm)		32 (30–36)	40 (38–46)	− 2.6	0.01*
		***N* (%)**	***N* (%)**	χ^2	***P***
Sex	Male	40 (80%)	44 (88%)	0.67	0.41
	Female	10 (20%)	6 (12%)		

SI smoking index, *BMI* body mass index, *NC* neck circumference, *MW* Mann-Whitney test, χ^2 chi-square test

*Significant

**Highly significant

Table 2 Comparison of PFTs sleep questionnaire and sleep parameter between both groups

Parameter	Groups		MW test	P
	Non-obese *N* = 50	**Obese *N* = 50**		
	Median (range)	**Median (range)**		
PRE FEV1 (%predicted)	60 (29–82)	52 (28–79)	−4.1	< 0.001**
PRE FVC (%predicted)	68 (40–80)	59 (32–73)	− 2.4	0.016*
Post FEV1 (%predicted)	68 (33–84)	59 (31–80)	− 3.9	0.00008**
SBQ	3 (2–5)	4 (3–7)	−2.8	0.005*
ESS	18 (11–24)	22 (11–24)	− 2.7	0.007*
mMRC	2 (2–3)	3 (2–4)	− 2.5	0.01*
Sleep efficiency	66.5 (33.7–80)	65.4 (17.5–79)	− 0.2	0.81
AHI	16.1 (3–65.2)	26.8 (4–106.8)	−2.1	0.04*
RDI	16.2 (5–65.2)	26.8 (7–106.8)	− 2.3	0.022*
ODI	15.8 (0.5–84.9)	44.4 (1.1–116.1)	− 2.9	0.003*

MW Mann-Whitney, *FEV1* forced expiratory volume in the 1st second, *FVC* forced vital capacity, *SBQ* STOP-Bang questionnaire, *ESS* Epworth Sleep Scale, *mMRC* modified medical research council, *AHI* apnea hypopnea index, *RDI* respiratory disturbance index, *ODI* oxygen desaturation index

*Significant

**Highly significant

Table 3 OSA prevalence in COPD

	COPD severity						Total ($N=100$)		χ^2	P
	Mild ($N=22$)		Moderate ($N=53$)		Severe ($N=25$)					
	N	%	*N*	%	*N*	%	*N*	%		
OSA	7	31.8%	22	41.5%	21	84%	50	50%	20.5	< 0.001**

COPD chronic obstructive pulmonary diseases, *OSA* obstructive sleep apnea

**Highly significant

Table 4 Prevalence of OSA among obese and non-obese COPD patients

	COPD severity															
	Non-obese						Total ($N=50$)	*P*	Obese						Total ($N=50$)	*P*
	Mild ($N=20$)		*Moderate ($N=26$)*		*Severe ($N=4$)*				*Mild ($N=2$)*		*Moderate ($N=27$)*		*Severe ($N=21$)*			
	N	%	N	%	N	%			N	%	N	%	N	%		
OSA	6	30%	6	23.1%	2	50%	28%	0.009*	1	50%	16	59.3%	19	90.5%	72%	0.009*

COPD chronic obstructive pulmonary diseases, *OSA* obstructive sleep apnea

*Significant

significantly higher SBQ, ESS, mMRC questionnaires, AHI, RDI & ODI. But, no difference was found in sleep efficiency between obese and non-obese OSA patients (Table 5). Both obese and non-obese COPD patients showed significant positive correlations between AHI and smoking index (SI), SBQ, ESS, mMRC, ODI, and NC, while AHI correlated negatively with FEV1. Only obese-COPD patients showed a significant positive correlation with BMI (Table 6). Average oxygen saturation correlated negatively with both NC and BMI in all COPD patients (Table 7). Multivariate logistic regression analysis disclosed that BMI, FEV1, SI, and ODI were independent predictors of OSA in COPD patients (Table 8).

