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Original Article

Clinical Characteristics of Moderate-to-Severe Obstructive Sleep Apnea: A Cross-sectional Analysis of 12,715 Adults from the TURKAPNE Cohort

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ABSTRACT

OBJECTIVE: Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder associated with cardiometabolic morbidity. However, its clinical presentation is heterogeneous, and subjective sleepiness does not consistently reflect disease severity. We aimed to describe the clinical and polysomnographic characteristics of moderate-to-severe OSA and to identify factors independently associated with disease severity in a large nationwide sleep clinic cohort.

MATERIAL AND METHODS: This cross-sectional study was conducted within the Turkish Sleep Apnea Database cohort and included 12,715 adults with complete baseline clinical and polysomnographic data from 34 sleep centers. Moderate-to-severe OSA was defined as an apnea–hypopnea index (AHI) ≥15 events/h. Demographic and anthropometric variables, sleep-related symptoms, Epworth Sleepiness Scale (ESS) scores, polysomnographic parameters, and comorbidities were analyzed. Independent factors associated with moderate-to-severe OSA were identified using multivariable logistic regression.

RESULTS: Overall, 8,393 patients (66.0%) had moderate-to-severe OSA and 4,322 (34.0%) had no or mild OSA (AHI <15 events/h). Patients with moderate-to-severe OSA were older, more frequently male, and had a higher body mass index (BMI) (all $P < 0.001$). Although excessive daytime sleepiness (ESS ≥11) was more common (25.8% vs. 17.7%, $P < 0.001$), ESS score was not independently associated with disease severity. Increasing age, male sex, higher BMI, snoring, witnessed apneas, and nocturnal dyspnea remained independent associates of moderate-to-severe OSA.

CONCLUSION: In this large nationwide sleep clinic cohort, objective risk factors and classic nocturnal symptoms were more informative than subjective sleepiness in identifying clinically significant OSA and support a risk-based approach in routine pulmonary practice.

KEYWORDS: Insomnia, sleep apnea, comisa, polysomnography

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INTRODUCTION

Obstructive sleep apnea (OSA) is a common chronic sleep-related breathing disorder characterized by recurrent upper airway obstruction during sleep, leading to intermittent hypoxemia and sleep fragmentation. Global prevalence estimates indicate that nearly one billion adults have OSA, with approximately 400–450 million affected by moderate-to-severe disease [apnea–hypopnea index (AHI) ≥15 events/h], underscoring its major public health impact.[1-3]

Moderate-to-severe OSA is strongly associated with adverse cardiometabolic outcomes. Large observational and longitudinal studies have demonstrated robust associations between OSA and hypertension (HT), coronary artery disease (CAD), heart failure (HF), diabetes mellitus (DM), and increased cardiovascular morbidity and mortality.[4-7] These associations are thought to be mediated by sympathetic nervous system activation, oxidative stress, endothelial dysfunction, metabolic dysregulation, and systemic inflammation driven by intermittent hypoxia and sleep disruption.[8,9] In addition, OSA is linked to impaired daytime functioning, neurocognitive complaints, reduced quality of life, and increased risk of motor vehicle and occupational accidents.[10-12]

Despite these well-established consequences, the clinical presentation of OSA is heterogeneous. While loud snoring, witnessed apneas, and excessive daytime sleepiness (EDS) are considered classic features, symptom burden does not consistently parallel objective disease severity.[10] EDS is commonly assessed using the Epworth Sleepiness Scale (ESS), a validated and widely used questionnaire.[13] However, a substantial proportion of patients with moderate-to-severe OSA report minimal subjective sleepiness, whereas some individuals with lower AHI values experience marked daytime impairment.[10,14]

Demographic and anthropometric factors remain among the most consistent clinical correlates of OSA severity. Increasing age, male sex, obesity, and central adiposity—often reflected by body mass index (BMI) and neck circumference—are strongly associated with moderate-to-severe OSA across populations.[2,3,15,16] Nevertheless, the relative contribution of symptoms, anthropometrics, and comorbidities may vary by population and referral setting.

Using data from a nationwide sleep clinic cohort, the present study aimed to characterize demographic, anthropometric, polysomnographic, symptomatic, and comorbidity profiles associated with moderate-to-severe OSA and to identify clinical factors independently associated with this disease severity.

