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Research Article

Conventional Versus Other Methods to Evaluate Patients with Obstructive Sleep Apnea

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Abstract

Background: Obstructive Sleep Apnea (OSA) is a common condition associated with significant cardiovascular and metabolic complications. The apnea–hypopnea index (AHI) has long been the gold standard for assessing OSA severity. However, emerging evidence highlights the role of oxygen desaturation index (ODI)and time spent below90%oxygen saturation(T90) as important markers for hypoxia-related outcomes. **Aim:** This study aimed to evaluate the clinical relevance of the ODI and the percentage of time spent with T90, in comparison to AHI, for assessing OSA severity. **Methods:** Its retrospective study,100 adult patients diagnosed with OSA at the sleep unit of the Cardiothoracic Hospital, Minia University, Egypt. Data were collected including clinical evaluation, physical examination, arterial blood gases, chest imaging, and echocardiography. Severity grading was based on AHI, ODI. Epworth Sleepiness Scale (ESS)scores used to assess daytime sleepiness. Associations with cardiometabolic comorbidities were analyzed, and the diagnostic performance of ESS and STOP-BANG questionnaires was compared. **Results:** A significant correlation was found between AHI and ODI, with a kappa agreement of 0.66 ($p < 0.001$), demonstrating strong consistency between both indices in grading OSA severity. Both ESS and STOP-BANG scores demonstrated significant correlations with AHI and ODI ($p < 0.001$), with ESS exhibiting stronger associations. The AHI and ODI values were significantly higher in patients with IHD and DM, indicating a correlation between the severity of OSA and these cardiometabolic conditions. The ROC curve analysis for predicting pulmonary hypertension showed that the ODI had a slightly higher area under the curve (AUC = 0.63) than the AHI (AUC = 0.59), with ODI being statistically significant ($p = 0.03$). **Conclusion:** AHI and ODI are robust markers of OSA severity. ESS is a more practical screening tool than STOP-BANG, particularly in identifying severe OSA. The strong association between OSA severity and cardiometabolic comorbidities highlights the need for early identification using both objective and symptom-based tools to optimize risk stratification and management.

Key words: Obstructive Sleep Apnea; Polysomnography (PSG); Epworth Sleepiness Scale; Apnea-Hypopnea Index; Oxygen Desaturation Index

Introduction

Obstructive sleep apnea (OSA) is a prevalent and potentially serious sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia and sleep fragmentation due to repeated arousals [1]. These respiratory disturbances are strongly associated with increased cardiovascular morbidity and mortality, primarily mediated by the adverse effects of intermittent hypoxemia on vascular function, oxidative stress, and systemic inflammation.[2]

Polysomnography (PSG) remains the gold standard for diagnosing OSA and assessing its severity [3]. However, due to its high cost,

limited availability, and technical complexity, there is growing interest in identifying alternative, accessible methods to stratify patients at risk and prioritize those requiring definitive testing. In this context, validated screening tools such as the Epworth Sleepiness Scale (ESS), STOP-BANG, STOP, and Berlin Questionnaires have emerged as low-cost, practical instruments to detect patients at high risk for moderate to severe OSA.[4,5]

The apnea-hypopnea index (AHI), defined as the total number of apneas and hypopneas per hour of sleep, is the most widely used metric for quantifying OSA severity [6]. According to the American Academy of Sleep Medicine (AASM), OSA severity is categorized as mild (5–15 events/hour), moderate (15–30), or severe (>30) based on AHI values [7]. However, AHI has notable limitations. It captures only the frequency—not the depth or duration—of respiratory events and does not account for the associated desaturation burden or cumulative hypoxic exposure [8,9]. As such, relying solely on AHI may underestimate disease severity and the risk of OSA-related complications.

To address these limitations, alternative metrics such as the oxygen desaturation index (ODI) and time spent with oxygen saturation below 90% (T90) have been proposed as complementary or superior indicators of disease burden. The ODI reflects the average number of oxygen desaturation events (≥3% drop in SpO_2 lasting ≥10 seconds) per hour of sleep, thus providing a surrogate marker for hypoxia severity [10]. Similar to AHI, ODI can be stratified into mild (5–15 events/hour), moderate (15–30), and severe (≥30) categories. Notably, recent evidence suggests that ODI may have stronger predictive value for adverse cardiovascular outcomes compared to AHI.[8]

Another important yet underutilized hypoxia metric is T90, which quantifies the cumulative duration or proportion of total sleep time (TST) spent with oxygen saturation below 90%. T90 can be expressed in absolute (TST90 in minutes) or relative (%T90) terms. Emerging data indicate that T90 may outperform both AHI and ODI in predicting cardiovascular morbidity and all-cause mortality, as it reflects the overall hypoxic load more comprehensively .[11]

The aim of this study was to provide a comprehensive evaluation of the clinical relevance of the oxygen desaturation index (ODI) and time spent with oxygen saturation below 90% (T90) in comparison to the traditionally used apnea-hypopnea index (AHI).

Patients and Methods

This retrospective study was conducted at the Sleep Unit of the Cardiothoracic Hospital, Minia University, Egypt. A total of 100 adult patients diagnosed with obstructive sleep apnea (OSA) through overnight polysomnography (PSG) were included in the analysis.

Patients were eligible for inclusion if they had a confirmed diagnosis of OSA based on PSG findings. Exclusion criteria included the presence of chronic respiratory diseases other than OSA (e.g., COPD, interstitial lung disease), age under 18 years, total sleep time less than 4 hours during PSG recording, prior PSG conducted at an external facility, or incomplete clinical or polysomnographic data. These criteria were applied to ensure data consistency and internal validity.

