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Research Article

Polysomnographic (Examination) Evaluation and Cap Scoring of Patients With Continuous Spikes and Waves During Slow Sleep Syndrome

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Summary

Objective: In our study, we aimed to demonstrate presence of sleep disorder which may associate with Contınuous Spike and Waves during Slow Sleep Syndrome (CSWS) clinical picture and to define changes in sleep structure of those patients by recording polysomnographs and full night sleep electroencephalogram (EEG) in our patients.
Methods: The study population included totally 10 patients (7 boys and 3 girls) with diagnosis of followed by Pediatric Neurology Department, Uludag University and a control group consisted of 10 healthy individuals with similar age and sex distribution.
Results: It is observed that there is no statistical difference between CSWS patients and control group in terms of total sleep time, Non-REM 1 (Stage-1) ,Non-REM 2 (Stage-2) and Non-REM 3+ Non-REM 4(Stage-3,Stage-4). It was found that REM period was shorter and the difference was statistically significant ($p<0.05$). It was observed that 60 % of patients with CSWS had non-scored epochs. Mean Cyclic alternating pattern (CAP) value in patients with CSWS was 45 % and it was 25.8 % in control group and it was found to be statistically significant in patients. ($p<0,001$).
Conclusions: We found that sleep EEG records of patients with CSWS were more pathologic than that of control group and that integrity of sleep is deteriorated in patient group. However, we could not find any pathology related with primary sleep disorder.We believe that this study may clear up a way for future studies which may make contributions.

Key words: Continuous Spikes and Waves During Slow Sleep Syndrome (CSWS), Polysomnography, Cyclic alternating pattern (CAP)

Yavaş Uykuda Elektriksel Status Epileptikusu Olan Hastalarda Cap Skorlaması ve Polisomnografik Değerlendirme

Özet

Amaç: Çalışmamızda Yavaş uykuda Elektriksel Status Epileptikus (ESES=CSWS)'lu hastaların uyku yapısındaki değişiklikleri tanımlamak, eşlik edebilecek uyku bozukluğunu gözlemlemek amacıyla tüm gece polisomnografi kaydı yapıldı.
Yöntem: Çalışmaya Uludağ Üniversitesi Pediatri-Nöroloji Bölümümde Yavaş uykuda Elektriksel Status Epileptikus tanısıyla takip edilen 10 hasta (7'si erkek, 3'ü kız) ve benzer yaş ve cinsiyet dağılımına sahip sağlıklı 10 kontrol olgu alındı.
Bulgular: CSWS' li hastaların kontrol grubuyla yapılan istatistiksel analizlerinde total uyku süresi, evre-1, evre-2 ve evre-3+ evre-4 uyku oranlarında bir farklılık olmadığı gözlendi. Uykunun REM dönemi ile ilgili yapılan çalışmalarda CSWS' li hastalarda kontrol grubuna göre daha kısa zaman olduğu istatistiksel olarak anlamlı bulundu.($p<0.05$) CSWS'li hastaların % 60'ında skorlanamayan epokların olduğu gözlendi. CSWS hastalarda ortalama CAP değeri% 45 ve kontrol grubunda% 25.8 idi ve istatistiksel olarak anlamlı bulundu. ($p <0,001$).

Sonuç: CSWS hastaların uyku EEG kayıtları uyku o bütünlüğü açısından kontrol grubuna göre bozulmuş olduğu bulundu. Ancak, patolojiye yönelik eşlik eden bir uyku bozukluğu gözlemlemedik. Biz bu çalışma ile gelecekte yapılabilecek diğer çalışmalara katkıda bulunabileceğimize inanıyoruz.

Anahtar Kelimeler: Yavaş Uykuda Elektriksel Status Epileptikus (CSWS=ESES), Polisomnografi, Siklik Alternan Patern

INTRODUCTION

Continuous Spikes and Waves During Slow Sleep Syndrome (CSWS) is an age dependent epileptic encephalopathy combined with motor and cognitive impairment[39]. The diagnostic criteria for syndrome consisted of continuous or near-continuous focal or generalized spike–and-wave discharges in at least 85% of non-rapid eye movement sleep, whether or not clinical seizures simultaneously, and persisting on 3 or more records over a period of at least 1 month[20]. However, more recent definition recognized by ILAE is epileptiform discharges with significant activation during sleep associated with favorable clinical findings[30]. Although CSWS is an age-related and self-limiting disorder, neuropsychiatric impairments may be permanent[34]. For final diagnosis of, electroencephalographic recording including the awake-sleep period is necessary[5,31].

