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RESEARCH ARTICLE

Comparison of Excessive Daytime Sleepiness Among Clinical Types of Obstructive Sleep Apnoea Syndrome

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ABSTRACT

Objective: Excessive daytime sleepiness (EDS) is a key symptom in obstructive sleep apnoea syndrome (OSAS). The relationship between EDS and polysomnographic parameters across different OSAS phenotypes has not been fully elucidated. This study evaluated demographic characteristics, polysomnographic findings, and EDS severity among OSAS clinical phenotypes (classical OSAS, REM [rapid eye movement]-related OSAS, positional OSAS [P-OSAS], and REM+positional OSAS) and subgroups defined according to Epworth Sleepiness Scale (ESS) scores.

Materials and Methods: A retrospective analysis was conducted on patients with OSAS diagnosed by polysomnography. Participants were categorised into clinical phenotypes and stratified into the EDS (ESS score >10) and non-EDS (ESS score ≤10) groups. Demographic data, apnoea–hypopnea index (AHI), oxygen desaturation index (ODI), minimum oxygen saturation, and heart rate (HR) parameters were compared between the EDS and non-EDS groups.

Results: EDS was associated with male sex, the presence of comorbidities (particularly hypertension), and higher AHI, BMI, and body weight values ($p<0.05$). The highest EDS prevalence was observed in the classical OSAS group, followed by the P-OSAS group ($p<0.05$). Patients with EDS showed lower mean and minimum oxygen saturation levels and higher ODI values ($p<0.05$). No significant difference was observed in HR values among the OSAS phenotypes or between the ESS-based subgroups.

Conclusion: EDS was associated with OSAS disease severity. Among the clinical phenotypes, the risk of EDS was observed to be highest in patients with classical OSAS and P-OSAS. The early identification of OSAS subtypes, particularly the REM- and position-dependent forms, is essential for personalised treatment and improved clinical outcomes.

Keywords: Excessive daytime sleepiness, obstructive sleep apnoea syndrome, phenotype, positional

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INTRODUCTION

Obstructive sleep apnoea syndrome (OSAS) is a prevalent medical condition that affects approximately 24% of men and 9% of women, characterised by recurrent episodes of upper airway obstruction, oxygen desaturation, and sleep fragmentation.[1] Existing evidence indicates that upper airway obstruction severity in OSAS varies with body position; in the supine position, the effect of gravity on the mandible and tongue leads to more pronounced upper airway obstruction, increasing OSAS severity.[1] Approximately 60% of patients with OSAS demonstrate a predominance of respiratory events while sleeping in the supine position;[2,3] in ~20% of patients, these events occur exclusively during supine sleep.[3,4]

Medullary sensitivity to hypoxia and hypercapnia is known to decrease during rapid eye movement (REM) sleep, predisposing individuals to a higher frequency of respiratory events in this sleep stage.[5] However, some researchers have reported no significant apnoea–hypopnea index (AHI) differences between the REM and non-REM (NREM) sleep stages.[6,7]

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OSAS is conventionally classified according to the total AHI, without considering the influence of body position or sleep stages.[4,5] However, definitions of positional and REM-related OSAS (P-OSAS and REM-OSAS, respectively) vary across studies. The most widely accepted criteria define REM-OSAS as an AHI during REM sleep (AHI-REM) at least twice that observed during NREM sleep (AHI-NREM) in patients with a total AHI of >5 events/hour. Similarly, P-OSAS is identified as an AHI in the supine position (AHI-supine) that is at least twice that in nonsupine (lateral) positions (AHI-lateral).[2,8]

Findings related to increased daytime sleepiness in OSAS patients remain inconsistent. Although the findings of one study demonstrated that patients with P-OSAS experienced increased sleepiness in comparison with those with non-positional OSAS, another study found the opposite.[2,3] Excessive daytime sleepiness (EDS) is associated with impaired attention, mood disturbances, and other neurocognitive deficits.[4,5] The relationship between the Epworth Sleepiness Scale (ESS) score and OSAS severity remains under investigation. EDS is potentially associated with more nuanced and complex disease parameters in addition to total AHI; for example, Punjabi et al. found no significant association between REM-OSAS and EDS, whereas two other studies demonstrated a positive association.[8,9] Therefore, variations in AHI related to sleep stage and body position should be considered in addition to total AHI when interpreting OSAS severity and planning treatment strategies.

