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The presence of sleep apnoea and evaluation of sleep structures in epilepsy subtypes

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Abstract

Background & Objective: Identifying seizure-triggering factors and taking precautions in epilepsy patients are important for seizure control and disease progression. Comorbidity between sleep disorders and epilepsy is frequently observed. The severity of epilepsy may affect the risk of sleep apnoea, and obstructive sleep apnoea is thought to exacerbate epilepsy. We thought that there may be differences in the rate of sleep apnoea, sleep structures and sleep characteristics between focal and generalized epilepsy patients. The aim of this study was to compare sleep apnoea and sleep characteristics between patients diagnosed with focal and generalised epilepsy. *Methods:* We evaluated polysomnography findings, clinical features, the Epworth Sleepiness Scale, the Pittsburgh Sleep Quality Index and the STOP–BANG tests of patients who were diagnosed with focal and primary generalised epilepsy and normal individuals without a diagnosis of epilepsy. The collected data were compared between the generalized epilepsy, focal epilepsy and control group. *Results:* We included 84 individuals (22 patients with generalised epilepsy, 26 patients with focal epilepsy and 36 controls) in our study. The median age was 37 years (range 19-69). The apnoea-hypopnea index (AHI) (p=0.402) and the sleep apnoea percentage (p=0.385) were no significantly difference between the generalised and focal epilepsy groups. Sleep apnoea was detected in 50% of the epilepsy patients. The STOP–BANG score was significantly higher in the sleep apnoea group than in the control (p = 0.021). The most common sleep disturbance symptoms were daytime sleepiness in the generalised and focal epilepsy groups. *Conclusion:* The sleep apnoea percentage were no difference between the generalised and focal epilepsy groups. When sleep apnoea is suspected in epilepsy patients, and when polysomnography is not possible, evaluation can be made with the STOP–BANG test.

Keywords: Sleep apnoea, epilepsy, polysomnography

INTRODUCTION

There is a well-known complex bidirectional relationship between sleep and epilepsy[1], part of which is that sleep disorders worsen epileptic seizures. Some antiseizure medications affect sleep structure independently of their effects on seizures[2], and antiseizure medications and other nonpharmacological epilepsy interventions can affect sleep quality.[3] In epilepsy patients, breathing irregularities may occur during seizures, after seizures and interictally. Sleep-disordered breathing (SDB), such as obstructive sleep apnoea (OSA), is observed in many epilepsy patients.[4] However, OSA worsens seizure frequency and severity through sleep disruption and hypoxemia. Continuous positive airway pressure (CPAP) treatment provides improvement in seizure frequency, daytime alertness and quality of life.[2,5,6] Potential mechanisms are thought to occur through changes in sleep architecture, sleep deprivation or hypoxia. In this way, another vicious cycle occurs in which epilepsy can increase the prevalence of OSA, which can worsen epilepsy.[3] Gammino *et al*. found no statistically significant difference in the prevalence of obstructive sleep apnoea syndrome (OSAS) between epilepsy patients and controls but showed that the percentage of OSAS was higher in male patients, the elderly, severe forms of focal epilepsy and secondary generalised seizures.[7] By functional connectivity, temporal lobe seizures originating in the insular cortex

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may cause sleep-related breathlessness.[8,9] There are few publications in the literature comparing sleep characteristics across epilepsy subtypes. We thought that there may be differences in the rate of sleep apnoea, sleep structures and sleep characteristics between focal and generalized epilepsy patients. Knowing the differences in sleep characteristics may help in monitoring different epilepsy groups. The current study aimed to compare polysomnography (PSG) data, sleep apnoea, sleep structures and sleep characteristics of focal and primary generalised epilepsy patients.

METHODS

Approval for this study was received from the Ankara Training and Research Hospital Ethics Committee. The patients with a diagnosis of focal or generalized epilepsy who came to the neurology outpatient clinic for routine control for epilepsy evaluated. They did not apply with a complaint specifically related to sleep. We evaluated the sleep characteristics of all epilepsy patients who applied, whether they had a sleep complaint or not. PSG was performed to patients who agreed to have PSG. Healthy individuals without epilepsy or sleep disorders were taken as the control group (Figure 1).

