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**Association of the six-minute walk test with severity of obstructive sleep apnea:
an observational study**

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Abstract

Obstructive sleep apnea (OSA) impairs sleep quality and exercise capacity through intermittent hypoxia and autonomic dysfunction. Simple tools that correlate objective exercise tolerance with OSA severity remain underutilized in clinical practice. The study aimed to find the association between 6-minute walk test (6MWT) performance and polysomnographic severity indices in patients with newly diagnosed OSA and whether functional exercise parameters can predict OSA severity. This cross-sectional observational study was conducted at a tertiary care center between July 2023 and January 2025. A total of 124 consecutive adults (age ≥ 18 years) with newly diagnosed OSA diagnosed from level I polysomnography (PSG) underwent standardized 6MWT as per American Thoracic Society Guidelines within one week of PSG testing. Primary endpoints included 6-minute walk distance (6MWD), oxygen saturation changes, post-test Borg dyspnea scale, and heart rate recovery at 1, 2, and 3 minutes. Correlations with Apnea-Hypopnea Index (AHI) and Oxygen Desaturation Index (ODI) were analyzed using Spearman's correlation, multiple linear regression, and one-way analysis of variance with Games-Howell post-hoc testing. The study included 124 participants (85.5% male, mean age 46 ± 11.3 years, mean body mass index 33.19 ± 5.9 kg/m²), of whom 79.8% were participants with severe OSA. Mean AHI and ODI were 52.25 ± 23.69 and 54.04 ± 29.22 events/hour, respectively. Mean 6MWD decreased significantly across OSA severity groups: mild 420.85 ± 17.4 m, moderate 396.99 ± 9.60 m, and severe 348.61 ± 22.01 m. Strong negative correlations were found between AHI and 6MWD ($\rho = -0.974$, $p < 0.001$) and ODI and 6MWD ($\rho = -0.775$, $p < 0.001$). Multiple linear regression results in two important predictive models: $AHI = 258.65 + [(0.58 \times \text{Epworth Sleepiness Scale (ESS)})] + (1.89 \times \text{Borg dyspnea post-6MWT}) - (0.1 \times 6MWD)$ and $ODI = 207.88 + (0.96 \times \text{ESS}) + (4.21 \times \text{Borg dyspnea post-6MWT}) - (0.51 \times 6MWD)$. As measured by the 6MWT, functional exercise capacity is strongly and inversely associated with the severity of OSA. The derived regression equations serve as a valuable, practical adjunct for assessing the functional impairment and hypoxic burden in patients with OSA, particularly in resource-limited settings.

Key words: sleep apnea, exercise tolerance, 6-minute walk test, polysomnography, functional capacity.

Introduction

Obstructive sleep apnea (OSA) is a common disorder involving repeated episodes of pharyngeal collapse during sleep, leading to apneas, hypopneas, and subsequent oxygen desaturation, hypercapnia, and sleep fragmentation. Clinically, it presents with symptoms like excessive daytime sleepiness. The prevalence of OSA is rising significantly, affecting around 936 million adults globally, which represents a significant public health burden [1]. OSA causes intermittent hypoxia, oxidative stress, and autonomic dysregulation, leading to cardiovascular morbidity, metabolic dysfunction, and impaired quality of life apart from sleep fragmentation [2,3]. The pathophysiological mechanism expands beyond nocturnal symptoms to significantly impact daytime functional capacity due to recurrent episodes of upper airway obstruction that lead to intermittent hypoxemia, hypercapnia, and sleep fragmentation [4]. These mechanisms result in sympathetic nervous system activation, endothelial dysfunction, and systemic inflammation, collectively contributing to decreased exercise tolerance and impaired functional status in OSA patients [5,6]. The clinical implications extend beyond subjective symptoms, as exercise capacity is an independent predictor of cardiovascular outcomes and overall mortality in various patient populations [7].

In various cardiopulmonary conditions, the six-minute walk test (6MWT) has been proven for a submaximal assessment of functional exercise capacity testing [8]. Initially used for patients with heart failure and chronic obstructive pulmonary disease, the 6MWT provides practical benefits including cost-effectiveness, safety, simple and correlate with daily living activities. [9]. Compared to the maximal exercise testing, 6MWT though provides submaximal assessment better represent real-world physical demands and correlates with peak oxygen consumption in respiratory diseases [10]. The association between objective exercise capacity and polysomnographic severity indices remains incompletely understood even though OSA patients frequently experience exercise intolerance and fatigue. Various studies examining exercise capacity in OSA used small cohorts of population or have used maximal exercise testing, such as cardiopulmonary exercise testing (CPET), which does not precisely reflect daily functional limitation as experienced by patients [11,12]. Easy availability of the test makes it valuable in settings where CPET is not available.