Sleep and sleep deprivation both affect the occurrence of seizures and also the frequency of epileptic discharges during Non-Rapid Eye Movement (NREM) sleep. NREM sleep has been shown to activate interictal epileptic activity in both partial and primary generalized epilepsy syndromes, while Rapid Eye Movement (REM) sleep has a contrary effects[8,11]. However, epilepsy makes changes in the structure and organization of sleep. On the other hand, epileptic patients may sleep related conditions such as sleep- related (disordered) breathing, and/or movement disorders. (respiratory disorder during sleep, periodic limb movement disorder). Therefore, those disorders may give rise to increase in the frequency of seizures[2].

Polysomnography is a golden standard method used for determining sleep disorders which may associate with epilepsy. It is highly valuable for demonstrating the structure of sleep[1]. Moreover, there are also studies conducted on epileptic patients about cyclic alternating pattern (CAP), which provide information about microstructure of sleep[21,32]. CAP is an EEG activity that may indicate sleep instability, sleep disturbance, or both. CAP can appear spontaneously in NREM sleep, but it can also occur in association with identifiable sleep pathophysiologies such as sleep-disordered breathing and periodic leg movement activity[40].

No polysomnographic studies have been performed in children with before. One of the study notes that 10% of children with CSWS have absence of sleep spindles. The objective of the present study was to evaluate the macro and microstructure of sleep of 10 patients with CSWS syndrome and to demonstrate the presence of possible sleep disorders[5].

MATERIAL AND METHODS

The study population included totally 10 patients (7 boys and 3 girls) with diagnosis of CSWS followed by Pediatric Neurology Department, Uludag University and a control group consisted of 10 healthy individuals with similar age and sex distribution. The study was approved by Ethical Committee of Uludag University and informed consent was obtained from the legal caretaker of each child. Children were studied in the Sleep Laboratory of Neurology Department in a dark, quiet room where one of their parents accompanied the child throughout all night. All of the patients continued their

current antiepileptic drugs and sleep recordings performed without sleep deprivation. All overnight polysomnography data were digitally recorded (Grass Telefactor, AS 40 Amplifier system). The electroencephalogram (EEG) was recorded using 10 scalp electrodes (Fp1, Fp2, F3, F4, F7, F8, T3, T4, T5 ,T6, C3, C4, P3, P4 , O1, O2) on the contrary to standard polysomnography. Two channel electro-oculogram (EOG), submental and anterior tibial muscle electromyogram (EMG) and electrocardiogram (ECG) electrodes were used. Thermistor (oronasal airflow) and pulse oximeter were also present. Movements of chest and abdomen were measured by body sensors. Using an infrared video camera, children were monitored by a sleep technician who recorded behaviour and other comments. Children were let to go to sleep in 22:00 and they were waken up at 7:00 in the morning. After they were waken up, 20-minute awake EEG recordings were obtained (Figure 1). In all children, sleep scoring was made on the basis of sleep classification according to Rechtschaffen and Kales standard criteria of 30-second epochs. Patients were diagnosed on the basis of "International Sleep Disorders Classification" criteria[18]. The following conventional sleep parameters were measured:

