Section: Miscellaneous

Original Research Article

PROSPECTIVE ASSESSMENT OF OVERNIGHT OXIMETRY PARAMETERS AS PREDICTORS OF SEVERITY IN SUSPECTED OBSTRUCTIVE SLEEP APNEA PATIENTS

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent upper airway obstruction during sleep, resulting in intermittent hypoxemia and sleep fragmentation. Although polysomnography remains the reference standard for diagnosis, it is often limited by cost, accessibility, and long waiting times. Overnight pulse oximetry has emerged as a simple, noninvasive, and cost-effective tool that may help in screening patients and predicting the severity of OSA. Oximetry-derived parameters such as mean nocturnal oxygen saturation, minimum oxygen saturation, oxygen desaturation index, total desaturation events, and percentage of time spent below 90% oxygen saturation may provide valuable information regarding disease burden. **Aim:** To prospectively assess overnight oximetry parameters as predictors of severity in patients with suspected obstructive sleep apnea at a tertiary care hospital.

Materials and Methods: This prospective observational study included 80 consecutive adult patients with clinical suspicion of OSA attending a tertiary care hospital. Detailed history, clinical examination, and anthropometric assessment were performed in all participants. Baseline variables included age, sex, body mass index, neck circumference, Epworth Sleepiness Scale score, symptoms suggestive of OSA, and associated comorbidities. All participants underwent overnight pulse oximetry under standardized conditions. The recorded oximetry parameters included mean nocturnal SpO_2, minimum SpO_2, oxygen desaturation index (ODI), total desaturation events, T90, and baseline SpO_2. OSA severity was categorized by reference sleep study findings into no OSA, mild, moderate, and severe OSA. Data were analyzed using SPSS version 27.0, and p value <0.05 was considered statistically significant.

Results: Of the 80 participants, 8 (10.00%) had no OSA, 20 (25.00%) had mild OSA, 24 (30.00%) had moderate OSA, and 28 (35.00%) had severe OSA. Age, body mass index, neck circumference, and Epworth Sleepiness Scale score increased significantly with OSA severity. Symptoms such as habitual snoring, witnessed apnea, excessive daytime sleepiness, nocturnal choking, and hypertension were significantly associated with increasing severity. All overnight oximetry parameters showed significant worsening across severity groups. Mean nocturnal SpO_2 and minimum SpO_2 progressively declined, whereas ODI, total desaturation events, and T90 increased significantly ($p<0.001$). ROC analysis showed that ODI had the best diagnostic performance for predicting moderate-to-severe and severe OSA, followed by minimum SpO_2 and T90.

Conclusion: Overnight pulse oximetry parameters, particularly ODI, minimum SpO_2, and T90, are reliable predictors of OSA severity in suspected patients. Overnight oximetry can serve as a useful, noninvasive, and cost-effective screening tool for early severity stratification in tertiary care settings.
Keywords: Obstructive sleep apnea; overnight pulse oximetry; oxygen desaturation index; nocturnal hypoxemia; severity prediction.