The relationship between 6MWT performance and OSA severity has been examined in limited research. Previous studies limited by small sample sizes and heterogeneous population studies suggest an association between decreased walking distance and higher Apnea-Hypopnea Index (AHI) [13-16]. Although the Oxygen Desaturation Index (ODI) is recognized as a key

pathophysiological driver in OSA, representing hypoxic burden, its use as a predictor of functional impairment has not been thoroughly explored.

We hypothesized that 6MWT parameters would correlate strongly with OSA severity markers and could serve as accessible functional biomarkers for disease assessment. This study aimed to determine the association between 6MWT performance and polysomnographic severity indices in an OSA cohort, and to develop predictive models for estimating OSA severity from functional exercise parameters.

Materials and Methods

This cross-sectional observational study was conducted at the Department of Pulmonary, Critical Care & Sleep Medicine, All India Institute of Medical Sciences, Bhubaneswar, between July 2023 and January 2025. The study protocol adhered to STROBE guidelines for observational research and received approval from the Institutional Ethics Committee [17].

Consecutive adult patients (≥ 18 years) with newly diagnosed OSA confirmed by level I polysomnography were enrolled. According to the American Academy of Sleep Medicine criteria, OSA was defined as AHI ≥ 5 events/hour with associated symptoms or AHI ≥ 15 events/hour regardless of symptoms [18].

Inclusion criteria

Adults aged ≥ 18 years with newly diagnosed OSA; normal chest radiography; previously diagnosed OSA but not initiated on positive airway pressure (PAP) therapy.

Exclusion criteria

1. Patients who refused to give consent
2. Patients diagnosed with neuromuscular disorders, Acute heart failure, Asthma, COPD, Interstitial Lung Disease (ILD), valvular heart disease, or ischemic heart disease
3. **Contraindications for the 6-MWT** [unstable angina symptoms within a month before the test, resting heart rate > 120 bpm, diastolic blood pressure ≥ 100 mmHg, and/or systolic blood pressure > 180 mmHg].
4. **Unable to perform the tests**, including those who are unable to stand, those who need oxygen support, those with psychosomatic disease, and those with acute arthritis
5. **Medication use** (beta blocker, diuretic, corticosteroid, bronchodilator)

Sample size calculation

The sample size is calculated by using the proportion formula

$$n = (Z^2) (P) (1-P)/d^2, \text{ where,}$$

n=required sample size,

Z Statistic for a level of confidence (For 95% level of significance, $Z = 1.96$)

P = prevalence of OSA in the world (24%)

d = precision or highest tolerable error (10%)

According to the Wisconsin Sleep Cohort [19], a population-based longitudinal research, OSA affects 9% of women and 24% of men on average. So, the prevalence is 24%, and the sample size is 70. We haven't considered the prevalence of OSA in the Indian population because most of the studies included only the urban population, which can underestimate the prevalence of OSA. But, during our study period, we included 124 patients who met our inclusion criteria and were diagnosed with OSA.

Polysomnography

All participants underwent supervised overnight level I polysomnography using standardized montages (Embletta MPR PG with ST+ Proxy, Natus Medical). The designated sleep technologist manually scored sleep stages and respiratory events according to American Academy of Sleep Medicine criteria version 2.6 [18]. Primary polysomnographic indices included: AHI, ODI ($\geq 3\%$ oxygen desaturations per hour), lowest oxygen saturation, and mean nocturnal oxygen saturation. OSA severity was classified as mild (AHI 5-14.9), moderate (AHI 15-29.9), or severe (AHI ≥ 30 events/hour). Obstructive apnea is the absence of airflow for more than 10 seconds with persistent respiratory effort. Here, hypopnea is defined as a 30% or greater decrease in airflow from baseline for more than 10 seconds, resulting in a 3% drop in oxygen saturation or arousal [18]. The AHI score was determined for the total duration of sleep time and is defined as the total number of apnea and hypopnea episodes per hour of sleep. The number of desaturation events per hour of sleep is known as the Desaturation Index (DI). The Oxygen Desaturation Index (ODI) classifies the severity of Obstructive Sleep Apnea (OSA) based on the number of desaturation episodes per hour of sleep, which is not included in AASM guidelines but is used only for research purposes. A desaturation episode is a drop in oxygen saturation of at least 3% for 10 seconds. Generally, ODI is graded as follows: mild (5-15), moderate (15-30), and severe (≥ 30). Some studies also categorize normal as ODI < 5 [20].