In this study, we aimed to compare polysomnographic findings and levels of daytime sleepiness among OSAS patients according to their clinical subtypes and to review the corresponding treatment approaches.

MATERIALS and METHODS

This study included 290 patients aged 18–66 years who were diagnosed with OSAS via clinical and polysomnography findings. The patients were followed at the Sleep Center between January 2020 and January 2025, Bakırköy Dr. Sadi Konuk Training and Research Hospital. The inclusion criteria were as follows: age ≥18 years, total AHI >5 events/hour, a minimum of 4 hours of total sleep time (TST), at least 15 minutes of REM sleep, and at least 30 minutes spent in both supine and nonsupine positions during the study.

Patients who had previously been diagnosed with OSAS or received treatment, had malignancies and/or psychiatric diseases, did not provide consent, did not complete the full diagnostic process and questionnaire, did not receive at least a primary education, or had uncontrolled chronic diseases (e.g., uncontrolled hypertension [blood pressure >140/90 mm Hg under medical treatment], diabetes [HbA1c level >8% under medical agents]), or cerebrovascular disease with modified Rankin Scale [mRS] scores >2 were excluded.

Our study was conducted in accordance with the tenets of the Declaration of Helsinki and approved by the local ethics committee (decision date: June 25, 2025; number: 11/05). Written informed consent was collected from each participant. No artificial intelligence (AI) tools were used.

Demographic and physical data (age, weight, and body mass index [BMI]), smoking status, comorbidities, and OSAS-related symptoms were recorded. Polysomnographic signals included electroencephalography (EEG), electro-oculography (EOG), submental electromyography (EMG), nasal pressure airflow, electrocardiography (ECG), thoracoabdominal respiratory effort, and oxygen saturation via pulse oximetry. Respiratory events were scored per the standard criteria established by the American Academy of Sleep Medicine (AASM) Task Force.[10] The AHI was determined as the number of apnoeas and hypopneas per hour of estimated TST. OSAS severity is defined on the basis of the AHI. There are three severity classifications: mild (AHI 5.0–14.9 events/hour), moderate (AHI 15.0–29.9 events/hour), and severe (AHI ≥30.0 events/hour).[10]

Polysomnographic assessments included the AHI, AHI-REM in patients with REM-OSAS, supine AHI in those with P-OSAS, TST, sleep efficiency, mean oxygen saturation (mean-SaO_2), minimum oxygen saturation (min-SaO_2), the oxygen desaturation index (ODI), and the mean heart rate (HR).

REM-OSAS was identified as a total AHI of >5/hour and a REM-AHI at least twice as high as the NREM-AHI. P-OSAS was defined as a total AHI of >5/hour with a supine AHI at least twice that of the nonsupine AHI.[10] We divided the patients into four groups: classical OSAS, REM-OSAS, P-OSAS, and REM+P-OSAS.

Subjective daytime sleepiness was assessed via the ESS score, obtained using a self-administered questionnaire. The validity and reliability of the ESS in Turkish populations have been previously confirmed.[11] Patients were asked to rate their likelihood of falling asleep during eight routine activities over the past month, using a scale from 0 to 3. The total ESS score was calculated as the sum of the eight items, ranging from 0 to 24, with higher scores (ESS>10) indicating increased levels of daytime sleepiness.[12] Accordingly, the study cohort was stratified into two subgroups (ESS ≤10 and ESS >10) to explore factors potentially associated with EDS.