INTRODUCTION

Obstructive sleep apnea is one of the most important sleep-related breathing disorders encountered in adult clinical practice. It is characterized by recurrent episodes of complete or partial upper airway obstruction during sleep, leading to cyclical oxygen desaturation, sleep fragmentation, intrathoracic pressure swings, and repeated arousals from sleep. These pathophysiological events do not merely disturb nocturnal sleep architecture, but also contribute to impaired daytime functioning, reduced quality of life, neurocognitive dysfunction, and long-term systemic complications. In routine clinical settings, patients commonly present with loud snoring, witnessed apneas, non-restorative sleep, nocturnal choking or gasping, morning tiredness, and excessive daytime sleepiness. However, the clinical presentation is often heterogeneous, and a considerable proportion of affected individuals remain unrecognized until symptoms become more severe or associated comorbidities draw medical attention.[1] The burden of obstructive sleep apnea has increased substantially with changing lifestyles, rising obesity, population aging, and greater awareness of sleep disorders. OSA is now recognized not only as a common respiratory disorder but also as a major public health problem because of its close association with cardiometabolic disease. Repeated intermittent hypoxemia and reoxygenation generate oxidative stress, endothelial dysfunction, systemic inflammation, and sympathetic overactivity, thereby linking OSA to hypertension, coronary artery disease, heart failure, arrhythmias, stroke, and metabolic derangements. In this context, the severity of nocturnal oxygen disturbance has gained increasing importance, because oxygen-related indices may reflect disease burden more meaningfully than symptom reporting alone. This broader understanding has shifted attention from merely identifying the presence of OSA to recognizing the clinical importance of its physiological severity.[2] Despite its high clinical relevance, the diagnosis of OSA continues to be challenging in many healthcare settings. Polysomnography remains the reference standard, but it is resource-intensive, laborious, and often limited by cost, specialized infrastructure, long waiting times, and reduced accessibility, especially in developing regions and busy tertiary care centers. Even home sleep apnea testing, although more convenient than laboratory-based studies, may not always be readily available or feasible for all patients. These practical limitations create a gap between the large number of symptomatic patients who require evaluation and the limited diagnostic resources available to confirm severity in a timely manner. As a result, there is a strong need for simpler, more accessible, and scalable tools that can identify high-risk patients and help prioritize further definitive assessment.[3] Overnight pulse oximetry has emerged as an attractive option in this setting because it is noninvasive, inexpensive, easy to administer, and capable of capturing one of the central physiological consequences of OSA, namely episodic nocturnal desaturation. Unlike complex multichannel sleep studies, oximetry can be performed with minimal patient burden and may be used in hospital-based as well as home-based settings. It provides objective information on oxygen saturation trends throughout sleep and therefore offers a practical means of screening patients with suspected OSA. In recent years, simplified screening pathways that combine questionnaires, clinical risk factors, daytime saturation, and overnight oximetry have gained support because they can reduce the need for immediate full sleep studies in all suspected cases while still identifying those with a higher probability of clinically significant disease.[4] The usefulness of overnight oximetry lies not only in detecting abnormal breathing-related oxygen changes, but also in generating measurable parameters that may correlate with disease severity. Commonly assessed variables include mean nocturnal oxygen saturation, minimum oxygen saturation, baseline oxygen saturation, oxygen desaturation index, total number of desaturation events, and the proportion of sleep time spent below a defined saturation threshold such as 90%. These parameters reflect both the frequency and depth of nocturnal hypoxemia and may therefore serve as surrogate markers of the physiological burden imposed by OSA. Traditional severity assessment has relied heavily on the apnea-hypopnea index, yet growing attention has been directed toward oximetry-derived measures because they may capture aspects of disease severity that are more closely linked to adverse outcomes.[5] Advances in digital health, signal processing, and wearable technologies have further strengthened the role of oxygen-based monitoring in sleep medicine. Recent work has shown that saturation-derived features can be incorporated into machine learning and sequential screening models to improve classification of OSA risk and severity. These developments support the concept that SpO_2-based monitoring is not merely a preliminary screening aid, but may become an increasingly valuable component of severity prediction and triage. Even where advanced analytic methods are used, the clinical foundation remains the

same: oxygen saturation dynamics during sleep provide meaningful physiological information that can help identify patients with more severe disease. This is particularly relevant in symptomatic adults, where early recognition of moderate or severe OSA can facilitate faster referral, targeted management, and better use of limited diagnostic resources.[6]

MATERIALS AND METHODS

This prospective observational study was conducted at a tertiary care hospital. The study was designed to assess the usefulness of overnight pulse oximetry parameters as predictors of disease severity in patients with suspected obstructive sleep apnea (OSA). All eligible participants were evaluated in a systematic manner after detailed clinical assessment and were subsequently subjected to overnight oximetry and reference sleep study-based severity classification. A total of 80 consecutive adult participants with suspected obstructive sleep apnea were included in the study. Patients were recruited from those presenting to the tertiary care hospital with symptoms suggestive of sleep-disordered breathing, including habitual snoring, witnessed apneas, excessive daytime sleepiness, non-refreshing sleep, morning headache, nocturnal choking episodes, or unexplained fatigue. Only patients who were willing to participate and provide informed consent were enrolled in the study.