In our research study, we used AHI and ODI to categorize OSA severity, but in general, OSA severity is based only on the AHI parameter, as per AASM guidelines.

6-minute walk test protocol

The 6MWT was performed within one week of polysomnography using the American Thoracic Society standardized protocol [8]. Tests were conducted over a 30-meter straight corridor under the supervision of trained pulmonary function technologists. Primary measurements included six-minute walk distance (6MWD), pulse oximetry, heart rate, Borg dyspnea scale before and after test, and heart rate recovery calculated as percentage decline from peak heart rate at 1, 2, and 3 minutes post-exercise.

6-minute walk test definitions

- A "sign of walking intolerance" will be described as pausing during the 6-MWT [8,21,22].
- "Clinically significant desaturation" will be defined as an oxygen saturation differential of >4 points before and after the test [8,21,22]
- Any dyspnea that occurred at the conclusion of the test and registered on the Borg scale between 5 and 10 will be deemed "clinically significant" and classified as walk intolerance [22].
- It will be considered "chronotropic incompetence" if the heart rate is less than 60% of the baseline value at the end of the 6-MWT .

Clinical assessment

Comprehensive demographic data, anthropometric measurements, cephalometry evaluation, Muller's maneuver, medical history, and validated questionnaires were recorded, including Epworth Sleepiness Scale (ESS), STOP-BANG questionnaire, and Berlin Questionnaire. Laboratory investigations like complete blood count(CBC), thyroid function test(TFT), HBA1C (Glycated hemoglobin), random blood sugar (RBS), renal function test(RFT), lipid profile, spirometry, echocardiography, Arterial Blood Gas(ABG) analysis, and pulmonary function testing were performed using standard protocols.

Statistical analysis

Statistical analysis was performed using JASP version 0.19.3. Data distribution was assessed using Shapiro-Wilk tests as shown in *Supplementary Table 1*. Continuous variables are

presented as mean $\pm$ standard deviation or median interquartile range. Categorical variables are expressed as frequencies and percentages.

Spearman's rank correlation coefficient assessed relationships between 6MWT parameters and OSA severity indices. One-way ANOVA compared 6MWD across OSA severity groups, with Games-Howell post-hoc testing for pairwise comparisons. Multiple linear regression models were developed with AHI and ODI as dependent variables. Statistical significance was set at $p < 0.05$, with effect sizes reported using Cohen's d and eta-squared (η^2). Linearity and Homoscedasticity were evaluated by plotting standardized residuals against fitted values and against each predictor (6MWD, Borg post-6MWT, ESS). Normality of residuals was assessed using quantile-quantile (Q-Q) plots (as shown in *Supplementary Figure 1-4*) and the Shapiro-Wilk test of normality on residuals where available. Independence of observations was assumed based on the cross-sectional design with one observation per participant. No substantial violations of regression assumptions were observed.

Results

A total of 124 patients were included in the final analysis (Table 1). The cohort was predominantly male (85.5%) with a mean age of 46.51 ± 11.33 years and a mean BMI of 33.19 ± 6.86 kg/m². Severe OSA was present in 79.8% of patients, of which 24.19% were categorized as having Grade 3 obesity. Hypertension was the most common comorbidity (43.5%), followed by diabetes mellitus (25.8%). Most common clinical symptoms were snoring (99.19%), followed by excessive daytime sleepiness (91.13%), restless leg syndrome (50.81%), dozing while driving (45.97%), observed apnea (44.36%), insomnia (29.84%), and parasomnia (5.65%). While both genders exhibited a high prevalence of snoring, a striking difference was observed in "Dozing While Driving," reported by 53.77% of males but none of the females. Restless Leg Syndrome (RLS) was more prevalent in females (77.78%) compared to males (46.23%), indicating a possible gender-specific association with this condition. Obesity Hypoventilation Syndrome (OHS) was notably higher in females (27.78%) than males (10.37%). A higher percentage of females (83.33%) showed abnormal cephalometry findings compared to males (62.26%), which could indicate differences in craniofacial structure that contribute to OSA.

Mean AHI was 52.26 ± 23.69 events/hour, and mean ODI was 54.04 ± 29.22 events/hour. 82% of males and 66% of females have severe OSA as classified by AHI. However, 75% males and 72% females are classified as severe OSA based on ODI. Interestingly, 2.83% of males

were classified as usual, and males were classified as normal based on ODI classification. Males had a higher mean AHI (53.59 ± 23.59) than females (44.46 ± 23.37). Similarly, the mean minimum nocturnal oxygen saturation was slightly lower in males ($71.41\% \pm 13.14$) compared to females ($75.11\% \pm 12.13$).