Table 1. Comparison of study subgroups and demographic values according to Epworth sleepiness scale score

	ESS score≤10		ESS score>10		Total		Chi-square	p
	n	%	n	%	n	%		
Female	65	62.5	39	37.5	104	100	5.340	0.021*
Male	90	48.4	96	51.6	186	100		
Smoking (-)	89	50.3	88	49.7	177	100	1.830	0.176
Smoking (+)	66	58.4	47	41.6	113	100		
Comorbidity (-)	59	68.6	27	31.4	86	100	17.870	<0.001*
Comorbidity (+)	51	39.2	79	60.8	130	100		
HT (-)	114	58.5	81	41.5	195	100	5.620	0.018*
HT (+)	41	43.6	53	56.4	94	100		
DM (-)	125	55.8	99	44.2	224	100	2.202	0.138
DM (+)	29	45.3	35	54.7	64	100		
CVD (-)	149	54.2	126	45.8	275	100	0.687	0.407
CVD (+)	6	42.9	8	57.1	14	100		
Cardiac disease (-)	136	53.5	118	46.5	254	100	0.876	0.921
Cardiac disease (+)	17	51.5	16	48.5	33	100		
Mild-moderate OSAS	123	66.8	61	33.2	184	100	36.326	<0.001*
Severe OSAS	32	30.2	74	69.8	106	100		
OSAS	19	25.3	56	74.7	75	100	52.457	<0.001*
REM-OSAS	55	73.3	20	26.7	75	100		
P-OSAS	30	41.7	42	58.3	72	100		
REM+P-OSAS	51	75	17	25	68	100		

*: p<0.05. Mann-Whitney U test. ESS: Epworth sleepiness scale; HT: Hypertension; DM: Diabetes Mellitus; CVD: Serebrovascular disease; OSAS: Obstructive sleep apnoea syndrome; P-OSAS: Positional-OSAS; REM-OSAS: Rapid eye movement related OSAS

Statistical Analysis

The data were analysed using IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as frequencies and percentages. The normality of distribution for continuous variables was assessed using the Shapiro–Wilk test. The continuous variables did not meet the assumption of normal distribution; thus, they are summarised using median, minimum, and maximum values. The chi-square test was used for comparisons between two categorical variables. When a significant difference was detected, pairwise comparisons of column proportions were conducted. The Mann–Whitney U test was employed to compare continuous variables between two categorical groups. For comparisons involving continuous variables across more than two categorical groups, the Kruskal–Wallis H test was used. In cases where the Kruskal–Wallis H test indicated a significant difference, pairwise comparisons were performed using the Bonferroni-adjusted Mann–Whitney U test. In all analyses, p<0.05 was considered to indicate significance.

RESULTS

Our study included 290 OSAS patients (104 female / 186 male). Seventy-five (25.8%) patients were diagnosed with REM-OSAS, 72 (24.8%) with P-OSAS, 75 (25.8%) with classical OSAS, and 68 (23.4%) with P+REM-OSAS. Of the 290 patients, 130 had comorbid diseases (94 with HT, 33 with cardiac disease, 64 with DM, 14 with cerebrovascular diseases with mRS scores <2, 15 with hyperlipidaemia, and 3 with benign prostate hypertrophy).

The distribution of the patients' demographic and clinical characteristics according to their ESS scores is shown in Table 1. The proportion of females was higher in the ESS ≤10 group (62.5%), whereas males predominated in the ESS >10 group (51.6%). This sex distribution difference between the groups was significant (χ^2=5.340, p=0.021). No significant difference was observed in smoking status between the ESS groups (χ^2=1.830, p=0.176). Patients with comorbidities experienced significantly higher sleepiness levels (χ^2=17.870, p<0.001).

Table 2. Comparison of demographic and polysomnography values according to Epworth sleepiness scale score