Patient data were extracted from electronic and paper medical records and included demographic characteristics, anthropometric measures, and relevant clinical information. All patients underwent comprehensive baseline evaluations including medical history, physical examination, vital sign measurement, chest auscultation, and investigations such as arterial blood gas analysis, chest radiography, 12-lead electrocardiography (ECG), and transthoracic echocardiography.

Subjective daytime sleepiness was assessed using the validated Arabic version of the Epworth Sleepiness Scale (ESS). OSA severity was evaluated using PSG-derived indices, including the Apnea-Hypopnea Index (AHI) and Oxygen Desaturation Index (ODI). The ESS scores were stratified as follows: ≤8 (no daytime sleepiness), 9–14 (mild to moderate excessive daytime sleepiness [EDS]), and ≥15 (severe EDS). AHI and ODI severity were classified according to standard thresholds:

- AHI: mild (5–15 events/hour)، moderate (15–30)، and severe.(30<)
- ODI: mild (5–14 events/hour), moderate (15–29), and severe .(30≤)

Inclusion criteria :

- Obstructive Sleep Apnea (OSA) Patients.

Exclusion criteria:

- Any Chronic Chest Disease Other Than OSA.
- Patients younger than 18 years.
- Patients who underwent PSG testing at other Centers.
- Patients with incomplete data.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Review Board of Minia University. All data were handled in compliance with institutional confidentiality standards and ethical research guidelines.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro-Wilk test. Normally distributed variables were expressed as means ± standard deviations (SD), and group comparisons were conducted using analysis of variance (ANOVA) followed by Tukey's post hoc test. Categorical variables were compared using the Chi-square (χ^2) test. All statistical tests were two-tailed, and a p-value < 0.05 was considered statistically significant.

Results

The study analyzed 100 patients with obstructive sleep apnea (OSA), categorized into mild (N=12), moderate (N=10), and severe (N=78) groups based on the Apnea-Hypopnea Index (AHI). Statistically significant differences were observed across several parameters. Severe OSA patients had significantly higher BMI (42.9 ± 5.8, p=0.001), neck circumference (44.9 ± 4.7, p=0.001), and waist circumference (123.1 ± 21.5, p=0.001) compared to milder cases. Comorbidities such as hypertension (HTN, 83% in severe OSA, p=0.002), diabetes mellitus (DM, 87%, p=0.001), and ischemic heart disease (IHD, 91%, p=0.02) were more prevalent in the severe group. Polysomnographic data revealed significant differences in REM AHI (50.6 ± 34.4, p=0.003), RDI (58.7 ± 27.7, p=0.0001), and minimal SPO2 (69.6 ± 11.9, p=0.0001), also number of desaturation less and SPO2 time less than 90 was significantly higher in severe cases indicating worse respiratory and oxygenation metrics in severe OSA. Additionally, the STOP BANG score (7.1 ± 0.8, p=0.0001) and Epworth Sleepiness Scale (ESS, 18.9 ± 2.2, p=0.0001) were significantly higher in severe cases, reflecting greater symptom severity (Table 1).

The overall Kappa agreement coefficient was 0.66, indicating moderate to strong agreement between both indices ($p < 0.001$). Notably, 94.9% of cases classified as severe by AHI were also identified as severe by ODI (Table 2).

Body Mass Index (BMI) showed a moderate positive correlation with AHI ($r = 0.39$, $p < 0.001$) and ODI ($r = 0.36$, $p < 0.001$), waist circumference and neck circumference were also positively correlated with both AHI and ODI (AHI $r = 0.47$, $p < 0.001$; ODI $r = 0.40$, $p < 0.001$). The STOP-BANG score has strong positive correlation with AHI ($r = 0.60$, $p < 0.001$) and a slightly weaker but still significant correlation with ODI ($r = 0.52$, $p < 0.001$). Similarly, the Epworth Sleepiness Scale (ESS) correlated strongly with both AHI ($r = 0.60$, $p < 0.001$) and ODI ($r = 0.59$, $p < 0.001$). However, age did not show a statistically significant correlation with either AHI ($r = 0.09$, $p = 0.33$) or ODI ($r = 0.17$, $p = 0.08$) (Table 3).

Gender was not significantly associated with AHI, ODI, or REM AHI. However, females had significantly lower minimal SpO_2 levels compared to males ($p = 0.04$). Smoking status was significantly associated with higher ODI values ($p = 0.02$), particularly among ex-smokers. AHI and other oxygen parameters did not show statistically significant differences across smoking categories. Hypertension was significantly associated with higher AHI ($p = 0.02$), ODI ($p = 0.001$), and lower minimal SpO_2 ($p = 0.04$). Hypertensive patients also exhibited more frequent desaturations ($p = 0.05$). Diabetes mellitus was significantly associated with increased AHI ($p = 0.003$), ODI ($p < 0.001$), and REM AHI ($p = 0.03$), along with lower minimal SpO_2 ($p = 0.005$), a greater number of desaturations ($p = 0.009$), and increased SpO_2 time <90% ($p = 0.006$). Ischemic heart disease (IHD) was associated with significantly elevated AHI ($p = 0.001$), ODI ($p = 0.002$), and REM AHI ($p = 0.002$), as well as lower minimal SpO_2 ($p < 0.001$) and higher desaturation burden ($p = 0.01$). SpO_2

time <90% was also significantly prolonged in IHD patients (p = 0.005). Pulmonary hypertension showed a significant association with minimal SpO_2 (p = 0.01) and SpO_2 time <90% (p = 0.02), with more severe cases demonstrating greater oxygen desaturation, although differences in AHI and ODI were not statistically significant (Table 4) .