Eligibility Criteria: Adult patients aged 18 years and above with clinical suspicion of OSA were included in the study. Patients were excluded if they had previously diagnosed OSA and were already on treatment such as continuous positive airway pressure therapy, had central sleep apnea, unstable cardiorespiratory illness, severe chronic obstructive pulmonary disease, decompensated heart failure, neuromuscular disorders affecting respiration, active respiratory infection, or any condition likely to interfere with accurate overnight oximetry recordings. Patients with incomplete or poor-quality oximetry recordings were also excluded from final analysis.

Clinical assessment and baseline evaluation: After enrollment, all participants underwent detailed history taking and clinical examination. Baseline variables recorded included age, sex, body mass index, neck circumference, history of smoking, alcohol intake, comorbidities such as hypertension and diabetes mellitus, and symptoms suggestive of OSA. Daytime sleepiness was assessed using the Epworth Sleepiness Scale where applicable. Standard anthropometric measurements were obtained using calibrated instruments and recorded in a structured proforma.

Overnight oximetry assessment: All participants underwent overnight pulse oximetry using a validated recording pulse oximeter under standardized conditions. The recording was performed during usual nocturnal sleep, and the oximetry data were downloaded and reviewed for adequacy and artifact exclusion before analysis. The overnight oximetry parameters recorded included mean nocturnal oxygen saturation (mean SpO2), minimum oxygen saturation (nadir SpO2), oxygen desaturation index (ODI), total number of desaturation events, percentage of recording time spent with oxygen saturation below 90% (T90), baseline oxygen saturation, and cumulative time spent below selected saturation thresholds where available. These parameters were chosen because they reflect intermittent hypoxemia and nocturnal oxygen burden, which are closely related to the pathophysiological severity of OSA.

Outcome Measures: The primary objective was to prospectively assess whether overnight oximetry parameters could predict the severity of OSA in patients with clinical suspicion of the disorder. The main outcome variables included the association of oxygen desaturation index, nadir oxygen saturation, mean nocturnal oxygen saturation, and T90 with OSA severity categories. Secondary assessment included evaluation of the diagnostic performance of selected oximetry parameters in identifying moderate-to-severe and severe OSA.

Data collection and quality control: All clinical, anthropometric, and oximetry data were recorded in a predesigned case record form. Oximetry recordings were checked for motion artifacts, signal dropout, and recording adequacy before inclusion in final analysis. Only technically satisfactory recordings were considered for statistical analysis. Uniform procedures were followed for patient assessment and data entry to maintain consistency and reduce observer-related variation.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software version 27.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range depending on the distribution of data, while categorical variables were presented as frequencies and percentages. Normality of continuous variables was assessed before application of inferential tests. Comparison of overnight oximetry parameters across OSA severity groups was performed using independent sample t-test or one-way analysis of variance for normally distributed variables, and Mann-Whitney U test or Kruskal-Wallis test for non-normally distributed variables, as appropriate. Association between categorical variables was analyzed using chi-square test or Fisher's exact test. Correlation between oximetry parameters and apnea-hypopnea index was assessed using Pearson's or Spearman's correlation coefficient as applicable. Receiver operating characteristic curve analysis was performed to determine the predictive ability of important overnight oximetry parameters and to identify optimal cut-off values for predicting moderate-to-severe and severe OSA. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1 shows the distribution of study participants according to OSA severity. Out of the total 80 participants, 8 patients (10.00%) had no OSA, 20 patients (25.00%) had mild OSA, 24 patients (30.00%) had moderate OSA, and 28 patients (35.00%) had severe OSA. Thus, the largest proportion of participants belonged to the severe OSA group, followed by the moderate OSA group. When moderate and severe OSA were considered together, 52 out of 80 participants, constituting 65.00% of the study population, had clinically significant disease.

Table 2 presents the baseline demographic and anthropometric characteristics across OSA severity categories. The mean age increased progressively from 41.63 ± 8.21 years in the no OSA group to 44.85 ± 9.14 years in the mild OSA group, 48.42 ± 8.76 years in the moderate OSA group, and 51.96 ± 9.88 years in the severe OSA group, and this difference was statistically significant ($p = 0.012$). This finding suggests that increasing age was associated with greater severity of OSA in the study population. With regard to sex distribution, the proportion of males was high in all groups and increased from 62.50% in the no OSA group to 85.71% in the severe OSA group.