Main Points

- In a large nationwide cohort of over 12,000 sleep clinic patients, two-thirds were diagnosed with moderate-to-severe obstructive sleep apnea (OSA), highlighting the substantial burden of clinically significant disease in routine practice.
- Objective risk factors—including increasing age, male sex, higher body mass index, and classic nocturnal symptoms (snoring, witnessed apneas, nocturnal dyspnea)—were independently associated with OSA severity.
- Although excessive daytime sleepiness was more prevalent in patients with moderate-to-severe OSA, subjective sleepiness (Epworth Sleepiness Scale score) was not an independent predictor of disease severity.
- These findings support a risk-based, symptom-informed diagnostic approach, emphasizing objective clinical characteristics over subjective sleepiness in the identification of clinically relevant OSA.

MATERIAL AND METHODS

Study Design and Data Source

The present study was conducted as a cross-sectional analysis within the framework of the Turkish Sleep Apnea Database (TURKAPNE), a nationwide, multicenter cohort established to investigate the clinical and polysomnographic characteristics of patients evaluated for OSA in routine clinical practice.[17] Since October 2017, adults referred to participating sleep centers for suspected OSA have been prospectively enrolled.

For the current analysis, records obtained from 34 accredited sleep centers were screened. Only patients with complete demographic information, anthropometric measurements, clinical characteristics, questionnaire data, and full-night polysomnography were included (Figure 1). Individuals with advanced systemic illnesses limiting life expectancy, active or uncontrolled malignancy, alcohol dependence, or prior treatment with positive airway pressure devices or mandibular advancement devices were excluded to avoid confounding.

Ethical Approval and Patient Consent

All procedures were performed in accordance with internationally accepted ethical standards. The study protocol received approval from the Ethics Committee of the Medical Faculty of Marmara University, İstanbul (approval no: 09.2016.311, date: 05.09.2016). Written informed consent was obtained from all participants prior to data collection. The TURKAPNE registry is listed on ClinicalTrials.gov (identifier: NCT02784977).

Registry Structure and Data Management

Clinical data were entered into a dedicated, password-protected electronic registry designed specifically for the TURKAPNE project. Each participating center accessed the system using individualized credentials and entered patient data through standardized electronic case report forms. Data were stored on a secure central server, while personally identifiable information remained locally archived at each center. To ensure data reliability, periodic random audits were conducted by an independent monitoring committee with unrestricted access to the database.

Clinical, Demographic, and Anthropometric Assessments

Baseline demographic characteristics included age and sex. Elderly status was defined as age ≥65 years. Anthropometric assessments comprised BMI, calculated as weight divided by height squared (kg/m^2), obesity was defined as a BMI ≥30 kg/m^2.

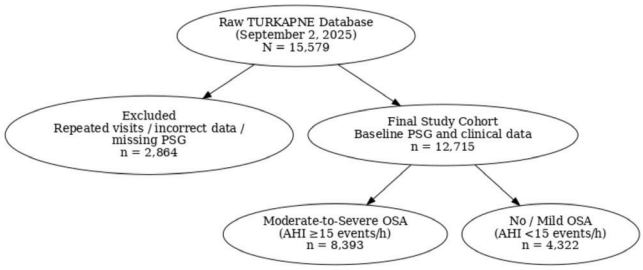

Figure 1. Flow chart of the participants

OSA: obstructive sleep apnea, PSG: polysomnography, AHI: apnea–hypopnea index

Clinical symptom variables were derived from standardized patient-reported items recorded in the registry and included sleep-related complaints (difficulty initiating sleep, difficulty maintaining sleep, subjective sleep latency ≥30 minutes, total sleep time <6 hours, and reduced sleep efficiency), nocturnal symptoms (snoring, witnessed apneas, nocturnal dyspnea, nocturia, night sweats, and morning headache), and daytime outcomes (daytime sleepiness, daytime tiredness, fatigue, impaired concentration, and history of sleep-related accidents).