ESS consistently outperformed STOP-BANG across all severity levels. For AHI ≥5, ESS showed excellent accuracy (AUC = 0.95, p = 0.02) with 92% sensitivity and 100% specificity at a cutoff >12. For AHI ≥15 and ≥30, ESS maintained high accuracy (AUCs = 0.98 and 0.92, respectively), with sensitivity 93–95% and specificity 81–100%.

STOP-BANG, with a consistent cutoff >6, showed good performance but lower accuracy compared to ESS. AUCs ranged from 0.78 to 0.89, with reduced specificity at higher AHI thresholds. ESS also outperformed STOP-BANG for ODI ≥5, ≥15, and ≥30, with AUCs between 0.92 and 0.94 (p < 0.001), offering high sensitivity (86–95%) and specificity (81–100%). STOP-BANG showed moderate accuracy (AUCs = 0.78–0.86) and was less discriminative at lower ODI levels (Table 5). Both AHI and ODI exhibited excellent predictive power for severe EDS, with AUCs of 0.94 and 0.92, respectively (p < 0.001 for both). AHI >30.05 provided 95% sensitivity and 85.7% specificity, while ODI >38.8 yielded 91% sensitivity and 86% specificity. Both demonstrated high PPVs (96.2% and 96%) and overall diagnostic accuracy (93% and 90%, respectively). (Table 6).

Table 1: characteristic data of studied cases

	Total cases (N=100)	Mild OSA (N=12)	Moderate OSA (N=10)	Severe OSA (N=78)	P value
Demographics					
Age (years) *	61.7 ±9.9	54.7±11.1	58.2±11.3	63.2±9.2	0.11
Gender **					
Males	54 (54%)	7 (58.3%)	6 (60%)	41 (52.6%)	0.86
Females	46 (46%)	5 (41.7%)	4 (40%)	37 (47.4%)	
BMI *	41.4±6.5	35.2±7.4	37.9±6.01	42.9±5.8	0.001
Neck circumference *	43.6±5.4	37.1±4.4	41.1±5.5	44.9±4.7	0.001
Waist circumference *	118.9±22.8	98.2 ±19.1	111.4±22.7	123.1±21.5	0.001
Symptoms					
STOP BANG score	6.84±1.11	5.3±1.4	6.4±0.9	7.1±0.8	0.0001
ESS	17.7±3.2	11.7±1.7	15.6±3.5	18.9±2	0.0001
Comorbidities					
HTN **	84 (84%)	6 (7%)	8 (10%)	70 (83%)	0.002
DM **	69 (69%)	3 (4%)	6 (9%)	60 (87%)	0.001
IHD	43 (43%)	3 (7%)	1 (2%)	39 (91%)	0.02
Hypothyroid disease **	21 (21%)	4 (19%)	3 (14%)	14 (67%)	0.36
Macroglossia	48 (48%)	2 (4%)	4 (8%)	42 (88%)	0.04
Pulmonary hypertension **					
Total	34 (34%)	3 (25%)	1 (10%)	30 (38.5%)	0.58
Mild	16 (16%)	2 (16.7%)	1 (10%)	13 (16.7%)	
Moderate	15 (15%)	1 (8.3%)	0 (0%)	14 (17.9%)	
Severe	3 (3%)	0 (0%)	0 (0%)	3 (13.8%)	
Cardiac disease onset (n=46) **					
Before OSA	13 (13%)	1 (8.3)	0 (0%)	12(15.4%)	0.11
After OSA	33 (33%)	3 (25%)	1 (10%)	29 (37.2%)	
Polysomnographic data					
Sleep Efficiency in % *	61.9±27.1	80.9±25.5	79.7±9.3	56.7±26.8	0.001
Sleep Latency in minutes *	15.5±20.7	10.9±9.8	15.3±17.8	16.2±22.3	0.71
Arousal Index *	21.8±15.8	9.8±8.3	14.9±6.4	24.6±16.4	0.20
REM AHI *	42.9±34.1	11.4±8.1	20.4±15.1	50.6±34.4	0.003

RDI *	48.9±30.9	7.4±3.4	22.2±4.9	58.7±27.7	0.0001
Minimal SPO2 *	72.5±12.4	84.5±9.3	79.8±7.5	69.6±11.9	0.0001
Baseline SPO2 *	89.6±6.2	93.4±3.2	92.7±3.05	88.7±6.5	0.013
Average SPO2 *	88.2±5.6	93.2±3.4	92.5±3.4	86.9±6.7	0.001
No of destructions < 90% *	124±114.9	14.5±19.3	78.3±101.1	146.6±114.4	0.0001
SPO2 Time < 90 % *	44.7±37.8	10.4±22.7	20.5±30.9	53.1±36.4	0.0001
Maximum HR *	110.4±21	109.7±23.7	103.2±19.4	111.5±20.8	0.49
Minimum HR *	62.5±15.3	62.1±12.7	55.3±12.3	63.4±15.8	0.28
Average HR *	80.9±15.2	74.2±13.7	72.8±6.2	83.05±15.6	0.034

* Data presented by mean ± SD ** data presented by number (%)

Table 2: association between ODI and AHI among all studied cases.