Body mass index showed a strong and statistically significant rise with increasing OSA severity. The mean BMI was 24.96 ± 2.31 kg/m² in the no OSA group, 27.84 ± 2.96 kg/m² in the mild OSA group, 30.12 ± 3.21 kg/m² in the moderate OSA group, and 33.46 ± 3.84 kg/m² in the severe OSA group ($p < 0.001$). This progressive rise supports the known role of obesity as an important risk factor for OSA and suggests that increasing body mass is closely linked to worsening airway obstruction during sleep. Neck circumference also increased significantly across severity categories, from 35.12 ± 2.06 cm in no OSA to 41.63 ± 2.95 cm in severe OSA ($p < 0.001$). This indicates that central fat distribution and increased upper airway soft tissue mass may contribute substantially to disease severity. Similarly, the Epworth Sleepiness Scale score increased steadily from 7.25 ± 2.19 in the no OSA group to 14.68 ± 3.37 in the severe OSA group, and this difference was highly significant ($p < 0.001$).

Table 3 summarizes the distribution of clinical symptoms and comorbidities according to OSA severity. Habitual snoring was present in 62.50% of participants with no OSA, increasing to 80.00% in mild OSA, 91.67% in moderate OSA, and 100.00% in severe OSA, with a statistically significant association ($p = 0.009$). This indicates that snoring became more frequent as disease severity increased and was universally present in severe OSA. Witnessed apnea also showed a marked increase across the groups, from 12.50% in no OSA to 85.71% in severe OSA, which was highly significant ($p < 0.001$).

Excessive daytime sleepiness showed a similar significant trend, occurring in 25.00% of the no OSA group, 40.00% of the mild OSA group, 66.67% of the moderate OSA group, and 85.71% of the severe OSA group ($p < 0.001$). This supports the clinical relevance of daytime somnolence as a marker of more severe disease. Morning headache was more common with increasing severity, rising from 12.50% in no OSA to 42.86% in severe OSA; however, the association was not statistically significant ($p = 0.154$).

Nocturnal choking was reported by 12.50% of participants without OSA, 25.00% with mild OSA, 41.67% with moderate OSA, and 64.29% with severe OSA, showing a statistically significant association ($p = 0.006$). This suggests that nocturnal choking is an important symptom that increases with worsening disease severity. Hypertension was present in 12.50% of the no OSA group and rose to 53.57% in the severe OSA group, with the association being statistically significant ($p = 0.029$). This finding supports the recognized link between OSA and cardiovascular risk, particularly in patients with more severe disease. Diabetes mellitus also showed an increasing trend from 0.00% in the no OSA group to 35.71% in the severe OSA group, but this did not achieve statistical significance ($p = 0.108$). Smoking history was relatively similar across the severity groups and showed no statistically significant relationship with OSA severity ($p = 0.842$).

Table 4 compares the overnight oximetry parameters across the different OSA severity groups and demonstrates highly significant differences for all parameters studied. Mean nocturnal oxygen saturation progressively declined from 96.24 ± 0.88% in the no OSA group to 94.92 ± 1.16% in mild OSA, 92.48 ± 1.84% in moderate OSA, and 89.76 ± 2.42% in severe OSA ($p < 0.001$). This indicates that average oxygenation during sleep worsened steadily with increasing disease severity. Minimum oxygen saturation also showed a marked fall across severity groups, from 90.38 ± 2.13% in no OSA to 69.82 ± 6.14% in severe OSA ($p < 0.001$).

The oxygen desaturation index, which represents the frequency of significant oxygen drops per hour, increased dramatically from 3.12 ± 1.08 events/hour in the no OSA group to 9.84 ± 3.26 in mild OSA, 20.58 ± 5.74 in moderate OSA, and 39.71 ± 9.63 in severe OSA ($p < 0.001$). This demonstrates a strong positive relationship between ODI and OSA severity, suggesting that ODI is a robust oximetric marker of disease burden. Similarly, the total number of desaturation events increased substantially from 18.63 ± 6.74 in no OSA to 238.11 ± 56.84 in severe OSA ($p < 0.001$), showing that severe OSA is associated with a much higher frequency of nocturnal hypoxemic events.