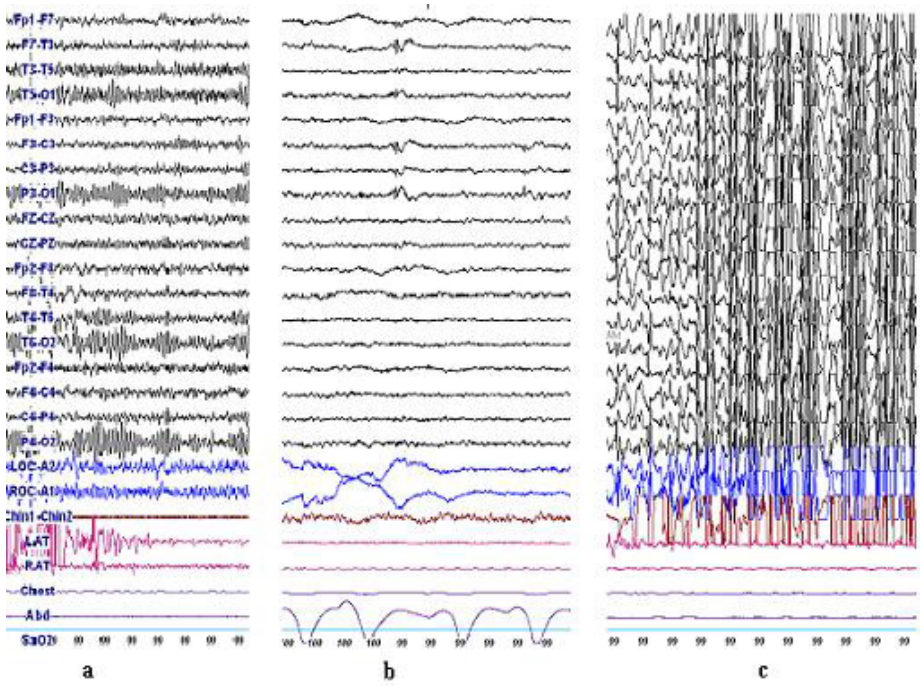

Figure 1: *Polysomnographic recording. (a) Wakefulness; (b) R sleep; (c) N sleep; continuous spike-and-wave discharge*

-Total sleep time (TST): the time from sleep onset to the end of the final sleep epoch minus time awake;

-Percentage of total sleep time spent in sleep NREM [Non-REM 1 (Stage-1) %], N2 [Non-REM 2 (Stage-2) %), slow wave sleep [Non-REM 3, Non-REM 4 (Stage-3+Stage-4 %) and REM sleep (REM %)

-Sleep Latency (SL): time from lights out to sleep onset, defined as the first of two consecutive epochs of sleep stage 1 or one epoch of any other stages in minutes.

The undetermined sleep stages due to intensive epileptic activity were defined as "non-scored epoch".

Scoring of respiratory variables was performed on basis of as standart set by American Thoracic Society and previously published data on children[22,26]. The following definitions were used to identify the respiratory problems.

- Apnea (Obstructive): Absence of airflow (as detected by the oronasal thermistor) for at least two respiratory cycles, associated with paradoxical movement of the chest and abdomen.

-Hypopnea: Decrease of 50% or more in oronasal thermistor signal and a concurrent arousal and/or fall 3% or more in SpO_2 from baseline levels.

-Apnea-Hypopnea Index: For each type of respiratory event, an index was calculated that presented the number of events per hour of sleep.

- Periodic limb movements in sleep (PLMS): PLMS were scored if they were part of a series of four or more consecutive leg movements lasting 0.5-5 sec, with an intermovement interval of 5-90 sec[3]. The PLMS index was the number of periodic limb movements (PLMs) per hour of sleep.

Cycling Alternating Pattern (CAP);

Cyclic alternating pattern (CAP) is a periodic EEG activity of NREM sleep characterized by repeated spontaneous sequences of transient events, recurring at intervals up to 2 min long. CAP is a periodical activity lasting between 2 and 60 seconds and it is consisted of A and B phases. A and B phases are consecutive. CAP appears throughout 1-2-3-4 stages of NREM sleep. It is discriminated from ground rhythm by sudden frequency and amplitude changes. When compared with B phase, A phase is composed of slower, higher-voltage rhythms, faster lower-voltage rhythms, or by mixed patterns including both. EEG patterns observed in A phase can be discriminated as delta bursts, vertex sharp waves, K complexes, polyphasic bursts, K alpha and EEG arousals. Non-CAP defines non-CAP period over 60 seconds[40].

CAP was scored according to the as criteria published in Sleep Medicine 2002.

The following CAP parameters were measured;

-CAP (time sleep in NREM) duration,

-CAP rate (percentage of total NREM sleep time occupied by CAP sequences)(Figure 2)

-Number and duration of CAP cycles.

-Number duration and percentage of A phase,

-Number duration and percentage of B phase.