The total mean hemoglobin level was 13.50 ± 1.57 g/dL, the mean TSH level was 4.08 ± 8.98 , the mean HbA1c level was $6.11 \pm 1.31\%$, and the mean random blood sugar level was 122.55 ± 52.54 mg/dL. LDL cholesterol levels were higher in the moderate and severe OSA groups than in the mild OSA group, with the mean LDL level being 109.52 ± 29.60 mg/dL. Thyroid hormone levels, blood sugar levels and lipid profile levels increase hormone levels, blood sugar levels, and lipid profile levels increase in severe OSA compared to Mild OSA. FEV₁ (Forced Expiratory Volume in 1 second) and FVC (Forced Vital Capacity) showed a slight decrease in the moderate and severe OSA groups compared to the mild OSA group, with the total mean FEV₁ and FVC being 2.37 ± 0.59 L and 2.81 ± 0.67 L, respectively. The mean PaCO₂ (partial pressure of carbon dioxide in arterial blood) was 39.37 ± 4.44 , and the mean HCO₃⁻ (bicarbonate) was 25.31 ± 2.25 .

Six-minute Walk test parameters variations across OSA severity groups as shown in Table 2. Mean 6MWD declined significantly across severity groups (Figure 1): mild OSA 420.85 ± 17.40 meters, moderate OSA 396.99 ± 9.60 meters, and severe OSA 348.61 ± 22.01 meters ($p < 0.001$). Exercise-induced oxygen desaturation $\geq 4\%$ occurred in 36.4% of severe OSA patients compared to none in the mild or moderate groups. Post-exercise Borg dyspnea scores increased progressively with OSA severity: mild 1.0 ± 0.0 , moderate 2.65 ± 1.17 , severe 4.50 ± 1.75 ($p < 0.001$). The change in HRR from 1 to 2 minutes is consistent across the groups. However, the change in HRR from 1 to 3 minutes decreases with increasing OSA severity, further supporting the observation that severe OSA may be associated with a reduced ability to recover heart rate over time.

Strong correlations were observed between 6MWT parameters and polysomnographic indices (Table 3 and *Supplementary Table 2*). The strongest correlation was found between AHI and 6MWD ($\rho = -0.974$, $p < 0.001$) as shown in Figure 2. ODI demonstrated a strong negative correlation with 6MWD ($\rho = -0.775$, $p < 0.001$). Post-exercise Borg dyspnea scores showed strong positive correlations with both AHI ($\rho = 0.765$, $p < 0.001$) and ODI ($\rho = 0.697$, $p < 0.001$). Exercise-induced desaturation correlated significantly with AHI ($\rho = 0.560$, $p < 0.001$) and ODI ($\rho = 0.557$, $p < 0.001$).

A one-way ANOVA revealed statistically significant differences in 6MWD between AHI-based OSA severity groups, $F(2,121) = 138.45$, $p < 0.001$ (*Supplementary Table 3*). The effect size was large ($\eta^2 = 0.563$), indicating that OSA severity accounted for 56.3% of the variance in 6MWD. Games-Howell post-hoc analysis showed all pairwise comparisons were statistically significant (*Supplementary Table 4*): mild vs moderate OSA (mean difference = 23.86 meters, $p = 0.014$, Cohen's $d = 1.16$), mild vs severe OSA (mean difference = 72.24 meters, $p < 0.001$, Cohen's $d = 3.52$), and moderate vs severe OSA (mean difference = 48.38 meters, $p < 0.001$, Cohen's $d = 2.36$).

Two predictive models were developed to estimate OSA severity indices using 6MWT-derived parameters as shown in *Supplementary Tables 5-7*. The AHI prediction model: **AHI = 258.65 + (0.58 × ESS) + (1.89 × Borg Dyspnea post-6MWT) - (0.61 × 6MWD)** explained 89.1% of variance in AHI (Adjusted $R^2 = 0.889$, $F = 325.84$, $p < 0.001$). The ODI prediction model: **ODI = 207.88 + (0.96 × ESS) + (4.21 × Borg Dyspnea post-6MWT) - (0.51 × 6MWD)** explained 65.4% of variance in ODI (Adjusted $R^2 = 0.645$, $F = 74.999$, $p < 0.001$). Both models met statistical assumptions with no evidence of multicollinearity.