Comorbid conditions were identified based on physician-reported diagnoses and/or ongoing medical treatment, and categorized as cardiometabolic (HT or antihypertensive medication use, DM, hyperlipidemia, CAD, and HF); respiratory (chronic obstructive pulmonary disease and asthma); neuropsychiatric (depression, restless legs syndrome, epilepsy, and other psychiatric disorders); endocrine (hypothyroidism); cerebrovascular disease (prior stroke or transient ischemic attack); and malignancy. Concomitant medication use, including antihypertensive, antidiabetic, lipid-lowering, antiepileptic, and immunosuppressive therapies, was also recorded and considered in the analyses.

Assessment of Sleep-related Symptoms and Daytime Function

Sleep-related symptoms were evaluated using standardized patient-reported items embedded in the TURKAPNE registry. These included habitual snoring, witnessed apneas, nocturnal dyspnea, nocturia, night sweats, morning headache, difficulties with sleep initiation or maintenance, prolonged subjective sleep latency (≥30 minutes), short sleep duration (<6 hours), impaired concentration, and history of sleep-related accidents.

Daytime sleepiness was quantified using the validated Turkish version of the ESS. The ESS yields a total score between 0 and 24, with higher scores indicating greater daytime sleep propensity. EDS was defined as an ESS score of 11 or higher.[13]

Polysomnographic Evaluation and Obstructive Sleep Apnea Classification

All participants underwent attended overnight polysomnography with a minimum recording duration of seven hours. Subjects were instructed to refrain from alcohol, caffeine, and sedative agents on the day of the study. Standard recordings included electroencephalography, electrooculography, electromyography, electrocardiography, airflow measurements, respiratory effort, oxygen saturation, snoring, and body position.

Sleep staging and respiratory event scoring were performed according to contemporary international criteria.[18] Apnea was defined as a reduction in airflow of at least 90% lasting 10 seconds or longer, while hypopnea was defined as a reduction in airflow of at least 30% lasting 10 seconds or longer, accompanied by either oxygen desaturation of ≥3% or an arousal.

The AHI was calculated as the number of respiratory events per hour of sleep. OSA was defined as an AHI ≥5 events/h. For the purposes of this study, patients were categorized as having moderate-to-severe OSA if AHI was ≥15 events/h. Severe OSA was defined as an AHI ≥30 events/h. Additional polysomnographic parameters included sleep architecture, arousal index, oxygen desaturation index, minimum and mean oxygen saturation, time spent with oxygen saturation below 90%, rapid eye movement (REM)-related AHI, and supine AHI.

Statistical Analysis

Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as counts and percentages. The normality of continuous variables was assessed visually and statistically. Between-group comparisons by OSA severity (moderate-severe vs. non-moderate-severe OSA) were performed using the independent-samples t-test for continuous variables and the chi-square test for categorical variables. Polysomnographic parameters, sleep architecture variables, sleep-related symptoms, daytime outcomes, and comorbid conditions were compared between groups using the same approach. A two-sided *P* value was considered statistically significant. To identify independent predictors of moderate-to-severe OSA, univariable logistic regression analyses were first performed on demographic, anthropometric, sleep-related, and clinical variables. Subsequently, multivariable logistic regression models were constructed using a hierarchical modeling strategy. Results of logistic regression analyses were reported as odds ratios with 95% confidence intervals. All analyses were conducted using a standard statistical software package, and statistical significance was defined as a two-tailed *P* value <0.05.

RESULTS

Study Population

As shown in Figure 1, 15,579 individuals were screened from the TURKAPNE database. After exclusion of repeated visits, incorrectly entered records, and missing polysomnographic data (n = 2,864), the final study cohort comprised 12,715 adults with complete baseline clinical and polysomnographic information. Of these, 8,393 patients (66.0%) had moderate-to-severe OSA (AHI ≥15 events/h), while 4,322 (34.0%) had no or mild OSA.

Demographic and Anthropometric Characteristics

Patients with moderate-to-severe OSA were older, more likely to be male, and had a higher prevalence of obesity and greater central and peripheral adiposity compared with those without moderate-to-severe OSA (all $P < 0.001$; Table 1).