Table 5 Comparison of Questionnaires data and polysomnographic parameter between both OSA groups

Parameter	Groups (OSA)		Test	P
	Non-obese	Obese		
	Median (range)	Median (range)		
SBQ	3 (2–5)	4 (3–7)	2.8	0.005*
ESS	18 (11–24)	22 (11–24)	2.7	0.007*
mMRC	2 (2–3)	3 (2–4)	2.5	0.01*
Sleep efficiency	69.4(27–79)	67.2(14.2–77)	− 0.63	0.52
AHI index	18.2(6–72)	30.9 (7.6–106.8)	2.01	0.04*
RDI	18.2 (6–72)	33.2 (10–106.8)	2.2	0.03*
ODI	15.8 (10–84.9)	48.5(11–116.1)	2.7	0.007*

SBQ STOP-Bang questionnaire, *ESS* Epworth Sleep Scale, *mMRC* modified medical research council, *AHI* apnea hypopnea index, *BMI* body mass index, *RDI* respiratory disturbance index, *ODI* oxygen desaturation index

*Significant

Discussion

Sleep disturbances might occur in up to 75% of individuals with COPD and may be due to nocturnal respiratory symptoms particularly cough, sleep-disordered breathing mainly OSA, or combinations of these processes. The simultaneousness of COPD and OSA is known as the overlap syndrome and, although its prevalence is not greater than would be anticipated from the prevalence of either disorder alone, the overlap syndrome is associated

Table 6 Correlations between AHI index and certain studied parameters within each group

Parameters	AHI index			
	Non-obese		Obese	
	r	*P*	*r*	*P*
Age	0.16	0.45	− 0.07	0.6
BMI	0.19	0.368	0.5	< 0.001**
FEV1	− 0.71	< 0.001**	− 0.73	< 0.001**
SI	0.66	< 0.001**	0.75	< 0.001**
SBQ	0.44	0.03*	0.51	< 0.001**
ESS	0.75	< 0.001**	0.73	< 0.001**
mMRC	0.5	0.011*	0.57	< 0.001**
ODI	0.69	< 0.001**	0.86	< 0.001**
NC	0.54	0.006*	0.34	0.003*

r correlation coefficient, *AHI* apnea hypopnea index, *BMI* body mass index, *FEV1* forced expiratory volume in the 1st second, *SI* smoking index, *SBQ* STOP-Bang Questionnaire, *ESS* Epworth Sleep Scale, *mMRC* modified medical research council, *ODI* oxygen desaturation index, *NC* neck circumference

*Significant

**Highly significant

Table 7 Correlation of average oxygen saturation with BMI and neck circumference in COPD patients

	Average oxygen saturation	
	R	***p* value**
NC	− 0.27	0.007*
BMI	− 0.23	0.02*

NC neck circumference, *BMI* body mass index

*Significant

with significant nocturnal hypoxemia and impaired sleep quality [15].

The estimated prevalence of sleep disturbance in COPD varies from 34 to 78% [16]. COPD and OSA are frequent in the general population and give out a huge danger of expanded morbidity and mortality when they exist together in a similar patient. Clinicians should cautiously assess the clinical results and the high danger of cardiovascular problems connected to the overlap [17]. In the current work, studied groups were matched regarding age and sex distribution (most of the participants were males) while BMI and NC were higher in obese COPD patients. Compared to women, men have a higher rate of being obese or overweight. Also, fat deposits in the upper respiratory tract and hormonal changes related to obesity (increase leptin and insulin) predispose to OSA [18].

Qinhan et al. demonstrated in a similar study on 116 COPD patients that OS was frequent in obese men characterized by higher BMI (38 kg/m^2), NC (38.8 cm), and SI (800 pack/year) [19]. In a comparative study, Soler and his colleagues found that most of the participants were elderly male, and subjects with OS had significant increases in BMI, NC, and greater smoking history with more prevalent OSA among overweight subjects (75%) [20]. Similarly, Gunduz et al. concluded that overweight COPD patients with BMI > 27 kg/m^2 had a higher possibility of developing OSA [21].

In the current study, the obese-COPD group had the worst spirometric parameter and higher sleep and dyspnea questionnaire score with statistically significant differences compared to non-obese COPD patients. The progressive impairment of pulmonary function noticed in COPD con increase the exertional dyspnea which in turn limits the activities of daily living. Also, the presence of both COPD and obesity contribute to the presence of dyspnea, fatigue, and tiredness [22]. Watson et al. went in line with this finding as they detected that the annual decline in FEV_1 in obese-COPD males was higher than in those with normal BMI. Furthermore, excessive weight gain had been linked to a decline in lung function, especially FVC. In general, obesity is connected with diminished FEV_1 and weight acquire has been shown to decrease pulmonary function longitudinally. Nevertheless, BMI as an adiposity marker cannot separate between body fat and muscle tissue which may influence lung function [23]. Also, non-obese COPD patients without OSA had higher FEV1/FVC and FEV1 compared to obese patients [19]. Similarly, Zhu et al. in their study reported that the FVC and FEV_1 in obese patients with OS were significantly diminished than in non-obese COPD patients ($P < 0.01$) [24]. On the other hand, Sleep quality and quality of life were impaired in patients with moderate to very severe COPD. Also, Pittsburgh Sleep Quality Index (PSQI), ESS, mMRC dyspnea scale, and short-form 36 health survey questionnaires (SF-36) were higher especially in obese patients with OSA [25]. In another comparable study, Berlin and modified Berlin scales were higher in OS than COPD patients [19].