				ODI 56.6±29.3	**Kappa Agreement**	**P value**
		Mild OSA 8±3.7	**Moderate OSA 20.5 ± 5.4**	**Severe OSA 67.6±22.1**		
AHI 48.7±30.6	**Mild OSA 7.34 ± 3.3**	8 88.9%	4 33.3%	0 0.0%	0.66	<0.001*
	Moderate OSA 22.1 ± 4.7	1 11.1%	5 41.7%	4 5.1%		
	Severe OSA 58.5 ± 27.4	0 0.0%	3 25.0%	75 94.9%		

Table 3: correlation between AHI and ODI with other variables among all cases

	AHI		**ODI**	
	r	**P value**	**r**	**P value**
Age	0.09	0.33	0.17	0.08
BMI	0.39	<0.001*	0.36	<0.001*
Waist circumference	0.36	<0.001*	0.30	0.002*
Neck circumference	0.47	<0.001*	0.40	<0.001*
Stop Bang score	0.60	<0.001*	0.52	<0.001*
Epworth score	0.60	<0.001*	0.59	<0.001*

* significant

Table 4: association of both AHI, ODI with other variables among all studied cases

	AHI	**ODI**	**REM AHI**	**minimal SPO2**	**Number of desaturations <90**	**SPO2 time 90**
Gender **						
Males	51.9±32.7	60.6 ±32	40.3±33.5	74.7±11.6	123.1±121.5	40.6±37.8
Females	44.9±27.9	51.8±25.3	45.8±39.4	69.7±13	125±101	49.5±37.7
P value	0.25	0.13	0.42	0.04*	0.93	0.24
Smoking **						
Non-smoker	43.4±27.8	50.1±26.1	43.8±35.2	71.02±13.3	112.8±98.8	46.9±39.2
Smoker	52.5±32.6	61.3±32.3	39.06±30.7	74.4±12.1	117.6±103.3	40.1±37.8
Ex-smoker	63.8±34.5	74.5±26.1	51.5±41.1	73.1±8	207±115	49.3±32.1
P value	0.10	0.02*	0.57	0.44	0.05*	0.65
HTN **						
No (n=16)	32.7±28.1	35.6±30.4	35.3±31.2	78.1±11.9	75.1±107	34.6±38.9
Yes (n=84)	51.8±30.3	60.6±27.5	44.3±34.6	71.4±12.3	133.3±114.6	46.6±37.6

P value	0.02*	0.001*	0.33	0.04*	0.05*	0.24
DM **						
No (n=69)	35.4±28.3	40.2±28.3	32.3±25.9	77.6±12.4	79.4±75.4	29.5±25.05
Yes (n=31)	54.7±30	63.9±29.8	47.6±36.4	70.1±11.8	144.01±117.9	51.6±37.3
P value	0.003*	<0.001*	0.03*	0.005*	0.009*	0.006*
IHD **						
No (n=57)	40±26.4	48.9±28.4	34.01±28.2	76.5±10.5	98.4±98.1	35.5±31.2
Yes (n=43)	60.2±32.4	66.8±27.7	54.6±37.8	67.02±12.8	157.8±126.9	56.9±36.9
P value	0.001*	0.002*	0.002*	<0.001*	0.01*	0.005*
Pulmonary hypertension **						
No (n=66)	45.5±29.9	52.4±28.4	41.1±34.5	74.2±12.2	117.8±111.4	39.5 ±37
Mild (n=16)	51±33.1	57.5±30	48.3±29.4	74.5±11.6	127.4±119.4	39.6±38.8
Moderate (n=15)	61.3±32.4	73±30	47±39.08	64.4±0.6	158.3±112.5	66.3±32.8
Severe (n=3)	44.1±16.4	61.9±23.5	31.2±24.7	62.6±15.9	69±41	79.9±33.8
P value	0.33	0.10	0.77	0.01*	0.53	0.02*

* significant * data presented by mean±SD

Table 5: ROC curve analysis for stop bang and ESS for prediction of different cut off for AHI and ODI

		AUC	95 CI	P value	Cut of value	Sensitivity	Specificity	PPV	NPV
AHI ≥30	Stop bang	0.78	0.66-0.90	<0.001*	>6	78%	66.7%	90%	46%
	ESS	0.92	0.83-1	<0.001*	>15	95%	81%	95%	81%
ODI ≥30	Stop bang	0.86	0.77-0.96	<0.001*	>6	83.5%	89%	94.3%	56.7%
	ESS	0.92	0.86-0.99	<0.001*	>15	95%	81%	95%	81%