We retrospectively evaluated the polysomnography findings, clinical features (duration of epilepsy, treatment, seizure type, seizures at night), the Epworth Sleepiness Scale (ESS), the Pittsburgh Sleep Quality Index (PSQI) and the Snoring-Tiredness-Observed apnoea-Pressure–Body Mass Index-Age-Neck circumference-Gender (STOP–BANG) tests of participants included in the study. The collected data were compared between the generalized epilepsy, focal epilepsy and control group. Pregnant and mentally retarded patients were not included in the study.

One night, polysomnography recording was performed with a Philips Alice 6 polysomnography device. Electroencephalography (EEG), electrooculography, chin and tibialis anterior electromyography and electrocardiography were recorded according to the American Sleep Disorders Academy criteria. Airflow with a nasal cannula and thermistor, respiratory effort with chest and abdominal belts and oxygen saturation were recorded. Body position was recorded with a sensor. The recordings were performed by a sleep technician and scored by a sleep physician according to the American Academy of Sleep Medicine 2.3 criteria.[10] The apnoea-hypopnea index (AHI) was determined as the number of apnoea-hypopneas per hour during sleep. If the AHI value was five or above, it was determined as sleep apnoea.

The PSQI is an 18-item questionnaire that measures sleep quality and disorders over a one-month period.[11] The ESS is a self-administered eight-item questionnaire that is used to assess daytime sleepiness in adults. The ESS Turkish version is a reliable and valid measure of daytime sleepiness.[12] The STOP–BANG questionnaire, which was used to identify patients at risk for OSA, contains eight questions, four of which are subjective (snoring, tiredness, observed apnoea and high blood pressure), and four that pertain to demographics (body mass index > 35 kg/m2, age > 50 years, neck circumference > 40 cm and male gender).[13]

Statistical analysis

All analyses were performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test was used to determine whether continuous variables

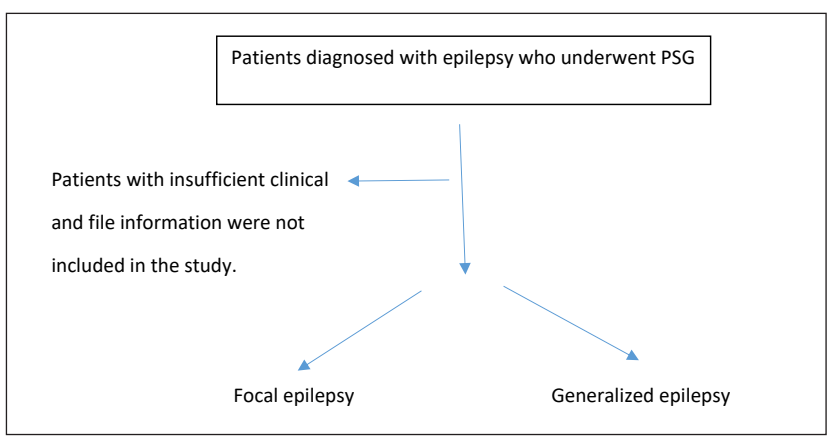

Figure 1. Flow diagram

were normally distributed. Data are given as mean ± standard deviation or median (1st quartile–3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables. Normally distributed continuous variables were analysed with one-way analysis of variances (ANOVA). Non-normally distributed continuous variables were analysed with the Mann–Whitney U test or Kruskal–Wallis test, depending on the group counts. Categorical variables were analysed with the chi-square test, Fisher's exact test or Fisher–Freeman–Halton test. Pairwise comparisons were adjusted using the Bonferroni correction method. Two-tailed p-values of less than 0.05 were considered statistically significant. Spearman correlation coefficients were calculated to evaluate the relationships between continuous variables.

RESULTS

We included 84 individuals (22 patients with generalised epilepsy, 26 patients with focal epilepsy and 36 controls) in our study. The median age was 37 years (range 19–69). We found no significant differences between the groups in terms of age, sex, body mass index, neck circumference and hypertension.

The focal epilepsy group consisted of 20 (76.9%) patients with occipital epilepsy, 5 (19.2%) with temporal epilepsy and 1 (3.8%) with frontal lobe epilepsy. In the generalised group, 19 (86.4%) had generalised seizures, 15 (68.2%) had myoclonic seizures and 9 (40.9%) had absence seizures. We found no significant differences between the generalised and focal epilepsy groups in terms of the duration of disease and seizures at night.