	ESS score≤10 median (min-max)	ESS score>10 median (min-max)	Total median (min-max)	Z	p
Age	49 (18–66)	50 (27–66)	50 (18–66)	-1.123	0.261
Height	1.7 (1.49–1.92)	1.7 (1.5–1.93)	1.7 (1.49–1.93)	-1.885	0.059
Weight	85 (46–135)	91.5 (62–145)	89 (46–145)	-3.709	<0.001*
BMI	30.09 (18–45.01)	31.25 (22.86–60.35)	30.928 (18–60.35)	-2.539	0.011*
AHI	18.5 (5.1–89.7)	33.2 (6.3–104.3)	21.55 (5.1–104.3)	-5.846	<0.001*
Supine-AHI	32.7 (6.5–114.1)	54.45 (11.9–134)	40.2 (6.5–134)	-3.281	0.001*
REM-AHI	41.1 (14.7–111.8)	48.5 (15.5–91.4)	44.7 (14.7–111.8)	-1.629	0.103
TST	374 (244.5–486.3)	390.75 (250–565.2)	382 (244.5–565.2)	-1.723	0.085
Sleep efficiency	78.9 (46.5–97.3)	81.7 (24.9–97.5)	80.1 (24.9–97.5)	-1.612	0.107
Mean-SaO_2	94 (86–97)	93.4 (78–97.2)	93.6 (78–97.2)	-2.561	0.010*
Min-SaO_2	85 (54–93)	81 (50–92)	83 (50–93)	-4.821	<0.001*
HR	66.75 (43.3–95.7)	68,4 (48.6–93.5)	67 (43.3–95.7)	-1.229	0.219
ODI	15.4 (2.7–90.5)	27.85 (5.2–96.5)	20.3 (2.7–96.5)	-3.026	0.002*

*: p<0.05. Mann-Whitney U test. ESS: Epworth sleepiness scale; BMI: Body mass index; AHI: Apnea-hypopnea index; REM: Rapid eye movement; TST: Total sleep time; Mean-SaO_2: Mean oxygen saturation; Min-SaO_2: Minimum oxygen saturation; HR: Mean heart rate; ODI: Oxygen desaturation index.

A significantly higher prevalence of HT was observed in the ESS >10 group (χ^2=5.620, p=0.018). No significant difference in DM, CVD, or cardiac disease presence was found between the ESS groups (p>0.05). Patients with mild-to-moderate AHI were more frequently observed in the ESS ≤10 group (66.8%), whereas those with severe AHI were significantly more common in the ESS >10 group (69.8%) (χ^2=36.326, p<0.001). Thus, the severe OSAS group demonstrated higher sleepiness levels than the mild-to-moderate OSAS group (Table 1).

ESS scores differed significantly among the OSAS clinical subgroups (χ^2=52.457, p<0.001). The classical OSAS and P-OSAS groups demonstrated greater sleepiness levels (ESS >10) than the REM-OSAS and REM+P-OSAS groups (ESS ≤10) (Table 1).

No significant differences in age, height, TST, sleep efficiency, or HR values were observed between the ESS groups (p>0.05; Table 2). Weight, BMI, AHI, and ODI values were higher in the group demonstrating greater sleepiness (ESS score >10); furthermore, the mean and minimum O_2 saturation values were lower than those in the group demonstrating lower sleepiness (ESS score ≤10) (Table 2).

A significant female predominance was observed in the REM-OSAS group, whereas male predominance was observed in the P-OSAS, classical OSAS, and REM+P-OSAS groups (p=0.001) (Table 3). Smoking history was present in 113 patients (38.9%), and no significant difference was found among the clinical types (Table 3). HT and DM were the most common comorbid conditions in all clinical types, and there was no significant difference in the incidence of comorbid diseases across the clinical types (p>0.05). Only DM demonstrated a significantly higher prevalence in the classical OSAS subgroup compared to the other OSAS subgroups (p=0.031) (Table 3).

No significant difference was observed in the median patient age across the OSAS clinical types (p=0.994; Table 4). The median BMI was 32.08 in classical OSAS patients and 29.39 in those with P-OSAS (p=0.005; Table 4). Patients with classical OSAS were significantly more likely to be overweight than those with REM-OSAS (p<0.001; Table 4).

The median total AHI was significantly higher in the classical OSAS subgroup compared to the other subgroups; this value was also higher in the P-OSAS group than in the REM-OSAS subgroup (p<0.001; Table 4).

The TST was significantly higher in the classical OSAS and REM-OSAS groups than in the REM+P-OSAS group (p<0.05). Mean-SaO_2 and min-SaO_2 were significantly higher in the P-OSAS subgroup compared to the classical OSAS subgroup (p<0.05). DSI was higher in the classical OSAS and P-OSAS subgroups than in the REM-OSAS and REM+P-OSAS subgroups (p<0.05). No significant difference was observed in the HR values across the clinical OSAS types (Table 4).