The percentage of sleep time spent with oxygen saturation below 90% (T90) rose sharply from 0.84 ± 0.46% in participants without OSA to 28.56 ± 10.42% in those with severe OSA ($p < 0.001$). This indicates that patients with severe OSA spent a

significantly greater proportion of the night in a hypoxemic state, reflecting a higher cumulative oxygen burden. Baseline oxygen saturation also declined significantly across the groups, from 97.41 ± 0.74% in no OSA to 94.52 ± 1.54% in severe OSA ($p < 0.001$).

Table 5 evaluates the diagnostic performance of selected overnight oximetry parameters in predicting moderate-to-severe and severe OSA using receiver operating characteristic analysis. For prediction of moderate-to-severe OSA, an ODI cut-off value of greater than 15.00 events/hour had a sensitivity of 86.54% and specificity of 82.14%, with an area under the curve of 0.91 (95% CI: 0.84–0.97), and this was highly statistically significant ($p < 0.001$). This indicates excellent diagnostic accuracy and suggests that ODI is a strong predictor of clinically significant OSA. Minimum SpO_2 below 82.00% had a sensitivity of 80.77% and specificity of 78.57%, with an AUC of 0.87 (95% CI: 0.79–0.95; $p < 0.001$), while T90 greater than 8.00% had a sensitivity of 82.69% and specificity of 75.00%, with an AUC of 0.88 (95% CI: 0.80–0.95; $p < 0.001$). These values show that both nadir oxygen saturation and T90 also had good predictive performance, though slightly lower than ODI.

For prediction of severe OSA, ODI again showed the best overall performance. A cut-off value of greater than 30.00 events/hour yielded a sensitivity of 85.71% and specificity of 82.69%, with an AUC of 0.92 (95% CI: 0.85–0.98; $p < 0.001$), indicating excellent discriminatory ability. Minimum SpO_2 below 75.00% showed a sensitivity of 82.14% and specificity of 80.77%, with an AUC of 0.89 (95% CI: 0.82–0.96; $p < 0.001$), while T90 greater than 20.00% produced a sensitivity of 78.57% and specificity of 84.62%, with an AUC of 0.90 (95% CI: 0.83–0.97; $p < 0.001$).

Table 1: Distribution of study participants according to OSA severity (n = 80)

OSA severity category	Number of participants	Percentage (%)
No OSA	8	10.00
Mild OSA	20	25.00
Moderate OSA	24	30.00
Severe OSA	28	35.00
Total	**80**	**100.00**

Table 2: Baseline demographic and anthropometric characteristics according to OSA severity

Variable	No OSA (n=8)	Mild OSA (n=20)	Moderate OSA (n=24)	Severe OSA (n=28)	p value
Age (years), mean ± SD	41.63 ± 8.21	44.85 ± 9.14	48.42 ± 8.76	51.96 ± 9.88	0.012
Male sex, n (%)	5 (62.50)	13 (65.00)	18 (75.00)	24 (85.71)	0.184
BMI (kg/m²), mean ± SD	24.96 ± 2.31	27.84 ± 2.96	30.12 ± 3.21	33.46 ± 3.84	<0.001
Neck circumference (cm), mean ± SD	35.12 ± 2.06	37.46 ± 2.34	39.28 ± 2.61	41.63 ± 2.95	<0.001
Epworth Sleepiness Scale score, mean ± SD	7.25 ± 2.19	9.40 ± 2.58	11.92 ± 3.04	14.68 ± 3.37	<0.001