During scoring phases with undermined sleep stages due to intensive epileptic activity were defined as '' non-scored epoch"(Figure 3). CAP was scored according to the as criteria published in Sleep Medicine 2002[40]. CAP is a periodic EEG activity of NREM sleep characterized by repeated spontaneous sequences of transient events (phase A), recurring at intervals up to 2 min long. The return to background activity identifies the interval that as seperates the repetitive elements (phase B). This is because the CAP procedure is based on the succession of complete CAP cycles (phase A + phase B).

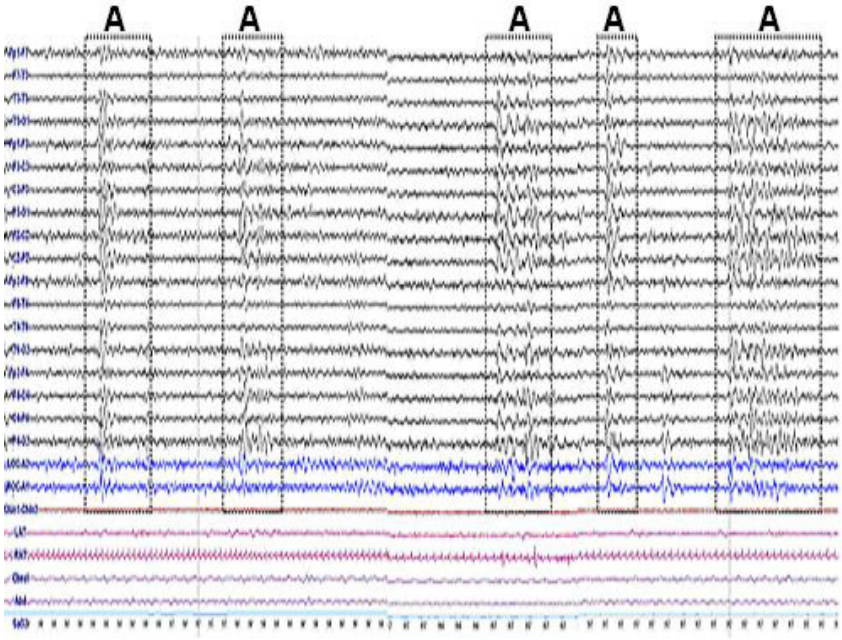

Figure 2: *The CAP sequence (phase A is marked by black spot boxes)*

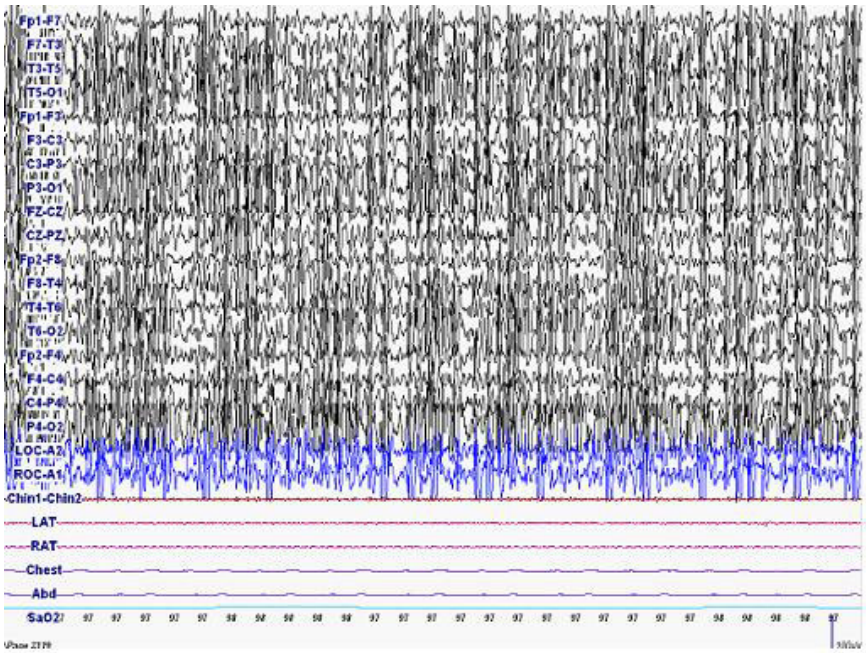

Figure 3: *An example of non-scoring epoch sleep stage of CSWS patient*

Pediatric Sleep Questionnaire

A questionnaire designed for the study of sleep characteristics of children was filled out by the parents. The sleep questionnaire consisted of 45 items with values of 1-5 (1=never, 2=occasionally, 3=often, 4=very often, 5=always) to find out the problems about sleep disorders such as disorders of initiating and maintaining sleep, parasomnias, night waking, movement

disorders during sleep, sleep breathing problems[4].