Polysomnographic Characteristics and Sleep-related Symptoms

Moderate-to-severe OSA was associated with a substantially higher respiratory event burden, more pronounced nocturnal hypoxemia, a higher arousal index, and altered sleep architecture, characterized by a higher proportion of N1 sleep and lower proportions of N3 and REM sleep (all $P < 0.001$; Table 2). Several nocturnal and daytime symptoms, including snoring, witnessed apneas, nocturnal dyspnea, daytime sleepiness, fatigue, and sleep-related accidents, were more prevalent in

Table 1. Demographic characteristics and anthropometric measures according to OSA severity

Variable	Moderate-to-severe OSA n = 8393	No/mild OSA n = 4322	*P* value
Age, years	51.8±11.7	46.9±12.3	<0.001
Female sex, %	26.7	40.9	<0.001
Elderly (≥65 years), %	14.2	7.8	<0.001
BMI, kg/m^2	32.7±6.0	29.6±5.6	<0.001
Obesity (BMI ≥30 kg/m^2), %	63.8	40.7	<0.001
Neck circumference, cm	42.0±5.3	39.3±5.1	<0.001
Waist circumference, cm	111.1±15.4	102.1±13.7	<0.001
Hip circumference, cm	113.1±12.6	109.2±29.9	<0.001

OSA: obstructive sleep apnea, BMI: body mass index

Table 2. Polysomnographic features, sleep architecture, and sleep-related symptoms according to OSA severity

Variable	Moderate-to-severe OSA	No/mild OSA	*P* value
Polysomnographic and sleep architecture parameters			
PSG-AHI	43.5±24.6	7.4±4.1	<0.001
ODI	42.8±44.2	8.0±9.5	<0.001
REM-AHI	43.3±27.0	16.6±26.4	<0.001
Supine AHI	56.9±27.7	15.8±15.0	<0.001
Nadir SpO_2, %	76.4±11.5	85.8±7.3	<0.001
Time with SpO_2 <90%, min	56.4±81.4	22.0±51.6	<0.001
N1 sleep, %	6.9±9.1	4.9±6.7	<0.001
N3 sleep, %	21.6±17.3	25.3±17.6	<0.001
REM sleep, %	13.6±9.1	14.8±8.0	<0.001
Arousal index	26.2±25.5	14.1±15.8	<0.001
Sleep-related symptoms and daytime outcomes			
Difficulty falling asleep	18.4	16.7	0.031
Sleep latency ≥30 min	17.7	24.3	<0.001
Difficulty maintaining sleep	15.4	14.4	0.204
Total sleep time <6 h	24.0	24.2	0.746
Sleep efficiency <85%	51.8	51.0	0.410
Night sweats	27.7	18.0	<0.001
Nocturia	27.6	18.1	<0.001
Morning headache	19.2	13.2	<0.001
Daytime sleepiness	46.8	30.8	<0.001
Daytime tiredness	46.8	33.6	<0.001
Fatigue	46.7	33.6	<0.001
ESS score	6.14±6.25	4.95±5.47	<0.001
ESS ≥11	25.8	17.7	<0.001
Difficulty in concentration	19.6	16.4	<0.001
Sleep-related accident	2.8	1.9	0.002

OSA: obstructive sleep apnea, PSG: polysomnography, AHI, apnea–hypopnea index, ODI: oxygen desaturation index, SpO_2: peripheral oxygen saturation, N1: stage N1 sleep, N3: stage N3 sleep, REM: rapid eye movement, ESS: Epworth Sleepiness Scale

patients with moderate-to-severe OSA. Although ESS scores and the prevalence of EDS (ESS ≥11) were higher in this group, subjective sleepiness showed limited discriminatory value when considered alongside objective disease characteristics (Table 2).

Comorbidities

Patients with moderate-to-severe OSA had a higher prevalence of cardiometabolic comorbidities, including HT, DM, CAD, HF, and chronic obstructive pulmonary disease, as well as depression and restless legs syndrome (all $P < 0.001$; Table 3). No significant between-group differences were observed for arrhythmia, cerebrovascular disease, asthma, hypothyroidism, or malignancy.

Factors Independently Associated with Moderate-to-Severe OSA

In multivariable logistic regression analyses (Table 4, Figure 2), increasing age, male sex, and higher BMI remained strong independent predictors of moderate-to-severe OSA across all models. After full adjustment, snoring, witnessed apneas, and nocturnal dyspnea were independently associated with moderate-to-severe OSA, whereas ESS score and nocturia were not. Cardiometabolic comorbidities did not retain independent associations with disease severity in the fully adjusted model.