The most commonly used treatment was valproic acid in the generalised and focal groups. Carbamazepine treatment was significantly higher in the focal group than in the generalised group ($p = 0.001$). We found no significant differences between the epilepsy groups in terms of the number of medications and other treatment agents (Table 1). The most common sleep disturbance symptoms were daytime sleepiness in the generalised and focal epilepsy groups and fatigue in the morning in the control group. There were no significant differences between the groups in terms of sleep disturbance symptoms.

Sleep latency was significantly higher in the generalised group than in the focal group ($p = 0.033$). AHİ ($p < 0.001$) and rapid eye movement (REM) sleep AHI ($p < 0.001$) scores were significantly lower in the control group than in the generalised and focal epilepsy groups (Figures 2 and 3). AHI ($p= 0.402$) and REM AHI ($p= 1$) scores were no significantly difference between the generalised and focal epilepsy groups. The oxygen desaturation index ($p = 0.023$) were significantly higher in the focal epilepsy group than in the control group (Figure 4). We found no significant differences between the groups in terms of other polysomnography results, ESS score, STOP–BANG Sleep Apnoea Questionnaire score and PSQI score (Table 2).

Sleep apnoea was detected in 50% of the epilepsy patients. Notably, the sleep apnoea percentage was no difference between the generalised epilepsy and focal epilepsy groups ($p= 0.385$) (Table 3). No significant relationship was found between the number of medications and the presence of sleep apnea ($p=1$) (Table 4).

The STOP–BANG score was significantly higher in the sleep apnoea group than in the others ($p = 0.021$). In addition, the STOP–BANG intermediate-risk and high-risk percentages were significantly higher in the sleep apnoea group than in the others ($p = 0.038$) (Table 4).

DISCUSSION

Sleep disorders constitute an important part of epilepsy comorbidity.[14] In our study, sleep apnoea was detected in 50% of epilepsy patients. Sleep apnoea rates and the AHI were no significantly difference between the generalised and focal epilepsy groups. Additionally, the most common sleep disorder in patients with generalised and focal epilepsy in our study was excessive daytime sleepiness (EDS).

Epilepsy is associated with changes in sleep macro and micro structures.[15,16] Non-REM and REM phases of sleep have variable effects on temporal and extratemporal epilepsy.[1] While interictal epileptiform discharges are activated at a maximum level in slow-wave sleep, they decrease in the REM phase.[2,17]

The most common sleep disorders in people with epilepsy are EDS, insomnia and SDB.[2,5,6,18,19,20] The prevalence of EDS in people with epilepsy varies between 10% and 47.5% (21). One study showed that epilepsy patients had more daytime sleepiness than controls (11.1% vs. 5.20%, respectively), but a statistically significant prevalence of EDS was not found.[7] Additionally, EDS may be associated with undiagnosed sleep disorders more often than epilepsy-related factors and may be improved by treating coexisting

Table 1: Summary of individuals' and epilepsy characteristics with regard to groups

	Groups			p
	Generalized epilepsy (n=22)	**Focal epilepsy (n=26)**	**Control (n=36)**	
Age(years)	36 (25 - 45)	36.5 (29 - 50)	38.5 (30 - 47)	0.397
Sex(n)				
Female	13 (59.1%)	14 (53.8%)	25 (69.4%)	0.437
Male	9 (40.9%)	12 (46.2%)	11 (30.6%)	
Body mass index(kg/m2)	25.10 (23.30 - 30.40)	26.00 (22.00 - 29.40)	25.60 (22.35 - 28.70)	0.868
Neck circumference(cm)	35.00 ± 2.15	36.98 ± 4.03	36.25 ± 3.67	0.117
Hypertension(mmHg)	2 (9.1%)	5 (19.2%)	4 (12.1%)	0.657
Duration of epilepsy (years)	17 (7 - 21)	8.5 (2 - 21)	-	0.144
Seizure at night	12 (54.5%)	15 (57.7%)	-	1.000
Number of medications(n)				
Monoteraphy	19 (86.4%)	16 (61.5%)	-	0.124
Polyteraphy	3 (13.6%)	10 (38.5%)	-	
Medication (n) [(1)]				
Valproic acid	15 (68.2%)	11 (42.3%)	-	0.133
Carbamazepine	0 (0.0%)	10 (38.5%)	-	**0.001**
Levetrasetam	7 (31.8%)	9 (34.6%)	-	1.000
Lamotrigine	1 (4.5%)	5 (19.2%)	-	0.199
Topiramate	0 (0.0%)	1 (3.8%)	-	1.000
Clobazam	0 (0.0%)	1 (3.8%)	-	1.000
Lacosamide	0 (0.0%)	1 (3.8%)	-	1.000
Pregabalin	1 (4.5%)	0 (0.0%)	-	0.458
Oxcarbazepine	1 (4.5%)	1 (3.8%)	-	1.000