DISCUSSION

EDS is a common symptom in patients with OSAS, adversely affecting daily activities, reducing occupational performance, and increasing the risk of injury. Among OSAS patients,

Table 3.Comparison of OSAS clinical types according to demographic values and disease severity

	OSAS		REM-OSAS		P-OSAS		REM+P-OSAS		Total		Chi-square	p
	n	%	n	%	n	%	n	%	n	%		
Female	21	20.2	39	37.5	16	15.4	28	26.9	104	100	17.166	0.001*
Male	54	29	36	19.4	56	30.1	40	21.5	186	100		
Smoking (-)	48	27.1	49	27.7	38	21.5	42	23.7	177	100	2.939	0.401
Smoking (+)	27	23.9	26	23	34	30.1	26	23	113	100		
Comorbidity (-)	30	23.3	35	26.7	29	22.1	36	27.9	130	100	4.009	0.260
Comorbidity (+)	50	31.5	41	25.4	41	25.4	28	17.7	160	100		
HT (-)	44	22.6	51	26.2	50	25.6	50	25.6	195	100	4.078	0.253
HT (+)	31	33	24	25.5	21	22.3	18	19.1	94	100		
DM (-)	50	22.3	64	28.6	58	25.9	52	23.2	224	100	8.464	0.037*
DM (+)	25	39.1	11	17.2	13	20.3	15	23.4	64	100		
Cardiac disease (-)	65	25.6	65	25.6	64	25.2	60	23.6	254	100	3.506	0.885
Cardiac disease (+)	10	30.3	9	27.3	7	21.2	7	21.2	33	100		
Mild-moderate OSAS	26	14.1	59	32.1	44	23.9	55	29.9	184	100	43.361	<0.001*
Severe OSAS	49	46.2	16	15.1	28	26.4	13	12.3	106	100		

*: p<0.05. Kruskal-Wallis H Test. OSAS: Obstructive sleep apnoea syndrome; REM-OSAS: Rapid eye movement related OSAS; P-OSAS: Positional-OSAS; HT: Hypertension; DM: Diabetes mellitus

Table 4. Comparison of OSAS clinical types according to demographic, polysomnography and ESS values

	OSAS (A) median (min-max)	REM-OSAS (B) median (min-max)	P-OSAS (C) median (min-max)	REM+P-OSAS (D) median (min-max)	Total median (min-max)	Z	p	Difference
Age	50 (29–65)	50 (18–66)	49.5 (24–66)	51 (24–65)	50 (18–66)	0.08	0.994	–
Height	1.7 (1.5–1.87)	1.65 (1.49–1.85)	1.71 (1.5–1.87)	1,7 (1.5–1.93)	1.7 (1.49–1.93)	11.322	0.010*	C>B
Weight	94 (62–145)	83 (60–117)	86 (60–135)	89 (46–135)	89 (46–145)	13.992	0.003*	A>B
BMI	32.08 (23.36–60.35)	30.86 (22.23–42.46)	29.39 (22.78–45.01)	31.25 (18–43.25)	30.93 (18–60.35)	12.864	0.005*	A>C
AHI	43.5 (5.5–104.3)	16.9 (5.1–58.7)	24.4 (5.6–89.4)	19.25 (5.2–89.6)	21.55 (5.1–104.3)	55.423	<0.001*	A>B, C, D C>B
Supine-AHI	(–)	(–)	52.15 (6.5–134.1)	31.8 (9.2–94.1)	40.2 (6.5–134.1)	10.43	0.005*	C>D
REM-AHI	(–)	43.8 (14.7–92.4)	(–)	45.6 (15.5–111.8)	44.7 (14.7–111.8)	0.082	0.960	–
TST	401.5 (245–521.1)	390.5 (245–497)	374.5 (240.5–565.2)	363 (244.5–473.4)	382 (244.5–565.2)	12.193	0.007*	A,B>D
Sleep efficiency	83.7 (44.9–96.8)	80.3 (51.1–97.5)	78.4 (41–97.3)	77.65 (46.5–96.6)	80.1 (44.9–97.5)	10.81	0.013*	A>D
Mean-SaO_2	93.1 (78–97)	94 (84.8–97)	93.9 (88.1–96.8)	93.4 (86–97.2)	93.6 (78–97.2)	8.909	0.031*	C>A
Min-SaO_2	81 (50–93)	83 (50–90)	84 (64–91)	84 (54–92)	83 (50–93)	10.659	0.014*	C>A
HR	68.5 (51.8–91.7)	67.15 (43.3–93.5)	65.9 (49.8–87.1)	67 (50.3–95.7)	67 (43.3–95.7)	1.515	0.679	–
ODI	39.95 (4–96.5)	13.1 (3.8–47.1)	25.7 (2.7–67.3)	16 (3.2–87.1)	20.3 (2.7–96.5)	21.987	<0.001*	A,C>B, A>D
ESS score	12 (1–24)	6 (1–22)	10 (1–20)	5 (1–21)	8 (1–24)	42.721	<0.001*	A,C>D, A,C>B