Discussion

This study demonstrates a remarkably strong correlation between the six-minute walk test and OSA severity, with 6MWD showing correlations of $\rho = -0.974$ with AHI and $\rho = -0.775$ with ODI. These results align with emerging evidence suggesting that OSA-related intermittent hypoxia, autonomic dysfunction, and systemic inflammation contribute to impaired functional capacity [1,23,24]. This is the strongest correlation reported compared with previous literature, and it extends earlier observations by providing regression equations that can predict OSA severity obtained from functional exercise capacity.

The strong negative correlation between 6MWD and AHI ($\rho = -0.974$, $p < 0.001$) suggests that the 6MWT may be a practical, low-cost biomarker for OSA severity. This finding is consistent with prior studies reporting reduced exercise tolerance in OSA patients [14-16,25]. However, our study extends previous work by demonstrating that the 6MWT, a submaximal test, effectively captures functional limitations correlating with nocturnal hypoxic burden. Unlike maximal cardiopulmonary exercise testing (CPET), which may not reflect real-world physical demands [6,26]. The 6MWT provides a clinically relevant assessment of functional impairment in OSA.

The progressive decline in 6MWD across OSA severity groups (mild: 420.85 ± 17.40 m; moderate: 396.99 ± 9.60 m; severe: 348.61 ± 22.01 m; $p < 0.001$) reinforces the concept that OSA-related physiological disturbances—such as oxidative stress, endothelial dysfunction, and sympathetic overactivation—cumulatively impair physical performance [27,28]. Notably, exercise-induced oxygen desaturation ($\geq 4\%$) was exclusively observed in severe OSA (36.4% of cases), suggesting that nocturnal hypoxemia may contribute to exertional desaturation, further limiting functional capacity [29,30].

In recent years, various pathophysiological mechanisms have been recognized in the relationship between OSA and exercise capacity, which reflects our results accordingly. The hallmark of OSA is intermittent hypoxia that leads to oxidative stress, mitochondrial dysfunction, and altered skeletal metabolism, reducing aerobic capacity and early fatigue during exercise [31,32]. Ultimately, these cellular-level changes manifest clinically as decreased exercise capacity, as shown by our 6MWT results.

The clinically meaningful difference in value of 6MWD is typically 25-35 meters, as shown in other respiratory conditions (COPD) [33]. The 72.24-meter difference in 6MWD between mild and severe OSA groups, as shown in our post hoc analysis result, has clinically meaningful functional impairment affecting daily activities and quality of life. The substantial effect sizes observed (Cohen's d up to 3.52) confirm that significant values are seen in statistical analysis and have clinical relevance in day-to-day practice.

The regression models obtained have significant clinical utility. The AHI prediction model ($R^2 = 0.891$) easily screens for severe OSA in resource-limited settings where polysomnography is unavailable. This model uses simple, easily obtainable parameters like ESS score, 6MWD, and post-exercise Borg dyspnea scale, which is similar to the ODI prediction model ($R^2 = 0.654$). Our ODI finding strongly correlates with functional capacity ($\rho = -0.775$), supporting the growing evidence of hypoxic burden as a key pathophysiologic driver of OSA [34]. The ODI prediction model is more critical because oxygen desaturation correlates more with cardiovascular outcomes than AHI alone, explaining that functional impairment may better reflect the clinically relevant hypoxic burden than looking at the traditional hypopnea events in manual scoring.

Heart rate recovery (HRR) and post-exercise dyspnea (Borg scale) exhibited significant associations with AHI and ODI, indicating autonomic dysregulation in severe OSA. The attenuated HRR in severe OSA patients aligns with prior studies demonstrating impaired parasympathetic reactivation following exercise in OSA [29,35]. These findings suggest that

autonomic dysfunction, driven by recurrent hypoxia and sleep fragmentation, may contribute to reduced exercise tolerance [4,27].

Our correlation findings are stronger than the last reported literature, potentially reflecting the several methodological advantages and use of numerous comprehensive statistical analysis tools such as ANOVA, Games-Howell post hoc testing, and multiple linear regression analysis that decreases the variance heterogeneity. Our study population is newly diagnosed, treatment naïve, and without significant comorbidities that could confound the exercise capacities; it represents a more homogenous study population than previous studies. Previous studies by Alameri et al. and Plywaczewski et al. showed a moderately negative correlation ($r=-0.3$ to -0.5) because the study population was small, heterogeneous, with multiple comorbidities, and a few cases were on treatment with positive airway pressure (PAP) therapy [13,14].