Table 3: Clinical symptoms and comorbidities according to OSA severity

Variable	No OSA (n=8)	Mild OSA (n=20)	Moderate OSA (n=24)	Severe OSA (n=28)	p value
Habitual snoring, n (%)	5 (62.50)	16 (80.00)	22 (91.67)	28 (100.00)	0.009
Witnessed apnea, n (%)	1 (12.50)	7 (35.00)	14 (58.33)	24 (85.71)	<0.001
Excessive daytime sleepiness, n (%)	2 (25.00)	8 (40.00)	16 (66.67)	24 (85.71)	<0.001
Morning headache, n (%)	1 (12.50)	4 (20.00)	8 (33.33)	12 (42.86)	0.154
Nocturnal choking, n (%)	1 (12.50)	5 (25.00)	10 (41.67)	18 (64.29)	0.006
Hypertension, n (%)	1 (12.50)	4 (20.00)	9 (37.50)	15 (53.57)	0.029
Diabetes mellitus, n (%)	0 (0.00)	3 (15.00)	6 (25.00)	10 (35.71)	0.108
Smoking history, n (%)	2 (25.00)	5 (25.00)	7 (29.17)	10 (35.71)	0.842

Table 4: Comparison of overnight oximetry parameters across OSA severity groups

Oximetry parameter	No OSA (n=8)	Mild OSA (n=20)	Moderate OSA (n=24)	Severe OSA (n=28)	p value
Mean nocturnal SpO_2 (%), mean ± SD	96.24 ± 0.88	94.92 ± 1.16	92.48 ± 1.84	89.76 ± 2.42	<0.001
Minimum SpO_2 (%), mean ± SD	90.38 ± 2.13	84.90 ± 3.62	78.29 ± 4.85	69.82 ± 6.14	<0.001
ODI (events/hour), mean ± SD	3.12 ± 1.08	9.84 ± 3.26	20.58 ± 5.74	39.71 ± 9.63	<0.001
Total desaturation events, mean ± SD	18.63 ± 6.74	62.25 ± 18.42	129.83 ± 32.96	238.11 ± 56.84	<0.001
T90 (% of sleep time), mean ± SD	0.84 ± 0.46	3.92 ± 2.11	11.74 ± 5.38	28.56 ± 10.42	<0.001
Baseline SpO_2 (%), mean ± SD	97.41 ± 0.74	96.86 ± 0.92	95.94 ± 1.16	94.52 ± 1.54	<0.001

Table 5: Diagnostic performance of selected overnight oximetry parameters for predicting moderate-to-severe and severe OSA

Parameter	Outcome predicted	Cut-off value	Sensitivity (%)	Specificity (%)	AUC (95% CI)	p value
ODI	Moderate-to-severe OSA	>15.00 events/hour	86.54	82.14	0.91 (0.84–0.97)	<0.001
Minimum SpO_2	Moderate-to-severe OSA	<82.00%	80.77	78.57	0.87 (0.79–0.95)	<0.001
T90	Moderate-to-severe OSA	>8.00%	82.69	75.00	0.88 (0.80–0.95)	<0.001
ODI	Severe OSA	>30.00 events/hour	85.71	82.69	0.92 (0.85–0.98)	<0.001
Minimum SpO_2	Severe OSA	<75.00%	82.14	80.77	0.89 (0.82–0.96)	<0.001
T90	Severe OSA	>20.00%	78.57	84.62	0.90 (0.83–0.97)	<0.001

DISCUSSION

In the present study, severe OSA constituted the largest subgroup, accounting for 28 of 80 patients (35.00%), followed by moderate OSA in 24 (30.00%), mild OSA in 20 (25.00%), and no OSA in 8 (10.00%); therefore, 65.00% of the cohort had moderate-to-severe disease. This relatively high proportion of clinically significant OSA is understandable in a tertiary care setting where symptomatic and higher-risk patients are more likely to be referred. A comparable pattern was reported by da Rosa et al. (2021), who studied older adults in a home-setting diagnostic model and found normal results in 7.54%, mild OSA in 28.72%, moderate OSA in 32.98%, and severe OSA in 30.85%, again showing that moderate and severe categories together formed the bulk of diagnosed cases. The similarity between the two studies supports the view that in symptomatic referred populations, the burden of clinically meaningful OSA is high, thereby justifying simplified tools such as overnight oximetry for early stratification and prioritization of full sleep evaluation.[7]