Data are given as mean ± standard deviation or median (1st quartile - 3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables. (1) Individuals may have more than one of the followings.

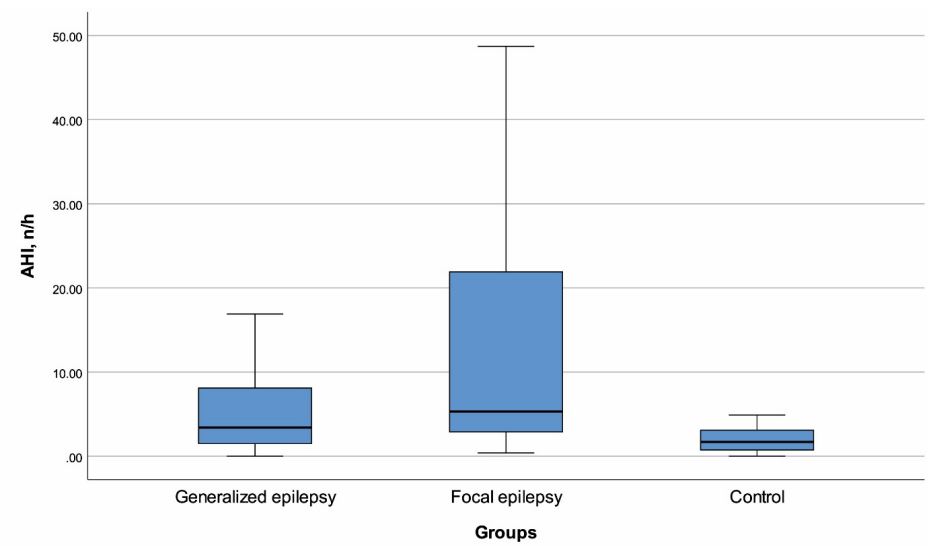

Figure 2. Box-plots of the apnea-hypopnea index with regard groups

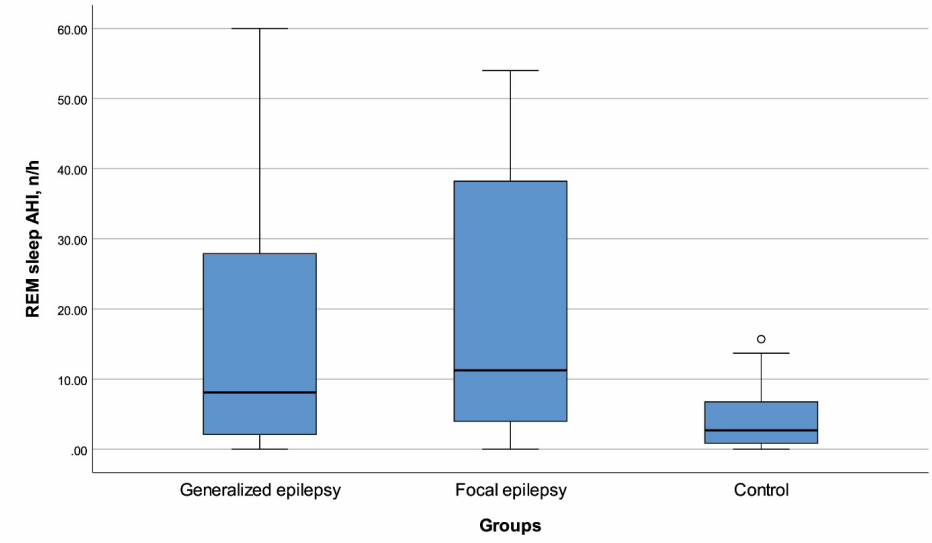

Figure 3. Box-plots of the REM sleep apnea-hypopnea index with regard groups

primary sleep disorders.[21] In our study, EDS was the most common sleep disorder symptom in the focal and generalised epilepsy groups. However, there was no significant difference between the groups in terms of the ESS and EDS.