The ANOVA test results showed differences in each OSA severity group using AHI classification, with 56.3% variance in 6MWD. Furthermore, the Games-Howell post-hoc test addresses the unequal variances and sample size, ensuring robust statistical inference. Post hoc analysis test showed a significant difference in 6MWD value between the mild and severe OSA groups by 72.24 meters, with Cohen's d test of 3.52, indicating large effects that support our results have clinically significant observed differences.

Findings of our study suggest that 6MWT can be incorporated in day-to-day practice for various reasons. First, it can be used as a functional assessment of a suspected OSA patient that complements the polysomnographic data in quantifying the real-world impact and providing a comprehensive disease burden. Second, exercise capacity gives prognostic evaluation beyond AHI of any patient that can independently predict risk and mortality in cardiovascular disease. Third, 6MWD results help prescribe exercise training and pulmonary rehabilitation strategies. Fourth, we can objectively monitor the treatment response of OSA patients after PAP therapy and non-PAP therapy treatment, such as lifestyle modification, sleep hygiene, weight reduction, and myofunctional therapy. Lastly, the derived equation can help in screening OSA patients in the outpatient department daily, where there is an unavailability of a sleep laboratory, or in any affordability issue for sleep testing.

Despite robust findings, several limitations must be acknowledged. Firstly, the cross-sectional design precludes causal inferences. Secondly, the predominance of male participants (85.5%) may limit generalizability to female OSA populations. Thirdly, while we excluded cardiopulmonary comorbidities, subclinical cardiovascular dysfunction in OSA patients may still influence 6MWT performance. Future studies incorporating CPET and advanced

hemodynamic monitoring could further elucidate the mechanisms linking OSA to exercise intolerance.

Future research should focus on an extensive multicentric study to validate the predictive formulas, longitudinal studies assessing effects of therapy on 6MWT parameters, wearable devices to capture real-time exercise and desaturation data, and combining 6MWT metrics with machine learning for enhanced predictive models.

Key clinical applications

Screening tool: Regression equations help in the estimation of OSA severity in primary care and resource-limited settings where polysomnography is unavailable

Functional assessment: 6MWT gives an actual functional assessment of OSA patients beyond traditional sleep metrics(AHI, ODI), providing a comprehensive disease assessment.

Treatment monitoring: Following OSA therapy, therapeutic response and functional improvement can be measured objectively.

Risk stratification: Exercise capacity provides cardiovascular risk assessment that complements traditional OSA severity indices.

Conclusions

This study demonstrated that 6MWT parameters strongly correlate with OSA severity, providing a clinically accessible tool for functional assessment. This study showed a strong association between AHI and ODI with functional impairments observed in the Six-Minute Walk Test (6MWT), including reduced 6MWD, lower oxygen saturation, delayed heart rate recovery at 3 minutes and heightened post-exercise dyspnea. Using correlation and regression models, key predictors such as Borg dyspnea score post-6MWT, ESS score, and 6MWD were found to help determine the severity of OSA effectively in resource-limited settings. The predictive models developed herein offer a potential alternative for OSA severity estimation in resource-constrained settings. Given the prognostic significance of exercise capacity in cardiovascular health, integrating 6MWT into OSA evaluation may enhance risk stratification and therapeutic monitoring. Further research should explore the impact of continuous positive airway pressure (CPAP) therapy on 6MWT performance and validate these findings in diverse populations.

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Online supplementary material

Supplementary Table 1. Shapiro-Wilk test for bivariate normality.

Supplementary Table 2. Correlation between Apnea Hypopnea Index, Oxygen Desaturation Index and 6-minute walk distance.

Supplementary Table 3. One-way analysis of variance: comparison of 6-minute walk distance by obstructive sleep apnea severity (Apnea-Hypopnea Index classification).

Supplementary Table 4. Games-Howell post-hoc comparisons: Apnea-Hypopnea Index groups.

Supplementary Table 5. Multiple linear regression analysis for OSA severity prediction.

Supplementary Table 6. Multiple regression analysis (dependent variable: Apnea-Hypopnea Index).

Supplementary Table 7. Multiple Regression analysis (dependent variable: Oxygen Desaturation Index).

Supplementary Figure 1. Q-Q plot for Apnea-Hypopnea Index regression analysis model.

Supplementary Figure 2. Q-Q plot for homogeneity testing in the regression analysis model.

Supplementary Figure 3. Descriptive plot for 6-Minute Walk Distance and Apnea-Hypopnea Index in analysis of variance testing.

Supplementary Figure 4. Q-Q plot for homogeneity testing in analysis of variance testing.

Table 1. Baseline patient characteristics.