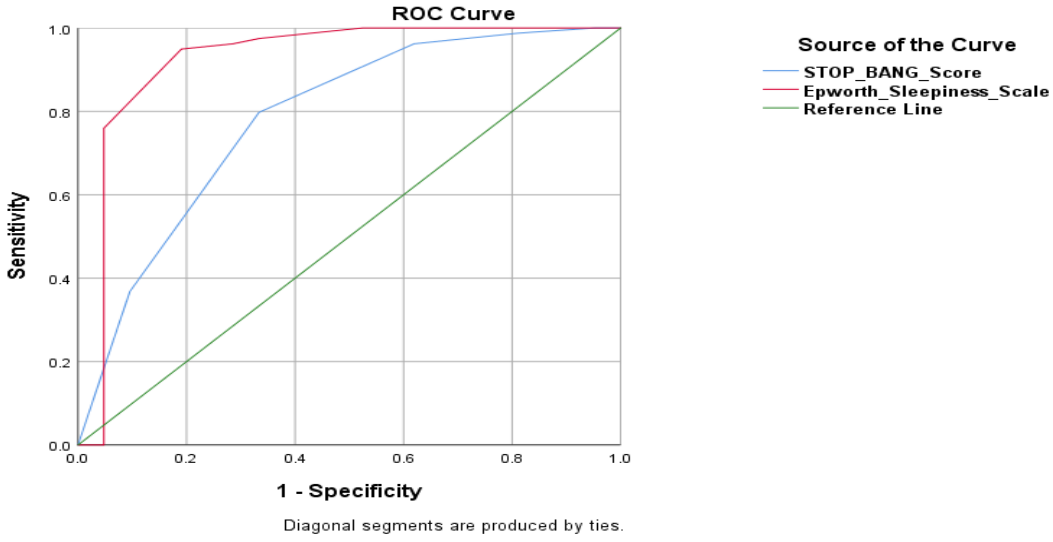

Figure (1) ROC curve analysis for stop bang and ESS for prediction AHI≥ 30

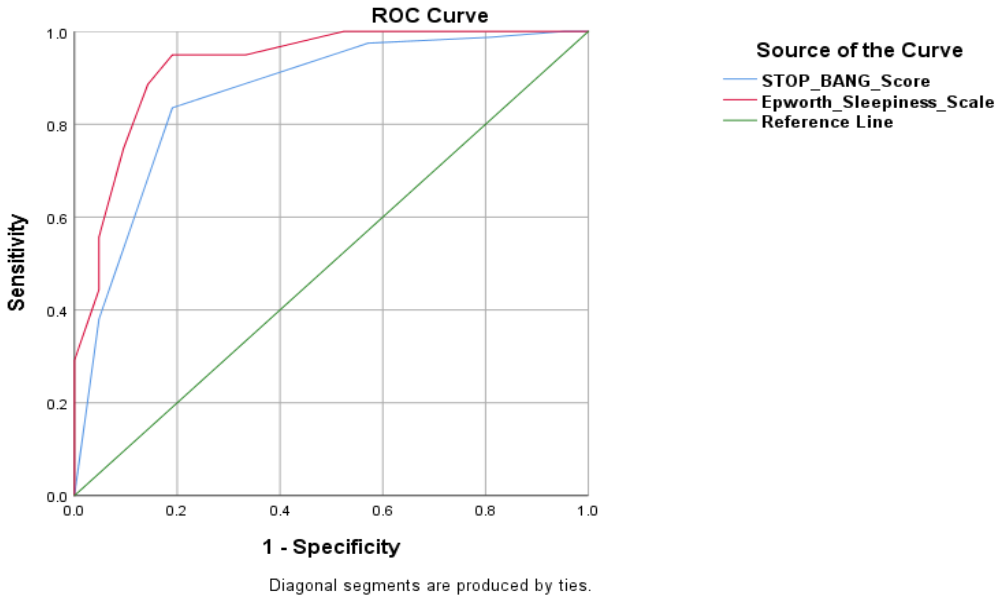

Fig (2) ROC curve analysis for stop bang and ESS for prediction ODI≥ 30

Table 6: ROC curve analysis for AHI and ODI for prediction of severe EDS

	AHI	ODI
AUC	0.94	0.92
95% CI	0.87-0.99	0.84-0.99
P value	<0.001*	<0.001*
Cut off value	>30.05	>38.8
Sensitivity	95%	91%
Specificity	85.7%	86%
PPV	96.2%	96%
NPV	81.8%	72%
Total accuracy	93%	90%