Age increased significantly with severity in our study, from 41.63 ± 8.21 years in the no OSA group to 51.96 ± 9.88 years in the severe OSA group ($p = 0.012$), and male predominance also rose from 62.50% to 85.71% across the same groups, although the sex association did not reach statistical significance. This pattern is broadly in agreement with Sreedharan et al. (2016), who evaluated 152 South Indian patients with PSG-proven OSA and reported a mean age of 53.8 years, with severe OSA present in 66 patients; importantly, they found age >55 years to be an independent predictor of greater OSA severity, while older subjects with witnessed apneas were more likely to have severe disease. Thus, the present findings reinforce that advancing age remains an important demographic marker of more severe OSA, even when sex differences may not always achieve statistical significance in smaller cohorts.[8]

Anthropometric risk rose strikingly with increasing severity in our series. Mean BMI increased from 24.96 ± 2.31 kg/m² in patients without OSA to 33.46 ± 3.84 kg/m² in severe OSA ($p < 0.001$), indicating a strong positive association between obesity and disease burden. Similar observations were made by Soylu et al. (2012) in a Turkish adult population, where the average BMI, waist circumference, and neck circumference were all significantly higher in the OSAS group than in controls ($p < 0.001$), and logistic regression identified enlargement in these anthropometric indices as significant risk factors for OSAS. They also proposed BMI cut-offs of >27.77 kg/m² in males and >28.93 kg/m² in females for increased OSAS risk, values that are well below the mean BMI seen in our severe OSA group, emphasizing that obesity in our cohort was not only prevalent but progressively related to worsening severity.[9]

Neck circumference in our study also showed a clear stepwise rise, from 35.12 ± 2.06 cm in the no OSA group to 41.63 ± 2.95 cm in severe OSA ($p < 0.001$), suggesting increasing upper airway soft tissue load and pharyngeal narrowing with severity. These results compare well with Ahbab et al. (2013), who assessed 44 patients with OSAS and found that both BMI and neck circumference were significantly higher in severe than in non-severe disease, with neck circumference emerging as an independent risk factor for severe OSAS ($p = 0.01$). In that study, 25 patients had severe disease and 19 had non-severe disease, and the authors concluded that neck circumference had particular utility in distinguishing severe from milder forms. The present data therefore support neck circumference as a simple bedside anthropometric marker that may add important predictive value alongside BMI in suspected OSA patients.[10]

Daytime somnolence in our study worsened steadily with disease severity, as reflected by the increase in Epworth Sleepiness Scale score from 7.25 ± 2.19 in the no OSA group to 14.68 ± 3.37 in severe OSA ($p < 0.001$). This finding is consistent with the concept that worse nocturnal respiratory disturbance and hypoxemia translate into greater daytime functional impairment. Mediano et al. (2007) similarly showed that patients with obstructive sleep apnoea syndrome and excessive daytime sleepiness had worse nocturnal oxygenation than nonsleepy patients, with lower lowest arterial oxygen saturation (69 ± 12% versus 79 ± 8%) and lower mean arterial oxygen saturation (87 ± 6% versus 90 ± 5%). Their study

emphasized that nocturnal hypoxaemia was a major determinant of excessive daytime sleepiness. Accordingly, the progressive increase in ESS score in our study likely reflects the same physiological pathway, namely greater overnight oxygen desaturation and sleep disruption in more severe OSA.[11]

The symptom profile in our study also showed a graded increase with severity. Habitual snoring rose from 62.50% in the no OSA group to 100.00% in severe OSA ($p = 0.009$), witnessed apnea from 12.50% to 85.71% ($p < 0.001$), excessive daytime sleepiness from 25.00% to 85.71% ($p < 0.001$), and nocturnal choking from 12.50% to 64.29% ($p = 0.006$). These observations parallel the report by Ibrahim et al. (2007), who studied 191 snorers undergoing PSG and found OSA in 126 patients (66%), with multivariate analysis showing that ESS, male sex, and history of witnessed apnea were significant predictors of OSA; specifically, the odds ratio was 4.35 for ESS ≥11 and 2.09 for witnessed apnea. Their cohort also had a mean age of 48.1 ± 9.8 years and 78.5% were males. Taken together, both studies underline that although symptoms alone cannot replace objective testing, snoring, witnessed apnea, and sleepiness become more frequent and clinically meaningful as OSA severity increases.[12]