One study showed that the prevalence of restless leg syndrome (RLS) in epilepsy patients was higher than in the healthy population.[14] Another reported a statistically significant relationship between RLS and epilepsy[7], and a high proportion of patients complained of insomnia.[7] In yet another study, epilepsy patients had more complaints about maintaining and initiating sleep than healthy controls, and it was stated that the severity of epilepsy affected insomnia.[22] The presence of nocturnal seizures, the absence of a longer seizure-free period and EEG abnormalities have been detected more frequently in patients with insomnia.[22] In our study, sleep latency in polysomnography was significantly higher in the generalised epilepsy group than in the focal epilepsy group. However, no significant difference was found in sleep disorder symptoms between the groups. There was also no significant difference between the groups in terms of PSQI scores and periodic limb movement.

The most common sleep comorbidity in patients with refractory epilepsy is OSA.[2,19] The prevalence of OSAS is estimated to be between 30% and 60% in children with epilepsy. Although

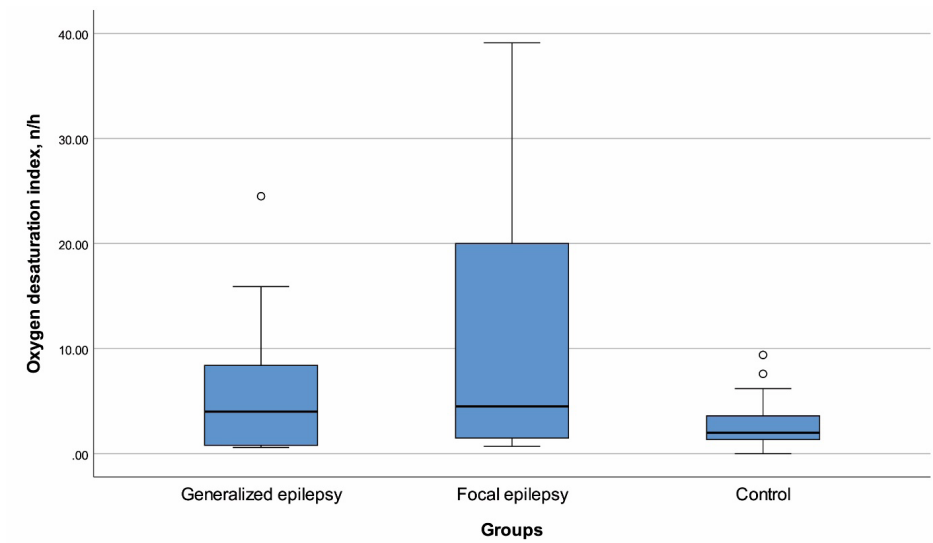

Figure 4. Box-plots of the oxygen desaturation index with regard groups

Table 2: Summary of sleep disturbance symptoms, polysomnography results and sleep assessment scores with regard to groups