*: p<0.05. Z: Kruskal-Wallis H Test. BMI: Body mass index; AHI: Apnoea–hypopnea index; REM: Rapid eye movement; TST: Total sleep time; HR: Mean heart rate; ODI: Oxygen desaturation index

58.3% experience varying degrees of daytime sleepiness.[13] Distinct OSAS phenotypes have been identified in recent years, and the presence of EDS has become a key determinant in OSAS classification.

In this study, demographic characteristics, polysomnographic data, and daytime sleepiness rates were compared among patients with different clinical types of OSAS—namely, REM-OSAS, P-OSAS, REM+P-OSAS, and classical OSAS. Our findings indicated that EDS was more prevalent in male patients diagnosed with OSAS, in those with comorbidities—particularly HT—and in patients with higher AHI, BMI, and body weight values. When comparing the prevalence of EDS across OSAS clinical types, the highest rate was observed in the classical OSAS group, followed by the P-OSAS group. Regarding polysomnographic findings, patients with increased daytime sleepiness had lower mean-SaO_2 and min-SaO_2 levels and higher ODI values. TST and sleep efficiency were lower in the REM+P-OSAS clinical type compared to the other types.

Notably, sex-related differences have been demonstrated in previous studies. It has been suggested that OSAS is more prevalent in males due to patterns of fat distribution.[14] Oestrogen and progesterone are believed to exert a protective effect by increasing upper airway muscle tone; however, this effect diminishes during REM sleep and after menopause, thereby increasing the risk of REM-OSAS in women and in older individuals.[15] In our study, REM-OSAS was observed more frequently in women, although the age range was similar to that of patients with the other clinical types. Some studies have demonstrated increased daytime sleepiness in women,[16,17] which is inconsistent with our study. Additionally, consistent with the literature, no significant difference was observed in the mean patient age between the EDS and non-EDS groups.[17]

Obesity is considered a chronic inflammatory condition and is one of the most important risk factors for OSAS.[18] Visceral fat accumulation and fat deposits in the upper airway contribute to hypoventilation and/or oxygen desaturation, leading to increased nocturnal awakenings and sleep fragmentation, resulting in EDS.[18] In our study, consistent with the literature, a significant association was found between higher body weight and BMI values and the presence of EDS.[19]

Numerous comorbid conditions are OSAS risk factors, such as cardiovascular diseases, HT, DM, and chronic obstructive pulmonary disease (COPD); a positive correlation between disease severity and comorbidity level has been demonstrated.[20,21] In our study, EDS was observed at a higher rate in OSAS patients with comorbidities, particularly those with hypertension. In one study, EDS was associated with increased cardiovascular risks in coronary artery disease patients, especially in those with OSAS.[22] However, in our study, we did not detect a statistically significant increase in sleepiness among patients with cardiac disease. Nevertheless, we consider that the relatively small number of OSAS patients with concomitant cardiac disease (n=33) may have contributed to this finding.