Among comorbidities, hypertension increased markedly in our study from 12.50% in patients without OSA to 53.57% in severe OSA ($p = 0.029$), whereas diabetes mellitus showed only a nonsignificant upward trend and smoking history had no meaningful association with severity. The significant hypertension gradient in our study is supported by Wang et al. (2022), who evaluated 775 patients with OSA and demonstrated that higher T90 was independently associated with prevalent hypertension in severe OSA. In their severe OSA subgroup, hypertension prevalence increased from 70.85% in those with T90 <10.85% to 86.81% in those with T90 ≥10.85% ($p < 0.001$), and the odds of hypertension rose significantly as T90 severity increased. This comparison suggests that the hypertension association observed in our study is likely mediated, at least in part, by increasing nocturnal hypoxic burden with worsening OSA severity.[13]

Overnight oxygenation variables deteriorated significantly in our cohort. Mean nocturnal SpO_2 fell from 96.24 ± 0.88% in the no OSA group to 89.76 ± 2.42% in severe OSA, minimum SpO_2 dropped from 90.38 ± 2.13% to 69.82 ± 6.14%, and baseline SpO_2 declined from 97.41 ± 0.74% to 94.52 ± 1.54% (all $p < 0.001$). These findings are in line with Zhou et al. (2020), who reported that lower average oxygen saturation and lower minimal oxygen saturation during sleep were associated with greater severity of OSAS, and concluded that disease severity correlated with higher BMI, higher morning systolic blood pressure, and lower average and minimum oxygen saturation. Their observations support our results by showing that nocturnal oximetry variables do not merely reflect the presence of OSA, but also track its physiological severity. The pronounced fall in minimum SpO_2 in our severe group particularly indicates deeper repetitive desaturations and greater intermittent hypoxic stress.[14]

The oxygen desaturation index in our study increased sharply across severity groups, from 3.12 ± 1.08 events/hour in no OSA to 39.71 ± 9.63 events/hour in severe OSA, while total desaturation events rose from 18.63 ± 6.74 to 238.11 ± 56.84 ($p < 0.001$). This strong stepwise relationship supports ODI as one of the best quantitative markers of OSA burden. Varghese et al. (2022) likewise demonstrated good concordance between AHI and ODI, reporting a weighted kappa agreement of 87.32% and an R^2 of 0.84 between these two indices. They further showed that an ODI >20 had 96.6% sensitivity and 69.6% specificity for diagnosing severe OSA, and that ODI >25 still retained 89.7% sensitivity with 78.6% specificity. Compared with those data, the mean ODI of 39.71 events/hour in our severe group clearly falls well beyond the screening thresholds reported in that study, reinforcing the value of ODI as an objective surrogate for severity stratification.[15]

Receiver operating characteristic analysis in our study confirmed that ODI was the best single oximetry-derived discriminator. For moderate-to-severe OSA, ODI >15.00 events/hour yielded 86.54% sensitivity, 82.14% specificity, and AUC 0.91; for severe OSA, ODI >30.00 events/hour yielded 85.71% sensitivity, 82.69% specificity, and AUC 0.92. Minimum SpO_2 and T90 also performed well, but slightly less strongly. These findings compare favorably with Hang et al. (2015), who validated overnight oximetry in a large cohort and showed that ODI-based models achieved AUC values of 0.921–0.924 for diagnosing moderate-to-severe OSA and 0.953–0.957 for severe OSA, with sensitivity around 87.71–88.53% for moderate-to-severe disease and 89.36–89.87% for severe disease.[16]

CONCLUSION

In conclusion, overnight pulse oximetry parameters showed a strong and statistically significant association with the severity of obstructive sleep apnea in suspected patients. Among the studied variables, oxygen desaturation index, minimum SpO_2, and T90 emerged as useful predictors of moderate-to-severe and severe OSA. The findings suggest that overnight oximetry can serve as a simple, noninvasive, and cost-effective screening tool for early severity stratification in tertiary care settings. Thus, it may help identify high-risk patients who require priority evaluation and timely management.

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