Table 8 Multiple regression analysis of age BMI, post FEV1, smoking index, baseline O_2% and neck circumference as an independent variable for AHI

	Unstandardized coefficients		Standardized coefficients	t	Sig.	95.0% confidence interval for β	
	B	SE	*β*			Lower bound	Upper bound
BMI	− 0.44	0.19	− 0.15	− 2.38	**0.02**	− 0.81	− 0.07
FEV1	− 0.55	0.22	− 0.33	− 2.48	**0.02**	− 0.99	− 0.11
SI	0.2	0.09	0.26	2.13	**0.04**	0.013	0.38
ODI	− 2.34	0.47	− 0.36	− 4.99	**< 0.001**	− 3.28	− 1.41
NC	1.75	0.92	0.12	1.89	0.06	− 0.09	3.58
PHT	0.16	0.20	0.049	0.78	0.44	− 0.25	0.56
(Constant)	224.75						

β regression coefficient, *SE* standard error $p < 0.05$ is significant

Dependent variable: AHI index, *AHI* apnea hypopnea index, *BMI* body mass index, *FEV1* forced expiratory volume in the 1st second, *SI* smoking index, *ODI* oxygen desaturation index, *NC* neck circumference, *PHT* pulmonary hypertension

In this study, there was a statistically significant difference between the studied groups (obese and non-obese COPD, obese and non-obese OSA) regarding AHI, RDI & ODI as they were significantly higher in obese COPD and COPD-OSA patients. However, there was no statistically significant dissimilarity as regard sleep efficiency.

This can be explained by the presence of well-known factors that exaggerate oxygen desaturation in obese patients as low baseline oxygen saturation, low lung volume, and high oxygen expenditure which could be exaggerated by diurnal and nocturnal hypoxemia of COPD [26].

Turcani et al. went in line with this result as they found that patients with ODI over 15/h had an elevated weight, BMI, and NC [27]. In a study done by Marin et al., ODI, RDI, and AHI were higher in the overlap syndrome in comparison to COPD patients without OSA [28]. Also, Anisa et al. demonstrated that severe COPD patients had a chance of 4.39 times to experience OSA than mild to moderate COPD [29].

In this work, OSA increases with increasing COPD severity (either obese or non-obese) with a total prevalence of 50%. OSA prevalence in non-obese COPD was 28% and in obese patients was 72%. This could be explained by the presence of common risk factors that lead to OSA and COPD including high BMI and smoking also utilization of inhaled corticosteroids in more advanced COPD may contribute to OSA by causing upper airway myopathy or extrapulmonary inflammation, which may impair upper airway reflexes or neuromuscular responses [30]. Patil et al. found that from 30 COPD patients, 23 of them had OSA. Four patients (17.33%) were within ordinary BMI and 19 (82.6%) were overweight [31]. While Wan et al. found a prevalence of OSA of 52.8% in patients with COPD [32], others reported a prevalence of 63.9% and 65.9% in moderate to severe COPD [20, 33]. Gunduz et al. found that the frequency of OS in mild hypoxemic COPD patients without symptoms of sleep apnea reached 58% [21]. On the other hand, Zhu et al. reported that 67.9% of COPD patients enrolled in their study had OS with wide range of OSA prevalence in different degrees of COPD extending from 2.9 to 65.9% [24]. Similarly, Narasimhan and his colleagues found that the prevalence of OSA in COPD was 53% [34].

In the current study, there were significant positive correlations between AHI and SI, SBQ, ESS, mMRC dyspnea scale, ODI, and NC in both obese and non-obese COPD patients while AHI correlated negatively with FEV1 in both groups. COPD patients usually are smokers or ex-smokers. Smoking can lead to inflammation of the upper airway which in turn leads to swelling, narrowing, and probably closure of the airway. OSA was reported to be approximately three times more prevalent in ever-smokers than never-smokers [35]. Mengqing et al. found that an SBQ score of ≥ 3 is able to determine OSA in COPD patients and a score of ≥ 4 can recognize a high chance for developing severe OSA and they concluded that it correlates well with the degree of disease severity [36]. Similarly, in a study done by Narasimhan et al. on 66 COPD patients, they found a significant correlation between AHI and ESS, mMRC, and FEV1. So, poor lung function (reflected by FEV1) was connected to higher grade of OSA [34]. However, Jaoude and El-solh did not find a significant relation between the severity of airflow limitation and AHI [37]. Turcani et al. found a statistically significant relation between categorized AHI and weight, BMI, NC, and ESS. It is obvious that with increasing weight, BMI, NC and ESS, AHI, and OSA severity increased. And they concluded that as long as the AHI increased, the oxygen saturation decreased [27].