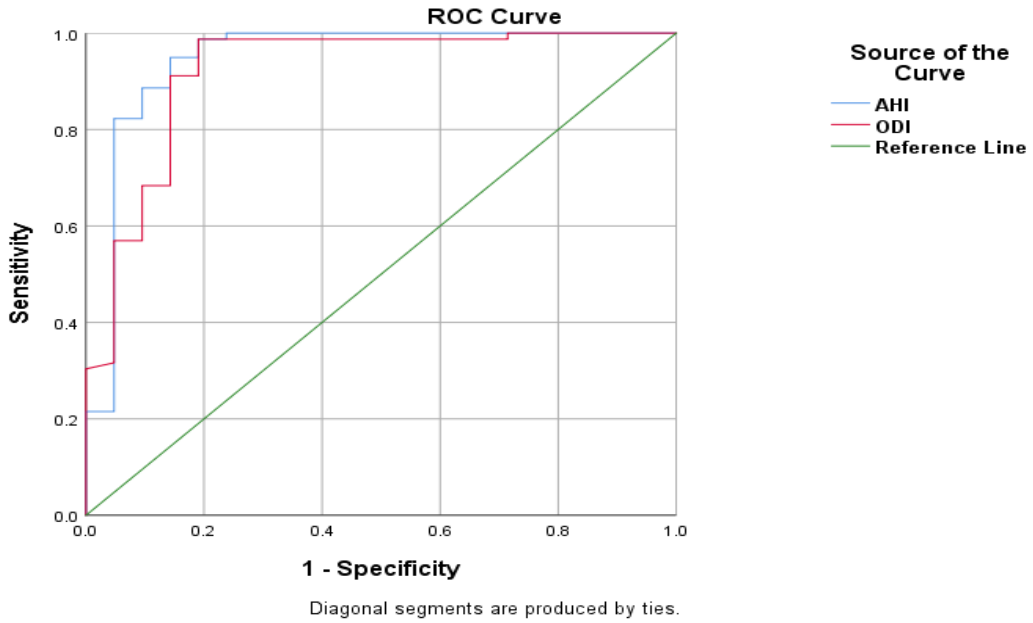

Figure 3: ROC curve analysis for AHI and ODI for prediction of severe EDS

Discussion

Obstructive sleep apnea (OSA) is a complex and underdiagnosed condition with far-reaching health implications, including increased risk for cardiovascular disease, metabolic dysfunction, and impaired quality of life. As awareness of OSA grows, there is a pressing need to refine diagnostic strategies and better understand the clinical markers that reflect disease severity [11, 12]. Current research emphasizes the importance of integrating objective physiological measures with symptom-based screening tools to improve early detection and targeted treatment .

In the present study, we found that the severity of OSA based on the Apnea-Hypopnea Index (AHI) was classified as mild in 12 patients (12%), moderate in 10 patients (10%), and severe in 78 patients (78%). The mean age was 61.7 ± 9.9 years ranged from (35 to 78) years. Males constituted 54% of the study population, while females represented 46%. Mean Body Mass Index (BMI) was 41.4 ± 6.5 kg/m², ranging from (27.3 to 55.5) kg/m² .

In line with our results, Veugen et al. showed 148 (73.6%) patients were male, aged 50.0 ± 12.6 years, with a mean BMI of 28.0 ± 4.8 kg/m2. Based on the AHI, OSA was present in 159 (79.1%) of the patients; 66 (41.5%) with mild OSA, 45 (28.3%) with moderate OSA, and 48 (30.2%) with severe OSA [13]. Myllymaa et al. reported that patient group was mostly male, middle-aged, and generally obese[14]. In the same line, Corral et al. showed that OSA and OHS patients are typically older due to the gradual development of obesity-related hypoventilation.[15]

In the present study, it was found that there was moderate to strong association between AHI and ODI where overall Kappa agreement coefficient was 0.66 ($p < 0.001$). In systematic review conducted by Rashid et al. it was found that a strong correlation between AHI and ODI for diagnosing OSA. The agreement between AHI and ODI was high in the severe OSA cases, with 78% of the severe OSA cases diagnosed as severe based on both indices [16]. These results are consistent with Kong et al. and Myllymaa et al. findings where higher ODI values were associated with more severe OSA. However, 22% of cases were misclassified between mild and moderate OSA [2, 14], where differing desaturation thresholds led to discrepancies in severity classification.

In the present study, there was a moderate positive correlation was observed between Body Mass Index (BMI) and both the Apnea-Hypopnea Index (AHI) ($r = 0.39$, $p < 0.001$) and the Oxygen Desaturation Index (ODI) ($r = 0.36$, $p < 0.001$). These findings align with previous research indicating that increased BMI

is associated with greater OSA severity. For instance, a study by Halima et al. reported a significant positive correlation between BMI and AHI ($\rho = 0.13$, $p = 0.005$) in a cohort of 150 patients, suggesting that higher BMI values are linked to increased OSA severity [17].

Additionally, we observed that waist circumference and neck circumference also demonstrated positive correlations with both AHI ($r = 0.47$, $p < 0.001$) and ODI ($r = 0.40$, $p < 0.001$). These results are consistent with findings from Lovin et al., who reported moderate correlations between AHI and neck circumference ($r = 0.54$) and abdominal circumference ($r = 0.75$), emphasizing the role of central obesity in OSA pathogenesis.[18]

In our study, we found that the STOP-BANG questionnaire showed a strong positive correlation with AHI ($r = 0.60$, $p < 0.001$) and a slightly weaker yet significant correlation with ODI ($r = 0.52$, $p < 0.001$). This supports the utility of the STOP-BANG questionnaire as an effective screening tool for identifying individuals at risk for OSA according to a study conducted by Easeem et al. study[19].

Also, Chen et al. who examined the diagnostic performance of the STOP-BANG questionnaire as a screening tool for obstructive sleep apnea (OSA) across different ethnic groups suggesting its association AHI and effective screening tool for identifying individuals at risk for OSA [20]. Additionally, Choi et al. found that the STOP-BANG questionnaire has moderate sensitivity and specificity for detecting OSA, particularly for moderate to severe cases. Specifically, for AHI ≥15, the sensitivity was 74.3%, and for AHI ≥30, it was 79.5%.[21]

Similarly, the Epworth Sleepiness Scale (ESS) correlated strongly with both AHI ($r = 0.60$, $p < 0.001$) and ODI ($r = 0.59$, $p < 0.001$). These findings are in line with Karakus et al. who found a moderate positive correlation between ESS scores and ODI, and weaker correlations with AHI and arousal index [22]. This suggests that ODI may be a more sensitive indicator of subjective daytime sleepiness in OSA patients.
Interestingly, age did not show a statistically significant correlation with either AHI ($r = 0.09$, $p = 0.33$) or ODI ($r = 0.17$, $p = 0.08$) in this cohort. While some studies have reported an increase in OSA prevalence and severity with age, others suggest that age-related changes in sleep architecture and upper airway anatomy may not uniformly affect all individuals [23]. Additionally, Liu et al. indicated that age modifies the associations between obesity indices and OSA severity in a sex-specific manner, with stronger associations observed in younger men compared to older men [24]. The lack of significant correlation in this study may be due to the age distribution of the sample or other confounding factors.