	Groups			p
	Generalized epilepsy (n=22)	Focal epilepsy (n=26)	Control (n=36)	
Sleep disturbance symptoms (1)				
Snore	12 (54.5%)	11 (42.3%)	18 (50.0%)	0.687
Apnoea	5 (22.7%)	10 (38.5%)	13 (36.1%)	0.462
Daytime sleepiness	15 (68.2%)	17 (65.4%)	23 (63.9%)	0.946
Fatigue in the morning	13 (59.1%)	16 (61.5%)	28 (77.8%)	0.237
Headache in the morning	6 (27.3%)	10 (38.5%)	12 (33.3%)	0.715
Difficulty in falling asleep	4 (18.2%)	10 (38.5%)	10 (27.8%)	0.298
Wake up during night frequently	4 (18.2%)	7 (26.9%)	6 (16.7%)	0.588
Total sleep time(minute)	385.17 ± 71.92	388.20 ± 54.23	392.46 ± 59.88	0.904
Total recording time(minute)	472.61 ± 48.40	452.94 ± 37.35	471.34 ± 58.02	0.281
Sleep latency(minute)	20 (5.5 - 38.5) [a]	7.5 (3 - 17) [b]	14.5 (7 - 20) [ab]	**0.033**
Sleep efficiency(%)	84.6 (72.7 - 89.6)	89.1 (76.9 - 92.8)	85.3 (74.55 - 92.85)	0.371
Wake after sleep onset(minute)	52.5 (21 - 80)	40.75 (18 - 68)	52 (21.5 - 82)	0.544
REM sleep latency(minute)	87.25 (76 - 153)	107.25 (84 - 124.5)	115 (72.5 - 171.5)	0.920
N1 percentage(%)	15.75 (10.3 - 24.5)	14.15 (9.7 - 21)	15.8 (11.15 - 19.5)	0.769
N2 percentage	49.7 (42.8 - 58.7)	50.85 (41.5 - 57.3)	48.65 (44.35 - 53.95)	0.988
N3 percentage	17.3 (12.9 - 22.4)	17.1 (12.1 - 27.1)	19.65 (14.15 - 23.25)	0.672
REM percentage	15.6 (10.2 - 20.3)	14.75 (11.8 - 18.8)	16.15 (13.8 - 18.85)	0.762
N1 episode(n)	34.5 (28 - 47)	35 (32 - 47)	41.5 (31 - 50.5)	0.541
N2 episode	30.5 (22 - 41)	32 (26 - 43)	34.5 (28 - 42.5)	0.394
N3 episode	5 (3 - 8)	6 (3 - 7)	6.5 (5 - 8)	0.254
REM episode	8 (5 - 10)	7 (4 - 10)	7 (6 - 9.5)	0.880
Apnoea-hypopnea index(n/h)	3.4 (1.5 - 8.1) [a]	5.3 (2.9 - 21.9) [a]	1.7 (0.75 - 3.1) [b]	**<0.001**
REM sleep apnea-hypopnea index(n/h)	8.1 (2.1 - 27.9) [a]	11.25 (4 - 38.2) [a]	2.7 (0.8 - 6.9) [b]	**<0.001**
Periodic limb movement index(n/h)	0.9 (0 - 7.7)	4.9 (0.9 - 9)	1.2 (0 - 4.35)	0.152
Arousal Index(n/h)	2.9 (2.1 - 5.1)	4.65 (2.4 - 6.6)	3.45 (1.85 - 7.35)	0.446
Oxygen desaturation index(n/h)	4 (0.8 - 8.4) [ab]	4.5 (1.5 - 20) [a]	2 (1.35 - 3.6) [b]	**0.023**
Time below oxygen saturation 90%(minute)	0.1 (0.1 - 0.2)	0.2 (0.1 - 3.2)	0.1 (0.0 - 0.2)	0.051
Minimum oxygen saturation (%)	91 (88 - 92)	87 (83 - 91)	90.5 (87 - 92)	0.061
Average oxygen saturation(%)	96 (95 - 97)	95 (93 - 96)	95 (94.5 - 96)	0.068
Epworth Sleepiness Scale score	7.5 (4 - 10)	4 (2 - 10)	8.5 (5 - 11)	0.119
STOP-BANG score	1 (1 - 2)	2 (1 - 3.5)	2 (1 - 3)	0.399
Low	12 (80.0%)	13 (54.2%)	16 (55.2%)	
Intermediate	2 (13.3%)	8 (33.3%)	9 (31.0%)	0.568
High	1 (6.7%)	3 (12.5%)	4 (13.8%)	
PSQI score	5.55 ± 3.38	6.19 ± 3.58	6.36 ± 3.71	0.698

Data are given as mean ± standard deviation or median (1st quartile - 3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables. (1) Individuals may have more than one of the followings. Same letters denote the lack of statistically significant difference between groups.
REM: Rapid eye movement, STOP-BANG: STOP-BANG Sleep Apnea Questionnaire, PSQI: Pittsburgh Sleep Quality Index

NOTE: The letters in Table 2 indicate pairwise comparison results. The lettering a a b indicates that the third group is different from the others and that there is no difference between the first and second groups. The lettering a b ab indicates that the first group and the second group are different, and the third group is similar to the other groups.