Some studies have demonstrated no strong correlation between total AHI and ESS scores.[8,23] Jung et al.[24] demonstrated that ESS scores increase with OSAS severity. In our study, higher AHI scores were found to be associated with increased sleepiness. These findings are consistent with previous studies demonstrating higher mean ESS scores and a greater prevalence of EDS.[25,26] Although some studies have shown a significant association between REM-OSAS and daytime sleepiness, others have found no such relationship.[8,9,27] Furthermore, Punjabi et al.[9] demonstrated a greater EDS prevalence in NREM-related OSAS via objective measures, including the Multiple Sleep Latency Test (MSLT). Our study and many previous studies employed the ESS, a subjective measurement tool, which may account for discrepancies across studies.

Moreover, body position plays a decisive role in OSAS severity. In particular, in the supine position, the gravitational effect on upper airway structures increases airway narrowing, thereby exacerbating OSAS.[2] However, whether P-OSAS is associated with increased EDS remains unclear, as conflicting findings have been reported.[2–4] In our study, patients with P-OSAS were found to have significantly increased daytime sleepiness, similar to that observed in classical OSAS patients.

REM+P-OSAS emerged as a clinically distinct phenotype. This group typically comprises women, individuals with lower BMI, and those with milder disease severity. Despite measurable respiratory events, these patients may exhibit lower subjective levels of sleepiness.[3] In our study, both AHI and ESS scores were lower in this group than in the classical OSAS and P-OSAS groups.

One study demonstrated a positive correlation between the ESS score and AHI and a negative correlation between the ESS score and min-SaO_2.[28] Another study revealed a mild correlation between the AHI and total ESS score in patients with sleep-related breathing disorders, as well as a mild negative correlation between the total ESS score and min-SaO_2, and between the total ESS score and mean-SaO_2.[12] In contrast, other studies have failed to demonstrate a significant correlation between the ESS score and AHI or min-

SaO_2.[29,30] In our study population, O_2 saturation parameters—such as mean-SaO_2 and min-SaO_2—were lower in the classical OSAS and high ESS score groups.

Adults with OSAS who experience EDS appear to be at a significantly higher risk for cardiovascular diseases than those without EDS.[31] Increased sympathetic activation and cardiovascular instability during apnoeas in REM sleep are associated with more severe hypoxemia and an elevated risk of arrhythmias.[32] In our study, no significant difference was observed in HR values among the clinical OSAS phenotypes or between the ESS subgroups.

Smoking can cause abnormal changes in the upper respiratory tract histologically and physiologically, and several mechanisms have been hypothesised to explain how smoking increases the risk of developing OSAS. Although a previous study has reported an association between smoking and both OSAS severity and increased daytime sleepiness, we did not observe EDS among OSAS patients who were smokers—consistent with the findings of another study.[33,34]

To our knowledge, no previous study has compared EDS across all clinical phenotypes of OSAS; thus, we believe that the findings of our study provide a valuable contribution to the existing body of knowledge.

However, our study has several limitations. First, its retrospective nature limits the study design. Furthermore, relying on data obtained from a single-night polysomnographic assessment limits the generalisability of the findings. Multiple-night PSG assessments may provide more robust evidence. Additionally, although the ESS is a widely used tool, it is a subjective measurement method dependent on the patient's responses to the test scale and may not capture all dimensions of sleepiness as sensitively as objective tests such as the MSLT.

CONCLUSION

In our study, EDS in patients with OSAS was found to increase with disease severity, and patients with P-OSAS demonstrated a similar risk of EDS to those with classical OSAS. This finding underscores the need for a more sensitive and individualised approach during the diagnostic and therapeutic process. Considering patient quality of life, it may be appropriate to initiate positive airway pressure therapy in P-OSAS patients earlier and at lower AHI thresholds. OSAS is a heterogeneous disorder; thus, identifying REM subtypes and, especially, positional subtypes is of critical importance for developing personalised treatment strategies and improving clinical outcomes.

Disclosures

Ethics Committee Approval: The study was approved by the Bakırköy Dr. Sadi Konuk Training and Research Hospital Non-interventional Scientific Research Ethics Committee (No: 11/05, Date: 25/06/2025).

Informed Consent: Written informed consent was collected from each participant.

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