In our study, we found that gender was not significantly associated with AHI, ODI, or REM AHI, females exhibited significantly lower minimal SpO_2 levels compared to males ($p = 0.04$). This aligns with prior research indicating that women often have milder OSA during non-REM sleep but may experience more pronounced oxygen desaturation during REM sleep while they had a greater clustering of respiratory events during REM sleep than men [25]. Additionally, Sutherland et al. suggested that male patients generally exhibit less REM-related apnea severity compared to females. Those results suggested a gender-specific pattern in OSA manifestation.[26]

In the current study, we found that smoking status was significantly associated with higher ODI values ($p = 0.02$), particularly among ex-smokers. However, AHI and other oxygen parameters did not show statistically significant differences across smoking categories. This finding is consistent with studies suggesting that smoking may exacerbate oxygen desaturation during sleep without necessarily increasing the frequency of apneic events. For instance, Oțelea et al. highlighted the deleterious effects of continued smoking on OSA, emphasizing its impact on oxygen desaturation [27]. This observation is consistent with findings from Wu et al. showed that ex-smokers tend to have more severe OSA, especially during REM sleep[28] .

Our results show that higher AHI and ODI values were significantly associated with major cardiovascular and metabolic conditions including (ischemic heart disease (IHD), diabetes mellitus) suggesting that nocturnal hypoxemia plays a crucial role in disease progression. These findings are consistent with those of Kong et al. who also highlighted the role of ODI as a strong predictor of

cardiovascular complications in OSA patients .[2]

Our study found that REM AHI was significantly elevated in patients with IHD and diabetes which corroborates the results by Liu et al. who found that REM-related apnea events are more pronounced in patients with cardiovascular diseases [29] suggesting that REM-related apneas contribute significantly to the progression of cardiovascular diseases, especially in patients with IHD and diabetes. Additionally, hypertension was associated with higher REM AHI though the difference was not statistically significant, similar to findings by Wang et al. indicated a possible link between REM sleep apneas and hypertension but did not find a significant statistical association in some cases [30].

The findings of this study reaffirm the crucial role of AHI and ODI in assessing the severity of OSA and its associated cardiovascular and metabolic risks. Both indices correlate strongly with other clinical parameters, such as sleep fragmentation and oxygen desaturation, as well as with obesity markers. These results are consistent with Blekic et al. emphasized the importance of using these indices as reliable tools in diagnosing and managing OSA [31]. Moreover, the relationship between REM AHI and severe comorbidities such as IHD and diabetes further highlights the need for comprehensive OSA management, including targeted interventions for those with significant nocturnal hypoxemia. A review by Reutrakul et al. highlighted the role of OSA in the pathogenesis of type 2 diabetes and its complications [32]. A previous study conducted by Embarak et al. revealed that there was a strong association between OSA and diabetes [33]. Moreover, our results support the established link between OSA and cardiovascular diseases [34].

Xu et al. emphasized the association between various polysomnographic measures of OSA and incident major adverse cardiovascular events [35]. Watanabe et al. emphasized the importance of oxygen desaturation metrics in heart failure and central sleep apnea patients, supporting the idea that desaturation levels serve as vital prognostic markers in OSA-related cardiovascular risks [36]. Furthermore, Varghese et al. and Frangopoulos et al. demonstrated that oxygen desaturation, as measured by ODI, provides a reliable alternative in screening for severe OSA, especially in patients with comorbidities like cardiovascular disease .[11 ,10]

In our study, we found that pulmonary hypertension showed a significant association with minimal SpO_2 ($p = 0.01$) and SpO_2 time <90% ($p = 0.02$), with more severe cases demonstrating greater oxygen desaturation, although differences in AHI and ODI were not statistically significant. This suggests that nocturnal hypoxemia may play a more critical role than the frequency of apneic events in the pathophysiology of pulmonary hypertension. A study by Huang et al. discussed the implications of prolonged nocturnal hypoxemia in patients with pulmonary hypertension and OSA.[37]

Our findings indicated that the Epworth Sleepiness Scale (ESS) demonstrated superior diagnostic accuracy over the STOP-BANG questionnaire across various thresholds of the Apnea-Hypopnea Index (AHI) and Oxygen Desaturation Index (ODI). Specifically, ESS achieved excellent accuracy for AHI ≥5 (AUC = 0.95, $p = 0.02$) with 92% sensitivity and 100% specificity at a cutoff >12. For higher AHI thresholds (≥15 and ≥30), ESS maintained high accuracy (AUCs = 0.98 and 0.92, respectively), with sensitivity ranging from 93% to 95% and specificity from 81% to 100%.

In contrast, STOP-BANG, with a consistent cutoff >6, showed good performance but lower accuracy compared to ESS, with AUCs ranging from 0.78 to 0.89 and reduced specificity at higher AHI thresholds. Similarly, for ODI thresholds (≥5, ≥15, and ≥30), ESS outperformed STOP-BANG, achieving AUCs between 0.92 and 0.94 ($p < 0.001$), with high sensitivity (86–95%) and specificity (81–100%), whereas STOP-BANG showed moderate accuracy (AUCs = 0.78–0.86) and was less discriminative at lower ODI levels.