DISCUSSION

Principal Findings

In this large, nationwide, multicenter sleep clinic cohort, moderate-to-severe OSA had a distinct clinical profile characterized by older age, male sex, and increased general and central adiposity. These demographic and anthropometric features remained the strongest independent predictors of disease severity, even after comprehensive adjustment for symptoms and comorbidities. In contrast, subjective daytime sleepiness showed limited discriminatory value once objective risk factors and nocturnal symptoms were considered.

Demographic and Anthropometric Correlates of Obstructive Sleep Apnea Severity

The strong associations between advancing age, male sex, and moderate-to-severe OSA observed in the present study are consistent with prior population-based and clinic-based investigations.[2,3,10] Age-related changes in upper airway anatomy, neuromuscular control, and ventilatory stability, together with sex-specific differences in fat distribution and airway collapsibility, are likely to underlie these associations.[2,3,10,19]

Obesity emerged as a central determinant of disease severity, in line with extensive evidence linking increased body mass to upper airway narrowing and collapsibility. Beyond BMI, patients with moderate-to-severe OSA had larger neck, waist, and hip circumferences, underscoring the importance of central and peripheral adiposity. Prior imaging and physiological studies have demonstrated that fat accumulation in cervical and abdominal regions contributes to reduced lung volumes and mechanical loading of the upper airway, thereby exacerbating airway instability during sleep.[20-22] Collectively, these findings reinforce the clinical relevance of anthropometric assessment when evaluating OSA severity in routine practice.

Sleep Architecture, Hypoxemia, and Symptom Burden

Patients with moderate-to-severe OSA demonstrated greater respiratory event burden, more pronounced nocturnal hypoxemia, and increased sleep fragmentation, consistent with earlier polysomnographic studies linking disease severity to disrupted sleep continuity.[23] The observed shift toward lighter sleep stages and relative reduction in slow-wave and REM sleep

Table 3. Cardiometabolic, respiratory, and neuropsychiatric comorbidities and medication use according to OSA severity

Variable	Moderate-to-severe OSA	No/mild OSA	*P* value
Hypertension/antihypertensive use	35.5	23.5	<0.001
Diabetes mellitus	18.0	11.7	<0.001
Hyperlipidemia	6.6	4.2	<0.001
Coronary artery disease	5.2	2.7	<0.001
Heart failure	3.8	1.9	<0.001
COPD	4.5	3.1	<0.001
Arrhythmia	4.2	3.9	0.356
Stroke/TIA	0.8	0.6	0.311
Depression	15.3	12.2	<0.001
Restless legs syndrome	14.7	11.6	<0.001
Psychiatric disorders (overall)	6.1	6.5	0.498
Asthma	8.2	9.1	0.090
Hypothyroidism	4.0	4.6	0.108
Cancer	0.4	0.4	0.752
Immunosuppressive drug use	0.3	0.6	0.028
Antiepileptic drug use	0.9	1.1	0.505

OSA: obstructive sleep apnea, COPD: chronic obstructive pulmonary disease, TIA, transient ischemic attack

aligns with experimental and clinical data showing preferential suppression of restorative sleep in the presence of recurrent respiratory events and arousals.[23,24]

Indices of nocturnal hypoxemia appear to play a particularly important role in shaping the clinical symptom burden. Prior work has suggested that measures such as oxygen desaturation index and cumulative hypoxemic exposure may correlate more closely with daytime impairment than AHI alone.[25] In agreement with this concept, patients with moderate-to-severe OSA in the present study reported higher rates of daytime sleepiness, fatigue, impaired concentration, and sleep-related accidents, supporting the clinical relevance of hypoxemia-driven sleep disruption.[25,26]

Subjective Sleepiness and Disease Severity

Despite higher ESS scores and a greater prevalence of EDS among patients with more severe OSA, subjective sleepiness did not remain independently associated with moderate-to-severe disease after multivariable adjustment. This finding corroborates previous observations that subjective sleepiness alone is an imperfect marker of OSA severity and may be influenced by interindividual differences in arousal threshold, sleep perception, comorbid conditions, and adaptive mechanisms.[25,27] These results highlight the limitations of relying solely on subjective sleepiness when stratifying disease severity and underscore the importance of integrating objective risk factors with nocturnal symptom profiles.