In this study, a significant negative correlation was found between average oxygen saturation and both NC and BMI in all COPD patients. This could be explained by the combined effect of obesity in reducing the functional residual capacity (FRC) and increasing the closing volume that leads to a greater tendency for small airway closure, causing ventilation-perfusion mismatching and pulmonary shunting, thus exacerbating oxygen desaturation [38]. Peppard et al. agreed with this as they found that BMI is the main factor to anticipate the intensity of oxygen desaturation during apnea or hypopnea [39].

In the current work, multivariate logistic regression analysis disclosed that BMI, Post FEV1, SI, and ODI were independent predictors of OSA in COPD patients with no effect of pulmonary hypertension on OSA in COPD patients. The presence of PH was mostly related to BMI, nocturnal hypoxia, and COPD itself but AHI was not an independent risk factor for PH. So, it is difficult to find out if PH is due to intermittent hypoxemia produced by sleep apnea or persistent hypoxemia related to chronic lung disease. Sreedharan et al. in a similar study found that ODI is an independent predictor of OSA and they found a positive correlation between OSA severity and nocturnal hypoxemia [40]. The median pulmonary artery pressure (PAP) was found to be similar in COPD patients irrespective to OSA presence. Also, AHI did not correlate with PAP in a similar study [41]. Izabella and his colleagues agreed with this finding as they found that BMI, NC, head circumference (HC), ESS, and smoking index were independent variables for OSA [42]. Another study demonstrated that common OSA predictors (e.g., gender, ESS) did not perform well in patients with more advanced COPD [43]. In the study done by Sharma et al. on 206 COPD patients, they demonstrated that BMI, male gender,

informed snoring index, and choking index were independent predictors of OSA in COPD patients. They suggested that the chance of developing OSA increases with increasing BMI [44]. It is well known that patients with COPD and OSA are more vulnerable to pulmonary hypertension, which is associated with a reduction in lung function and increased OSA severity. So, hypoxemia and OSA in elderly COPD patients should be recognized and managed properly [41].

Conclusion

From this study, it can be concluded that moderate and severe COPD patients had a higher diagnosis of sleep-disordered breathing. Also, obese-COPD patients are more susceptible to develop OSA.

Abbreviations

COPD: Chronic obstructive pulmonary disease; OSA: Obstructive sleep apnea; OS: Overlap syndrome; BMI: Body mass index; FEV1: Forced expiratory volume in the 1st second; FVC: Forced vital capacity; ENT: Ears, nose, and throat; NC: Neck circumference; ESS: Epworth Sleepiness Scale; SBQ: STOP-Bang Questionnair; mMRC: Modified medical research council; T3: Triiodothyronine; T4: Thyroxine; TSH: Thyroid-stimulating hormone; PSG: Polysomnography; EEG: Electroencephalogram; EOG: Electrooculogram; ECG: Electrocardiogram; EMG: Electromyogram; AHI: Apnea-hypopnea index; RDI: Respiratory disturbance index; ODI: Oxygen desaturation index; SI: Smoking index; PSQI: Pittsburgh Sleep Quality Index; SF-36: Short-form 36 health survey questionnaires; FRC: Functional residual capacity; PAP: Pulmonary artery pressure; 6MWT: Six-minute walk test; PaO2: Arterial oxygen tension; PaCO2: Carbon dioxide oxygen tension; HC: head circumference.

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Authors' contributions

AE made a substantial subscription to the conception and plan of the work, OM contributed to acquiring, evaluation, and explanation of evidence. SM contributed to the design of new software utilized in this work. ME outlined the work and rescripted it. All authors read and authorized the final manuscript.

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Availability of data and materials

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Declarations

Ethics approval and consent to participate

The research ethics committee at the Faculty of Medicine, Benha University (no. 63), has approved the study (12/6/2018) and all patients provided a written consent before participation.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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