Characteristic	All patients (N=124)
Demographics	
Age (years), mean $\pm$ SD	46.51 $\pm$ 11.33
Male sex, n (%)	106 (85.5)
BMI (kg/m ²), mean $\pm$ SD	33.19 $\pm$ 6.86
Neck circumference (cm), mean $\pm$ SD	39.96 $\pm$ 2.99
Clinical Questionnaires	
ESS score, mean $\pm$ SD	12.25 $\pm$ 5.09
STOP-BANG score, mean $\pm$ SD	4.77 $\pm$ 1.22
Berlin criteria positive, n (%)	120 (96.8)
Comorbidities, n (%)	
Hypertension	54 (43.5)
Diabetes mellitus	32 (25.8)
Hypothyroidism	13 (10.5)
Sleep Study Parameters	
AHI (events/hour), mean $\pm$ SD	52.26 $\pm$ 23.69
ODI (events/hour), mean $\pm$ SD	54.04 $\pm$ 29.22
Lowest SpO ₂ (%), mean $\pm$ SD	71.94 $\pm$ 13.02
Mean nocturnal SpO ₂ (%), mean $\pm$ SD	92.33 $\pm$ 4.14
OSA Severity by AHI, n (%)	
Mild (5-14.9 events/hour)	8 (6.5)
Moderate (15-29.9 events/hour)	17 (13.7)
Severe ($\geq$ 30 events/hour)	99 (79.8)

N, number; SD, standard deviation; BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, Apnea-Hypopnea Index; ODI, Oxygen Desaturation Index; OSA, obstructive sleep apnea.

Table 2. 6-minute walk test results by OSA severity.

Parameter	Mild OSA (N=8)	Moderate OSA (N=17)	Severe OSA (N=99)	p
Exercise performance				
6MWD (meters), mean $\pm$ SD	420.85 $\pm$ 17.40	396.99 $\pm$ 9.60	348.61 $\pm$ 22.01	<0.001
Maximum heart rate (bpm), mean $\pm$ SD	108.38 $\pm$ 12.55	119.47 $\pm$ 11.16	124.89 $\pm$ 11.29	0.028
Post-exercise measurements				
End SpO ₂ (%), mean $\pm$ SD	97.75 $\pm$ 0.89	96.77 $\pm$ 0.66	94.96 $\pm$ 2.65	<0.001
SpO ₂ desaturation (%), mean $\pm$ SD	0.88 $\pm$ 0.64	1.58 $\pm$ 0.86	2.97 $\pm$ 2.01	<0.001
Exercise desaturation >4%, N (%)	0 (0)	0 (0)	36 (36.4)	<0.001
Borg dyspnea scale, mean $\pm$ SD	1.0 $\pm$ 0.0	2.65 $\pm$ 1.17	4.50 $\pm$ 1.75	<0.001
Heart rate recovery				
HRR at 3 minutes (%), mean $\pm$ SD	22.25 $\pm$ 7.23	21.00 $\pm$ 5.24	18.53 $\pm$ 7.96	0.168

Nm number; SD standard deviation; 6MWD, 6-minute walk distance; HRR, heart rate recovery; SpO₂, oxygen saturation, bpm, beats per minute.

Table 3. Correlation analysis: 6MWT parameters and clinical variables.

Variable	6MWD (meters)	Post-6MWT Borg Scale	HRR at 3 minutes (%)
Polysomnographic parameters			
AHI (events/hour)	-0.974 (<0.001)	0.765 (<0.001)	-0.297 (<0.001)
ODI (events/hour)	-0.775 (<0.001)	0.697 (<0.001)	-0.240 (0.007)
Lowest SpO ₂ (%)	0.642 (<0.001)	-0.548 (<0.001)	0.218 (0.016)
Mean nocturnal SpO ₂ (%)	0.521 (<0.001)	-0.421 (<0.001)	0.164 (0.071)
Clinical parameters			
Age (years)	-0.268 (0.003)	0.189 (0.036)	-0.142 (0.124)
BMI (kg/m ²)	-0.412 (<0.001)	0.356 (<0.001)	-0.156 (0.087)
ESS score	-0.324 (<0.001)	0.284 (0.001)	-0.198 (0.026)

Values represent Spearman correlation coefficients (p-values). 6MWD, 6-minute walk distance; 6MWT, 6-minute walk test; HRR, heart rate recovery; AHI, Apnea-Hypopnea Index; ODI, Oxygen Desaturation Index; BMI, body mass index; ESS, Epworth Sleepiness Scale; SpO₂, oxygen saturation.