Comorbidities and Obstructive Sleep Apnea Severity

Moderate-to-severe OSA was associated with a higher prevalence of cardiometabolic comorbidities, including HT, DM, CAD, and HF, consistent with extensive epidemiological evidence linking OSA severity to cardiometabolic disease burden.[4-7,9] The increased prevalence of chronic obstructive pulmonary disease among patients with more severe OSA aligns with prior reports describing the overlap syndrome and its association with more severe nocturnal hypoxemia and adverse cardiopulmonary outcomes.[28]

However, most comorbid conditions did not retain independent associations with disease severity in fully adjusted models,

Table 4. Multivariable logistic regression analysis of factors associated with obstructive sleep apnea across sequentially adjusted models

	OR	95% CI	*P* value
Model 1. Demographic and anthropometric variables			
Age (per year)	1.041	1.038–1.045	<0.001
Male sex	3.196	2.915–3.505	<0.001
BMI (kg/m^2)	1.112	1.104–1.121	<0.001
Model 2. Model 1 + sleep-related symptoms and ESS			
Age (per year)	1.045	1.040–1.049	<0.001
Male sex	3.023	2.718–3.362	<0.001
BMI (kg/m^2)	1.104	1.094–1.114	<0.001
Snoring	2.355	2.120–2.617	<0.001
Witnessed apneas	1.865	1.645–2.114	<0.001
Nocturnal dyspnea	1.258	1.103–1.435	<0.001
Nocturia	0.992	0.881–1.118	0.897
ESS score	1.007	0.999–1.015	0.097
Model 3. Fully adjusted model			
Age (per year)	1.045	1.040–1.050	<0.001
Male sex	3.037	2.729–3.379	<0.001
BMI (kg/m^2)	1.104	1.094–1.114	<0.001
Snoring	2.353	2.118–2.615	<0.001
Witnessed apneas	1.863	1.643–2.113	<0.001
Nocturnal dyspnea	1.260	1.104–1.437	<0.001
Nocturia	0.994	0.882–1.121	0.928
ESS score	1.007	0.999–1.015	0.096
COPD	0.871	0.676–1.123	0.287
Coronary artery disease	0.962	0.741–1.249	0.770
Hypertension/antihypertensive use	1.003	0.892–1.127	0.964
Diabetes mellitus	1.018	0.885–1.172	0.799

AHI: apnea–hypopnea index, BMI: body mass index, CI: confidence interval, COPD: chronic obstructive pulmonary disease, ESS: Epworth Sleepiness Scale, OR: odds ratio, OSA: obstructive sleep apnea

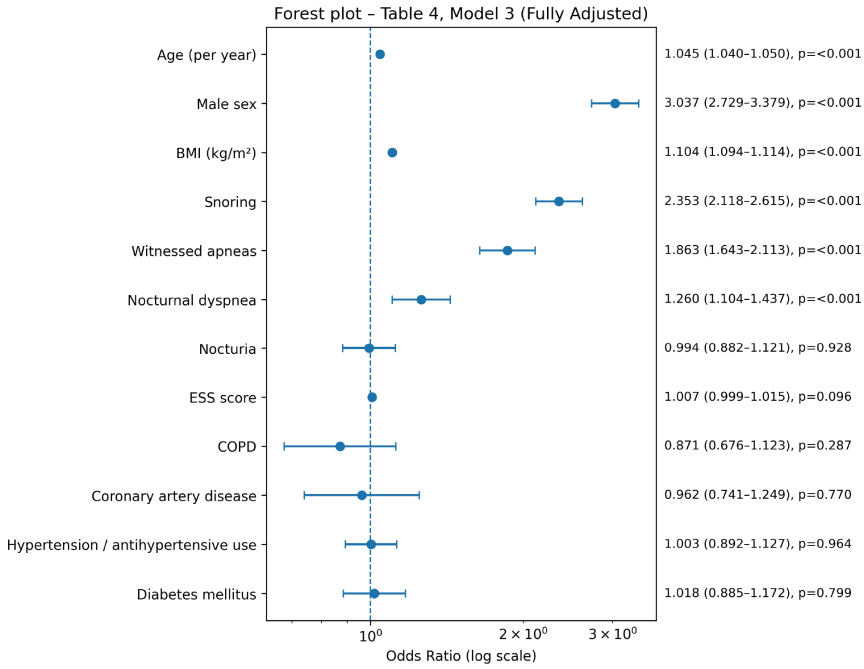

Figure 2. Multivariable regression analysis of the parameters related to moderate-to-severe OSA