Table 3: Summary of sleep apnea with regard to groups

	Groups		p
	Generalized epilepsy (n=22)	Focal epilepsy (n=26)	
Sleep apnoea(n)			
No, AHI<5	13 (59.1%)	11 (42.3%)	0,385
Yes, AHI≥5	9 (40.9%)	15 (57.7%)	

weight gain is a possible risk factor, the risk of OSAS is highest in those with poorly controlled epilepsy requiring polypharmacy.[3,23] Another study found that the duration of epilepsy was longer and polytherapy was more frequent in patients at risk of sleep apnoea.[22] The chronicity of epilepsy with increasing age and the long-term effects of antiepileptic drugs may lead to an increase in the risk of sleep apnoea.[22,24] According to a meta-analysis, the prevalence of OSA in epilepsy patients is higher than in the general population, but the prevalence of OSA in patients with resistant epilepsy is not higher than in patients with general epilepsy, and there is no significant difference in the prevalence of OSA, seizure type and number of antiepileptic drugs.[25] CPAP treatment was thought to reduce seizures.[25] In our study, no association was found between the anti-seizure medications used and the presence of sleep apnoea. The number of patients in our study receiving polytherapy was quite low, with the majority receiving monotherapy. Since our rate of refractory epilepsy patients was low, a relationship between polypharmacy and was not detected.

Sleep apnoea syndrome can worsen the control of seizures, mood disorders, cognitive dysfunction, daytime sleepiness and quality of life by worsening the clinical picture of epilepsy patients.[7] Monoaminergic neurons, including serotonin, norepinephrine and orexin, play important roles in respiratory function and have anti-seizure properties. Levels of monoaminergic neurons activities decrease throughout the night and decrease further during sleep.[4] Sudden unexpected death in epilepsy is the leading cause of death in patients with refractory epilepsy, and respiratory dysfunction may be a contributing factor.[4] It has also been stated that hypoxemia due to apnoea may be associated with seizures.[7] In one study, 74.9% (296 of 395) of epilepsy patients who underwent full-night PSG testing were diagnosed with OSA.[26] In another study, a higher prevalence of OSAS was not observed in patients compared to controls (19.1% vs. 14.5%, respectively), and although moderate to severe forms of OSAS were found to be more common in epileptics compared to controls, it was not considered significant due to the small sample size.[7] In our study, sleep apnoea was seen in 50%

Table 4: Summary of number of medications, STOP-BANG score with regard to sleep apnoea

	Sleep apnoea		p
	No, AHI<5	Yes, AHI≥5	
Number of medications			
One	18 (75.0%)	17 (70.8%)	1.000
Two	5 (20.8%)	6 (25.0%)	
Three	1 (4.2%)	0 (0.0%)	
Four	0 (0.0%)	1 (4.2%)	
STOP-BANG score	2 (1 - 3)	3 (2 - 4)	**0.021**
Low	34 (69.4%)	7 (36.8%)	
Intermediate	11 (22.4%)	8 (42.1%)	**0.038**
High	4 (8.2%)	4 (21.1%)	

Data are given as median (1st quartile - 3rd quartile) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables. AHI: Apnea-hypopnea index, STOP-BANG: STOP-BANG Sleep Apnea Questionnaire

of epilepsy patients.

Ictal respiratory changes are a common clinical phenomenon that is more likely to occur in focal epilepsies.[27] In a study using scales that determine the risk of sleep apnoea, when extratemporal lobe epilepsy was compared with temporal lobe epilepsy, it was found that those with temporal lobe epilepsy had a higher risk of OSA.[8] The rate of people diagnosed with sleep apnoea was 40.9 in the generalised group and 57.7 in the focal epilepsy group. Although there was more sleep apnoea in the focal group, no statistically significant difference was detected between the groups. This may be related to the fact that the majority of our focal epilepsy group consisted of patients diagnosed with occipital lobe epilepsy and that we had few patients with temporal lobe epilepsy.

Waking up after sleep onset is an important feature that can be seen in epilepsy patients.[13,28-31] There was no difference between our groups in terms of wakefulness after sleep onset and the arousal index.

Our study has several limitations. Most of our focal epilepsy group had occipital lobe epilepsy, with few having temporal lobe epilepsy. Additionally, the number of patients resistant to treatment was low, and our study was retrospective.

In conclusion, we detected sleep apnoea in 50% of epilepsy patients. There was no significant difference between the generalized and focal epilepsy group. The STOP–BANG score was higher in patients with sleep apnoea. Thus, when polysomnography is not possible, evaluation can be made with the STOP–BANG test.

DISCLOSURE

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Conflict of interest: None

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