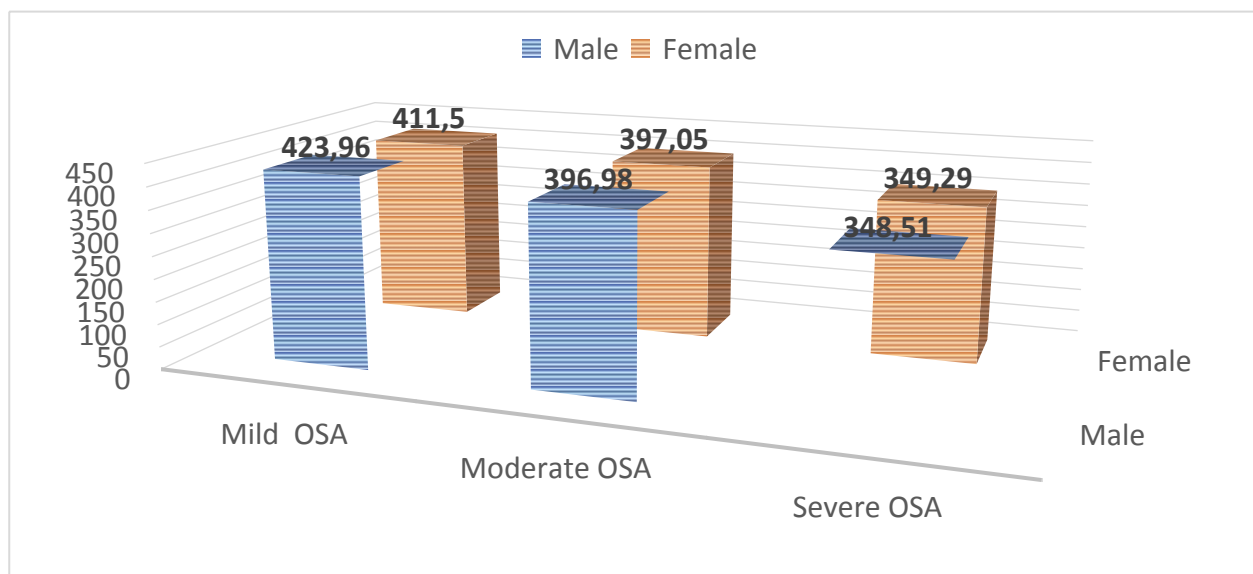


Figure 1: Bar diagram comparison of six-minute walk distance across OSA severity groups classified by AHI and sex. Bar diagram demonstrating clear separation between, OSA severity groups in both sexes; AHI, Apnea Hypopnea Index.

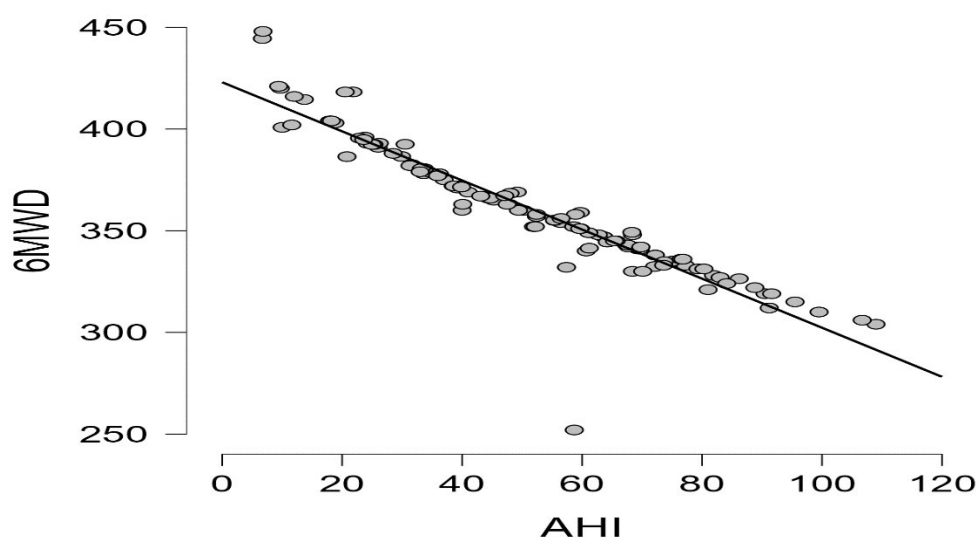


Figure 2: Correlation between six-minute walk distance and Apnea-Hypopnea in in OSA patients. Scatter plot showing the strong negative correlation between six-minute walk distance and AHI across different OSA severity groups($\rho = -0.974$, $p < 0.001$).