OSA: obstructive sleep apnea, BMI: body mass index, ESS: Epworth Sleepiness Scale, COPD: chronic obstructive pulmonary disease

suggesting that their observed relationships with OSA may be largely mediated through shared risk factors such as age, sex, and obesity. With respect to neuropsychiatric conditions, the higher prevalence of depression and restless legs syndrome is consistent with earlier reports,[29-31] whereas the absence of differences in overall psychiatric morbidity underscores the heterogeneity of neuropsychiatric manifestations in OSA.

Clinical Implications

The present findings have important implications for clinical practice. Classic nocturnal symptoms, together with demographic and anthropometric risk factors, provided greater discriminatory value for identifying moderate-to-severe OSA than subjective sleepiness alone did. These results support a comprehensive, risk-based approach to OSA assessment that integrates objective clinical characteristics with symptom profiles, rather than reliance on daytime sleepiness as a primary indicator of disease severity.

Strengths and Limitations

Key strengths of this study include the large sample size, nationwide multicenter design, and standardized data collection within a well-established registry. Several limitations merit consideration. The cross-sectional design precludes causal inference, and the sleep clinic–based population may limit generalizability to community settings. Symptom reporting and comorbidity data were based on patient report and physician documentation, introducing potential misclassification. Residual confounding by unmeasured factors cannot be excluded, and the lack of longitudinal follow-up precludes assessment of disease progression or treatment effects.

CONCLUSION

In this large multicenter sleep clinic cohort, moderate-to-severe OSA was associated with a distinct demographic, anthropometric, and clinical profile characterized by older age, male sex, greater adiposity, more severe nocturnal hypoxemia, and increased sleep fragmentation. While subjective sleepiness was more prevalent in patients with more severe disease, objective risk factors and classic nocturnal symptoms demonstrated greater discriminatory value. These findings emphasize the importance of integrating demographic, anthropometric, and symptom-based information for the identification and risk stratification of clinically significant OSA in real-world practice.

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Ethics

Ethics Committee Approval: All procedures were performed in accordance with internationally accepted ethical standards. The study protocol received approval from the Ethics Committee of the Medical Faculty of Marmara University, İstanbul (approval no: 09.2016.311, date: 05.09.2016).

Informed Consent: Written informed consent was obtained from all participants prior to data collection.

Footnotes

Authorship Contributions

Concept: Y.P., Ö.K.B., Design: Y.P., Ö.K.B., Data Collection or Processing: B.B., A.P., E.K., M.S.T., Ö.K.B., Ş.A., A.Ç., N.D., N.A.Y., Y.P., TURKAPNE Study Group, Analysis or Interpretation: B.B., Literature Search: B.B., Writing: B.B.

Conflict of Interest: Metin Akgün, MD, serves as Editor-in-Chief of Thoracic Research and Practice. He is also a member of the TURKAPNE Study Group and contributed to data collection for this study. In line with the journal's editorial conflict of interest policy, he was fully excluded from all editorial and peer review processes related to this manuscript. He had no access to reviewer identities, reviewer reports, editorial evaluations, or the final decision-making process.

Canan Gündüz Gürkan, MD, serves as an Editor of Thoracic Research and Practice. She is also a member of the TURKAPNE Study Group and contributed to data collection for this study. She was not involved in any stage of the peer review or editorial decision-making process for this manuscript and had no access to reviewer information or reports.

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The peer review and editorial decision-making processes for this manuscript were conducted exclusively by the other Editor-in-Chief of the journal, who had no involvement in the study and no conflict of interest with the authors or the study group. The conflicted Editor-in-Chief was completely blinded to the process. The handling editor and reviewers were assigned

independently, and a strict double-blind peer review process was maintained. The final decision was made solely by the non-conflicted Editor-in-Chief based on the reviewers' evaluations and the journal's editorial standards.

No conflict of interest was declared by the remaining authors.

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