However, it's important to note that existing literature presents varying results regarding the comparative performance of ESS and STOP-BANG. For instance, a bivariate meta-analysis by Senaratna et al. evaluated the diagnostic accuracy of the Berlin Questionnaire, STOP-BANG, STOP, and ESS in detecting obstructive sleep apnea (OSA). The study found that for

mild OSA (AHI ≥5), the pooled sensitivity of STOP-BANG was 88%, while ESS had a sensitivity of 54%. For moderate OSA (AHI ≥15), STOP-BANG's sensitivity was 90%, compared to ESS's 47%. In severe OSA (AHI ≥30), STOP-BANG achieved a sensitivity of 93%, whereas ESS had 58%. Although ESS demonstrated higher specificity than STOP-BANG across all severity levels, its lower sensitivity suggests it may miss a significant number of OSA cases .[38]

Furthermore, a study by van der Veen et al. compared the predictive performance of the NoSAS score, STOP-BANG questionnaire, and ESS concerning both AHI and ODI. Their findings indicated that the STOP-BANG questionnaire and the NoSAS score were equally adequate screening tools for OSA severity based on both AHI and ODI. In contrast, the ESS did not show adequate discrimination for screening OSA, with AUCs ranging from 0.450 to 0.525 .[13]

These findings are particularly noteworthy given the longstanding emphasis on STOP-BANG as a preferred OSA screening tool. In its foundational validation studies, STOP-BANG was shown to have high sensitivity (often >90%) but lower specificity, particularly in populations with high pre-test probability of OSA [12, 39].

While useful in identifying patients at risk, the lower specificity often leads to over-referral for sleep studies, placing a burden on diagnostic resources. In contrast, the ESS, historically viewed as a subjective measure of daytime sleepiness, has often been criticized for poor sensitivity, particularly in asymptomatic or minimally symptomatic individuals. However, the present study shows that when an optimal cutoff (ESS >12) is used, the tool can achieve both high sensitivity and specificity, particularly in clinical populations where symptoms correlate more directly with disease severity.

These results align partially with previous research. For example, a systematic review by Abrishami et al. found that STOP-BANG's specificity could be as low as 25% depending on the cutoff used, while the ESS performed better in select clinical samples [40]. Moreover, Silva et al. found poor overall predictive value for ESS in population-based cohorts [41]. The strong correlation between ESS and OSA severity in this context suggests that subjective sleepiness, when quantified carefully, can serve as a reliable proxy for underlying respiratory disturbance, particularly when hypoxia is a dominant feature. These discrepancies highlight the importance of considering population characteristics, study design, and the specific metrics used when interpreting the diagnostic performance of these screening tools .

Both AHI and ODI exhibited excellent predictive power for severe EDS, with AUCs of 0.94 and 0.92, respectively ($p < 0.001$ for both). AHI >30.05 provided 95% sensitivity and 85.7% specificity, while ODI >38.8 yielded 91% sensitivity and 86% specificity. Both demonstrated high PPVs (96.2% and 96%) and overall diagnostic accuracy (93% and 90%, respectively).

Aligning with findings by Zhang et al. found that AHI and ODI to be effective in predicting daytime sleepiness in OSA patients. Our study reported comparable sensitivity values (95% for AHI, 91% for ODI), indicating both indices are highly sensitive in detecting EDS. However, specificity was lower than sensitivity (85.7% for AHI and 86% for ODI), pointing to a higher rate of false positives [42]. This is in accordance with Li et al. who noted that while AHI and ODI are sensitive for predicting EDS, there is a need to refine diagnostic criteria to enhance specificity.[43]

In a study by Sharma et al. studying the relationship between AHI and ODI in OSA was analyzed, revealing that both indices are crucial in determining the severity of the condition. This supports our findings that AHI and ODI show strong correlations in assessing the severity of sleep apnea [44]. Similarly, Chokesuwattanaskul et al. examined the association of hypoxia, measured by ODI, with cognitive function in middle-aged and older adults, highlighting the broader impact of OSA-related hypoxia on various health parameters .[45]

To enhance the management and outcomes of obstructive sleep apnea (OSA), a multifaceted approach is essential. Strategies to improve CPAP compliance—such as patient education, close follow-up, and support systems—can

significantly boost treatment adherence and efficacy. Combining AHI and ODI offers a more comprehensive assessment of OSA severity, particularly in patients with cardiovascular comorbidities, where reliance on a single index may underestimate disease impact. Regular monitoring of minimal SpO_2 levels and desaturation events is also crucial, especially in high-risk populations, to mitigate the risk of adverse outcomes. Given the strong association between OSA and cardiometabolic conditions like hypertension, diabetes, and heart disease, aggressive management of these comorbidities should be a priority in OSA care.

Additionally, greater attention should be paid to REM-related apneas, as they play a key role in exacerbating sleep fragmentation and nocturnal hypoxemia, further amplifying disease burden.
Our limitations lie in its retrospective design that may have introduced potential biases related to data accuracy and completeness, and the findings may not have been generalizable to populations outside the study context.

Conclusion

This study highlights the strong diagnostic concordance between AHI and ODI in evaluating OSA severity and their significant associations with clinical and anthropometric parameters. The Epworth Sleepiness Scale demonstrated superior accuracy over STOP-BANG in predicting OSA severity and excessive daytime sleepiness, particularly in severe cases. Cardiometabolic comorbidities—including hypertension, diabetes, and ischemic heart disease—were closely linked to greater OSA severity and oxygen desaturation burden. These findings emphasize the importance of integrating symptom-based screening tools with objective sleep study data to enhance early detection, risk stratification, and management of patients with OSA.

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