Pediatric Daytime Sleepiness Scale

Each of patients were undergone Pediatric Daytime Sleepiness Scale-PDSS-31. The PDSS is an 8-item questionnaire for evaluating the relationship between daytime sleepiness and school-related outcomes. Each question is rated on a 4-point scale (0=never, 1=seldom, 2=sometimes, 3=frequently, 4=always), with higher scores indicating more sleepiness[10]. Questions were more frequently answered by mothers of patients.

Statistical analysis

Statistical analyses of data were performed by SPSS13.0 statistical software package. It is examined by Shapiro-Wilk test whether data has normal distribution or not. For data with normal distribution, t-test was used from comparison of two groups and for data without normal distribution, Mann-Whitney U test coefficient was used for comparison of two groups. In analysis of categorical data, Fisher's Chi-Square test was used. Significance level was determined as $\alpha<0.05$.

RESULTS

The age range of 10 patients was between 3.5 and 12 years (mean 7.46±1.98 years.) The ratio of male/female was 7/3. Physical and neurologic examinations of 8 of the 10 patients were normal (Table-1a). 2 patients were not mentally retarded. Demographic and clinical findings of control group are given in Table 1-b. None of the children had documented seizures during the overnight polysomnogram study. Polysomnography results of CSWS patients are given in Table 2, while results of control group are given in Table 3. It is observed that there is no statistical difference between patients and control group in terms of TST, Stage-1, Stage-2 and Stage-3+Stage-4. Considering studies conducted on REM stage of sleep, In patients with it was found that REM period was shorter and the difference was statistically significant when patients with CSWS and control group was compared ($p<0.05$) Table 4. It was observed that 60 % of patients with CSWS had non-scored epochs. We revealed several findings that indicated presence of electrophysiological seizures with various degrees observed by means and occurred during sleep. For 20-minute wakefulness records obtained at the morning of full night sleep, In the awake state, was observed that intense discharges appeared in NREM sleep were significantly decreased and they were even absent in three patients. However, no pathologies related to other primary sleep disorders (sleep respiratory disorder, periodical extremity movement disorder, parasomnia etc) could not be observed. In Pediatric Sleep questionnaire, two patients had difficulties in falling asleep and in continuity of sleep as well as one patient had complaint of snoring. Mean PDSS of patients with were measured as 2.

According to the sleep questionnaire related to night awakenings, periodic limb movement disorder or parasomnias were as reported by the parents. PDSS mean score of patients with was maintaining sleep.

The assessment of sleep microstructure as relevated that patients with had differences from controls regarding CAP parameters. CAP rates were, higher than the controls, 45%, 25% respectively.

Mean CAP value in patients with CSWS was 45 % and it was 25.8 % in control group and it was found to be statistically significant in patients. ($p<0,001$). Moreover, in intra-group comparison of patients with and control group – patients comparisons, CAP was found to be statistically significant against phase A and B ($p<0,001$) (Table 5).

Table 1-a. Demographic and clinical findings in patients with CSWS

Cases	Sex	Age,year	Seizure (Age at onset,year)	Seizure/Type	Seizure /Freq	Treatment	Neurological Findings
1	M	3.5	1	Atonic, GTCS	4-5/mount	VA, TPM	Normal
2	M	5	2.5	PMS, GTCS	2-3/mount	VA, TPM	Bilateral pyramidal sign, microcephaly
3	M	5.5	1	GTCS,PMS	2-3/mount	VA, LEV	Normal
4	F	10	4	Atonic, PMS	5-6/mount	VA, TPM	Normal
5	F	9	2	GTCS	1-2/mount	FB, CBZ	Normal
6	F	12	3	PMS	2-3/mount	VA,FB	Normal
7	M	7	1.5	PMS	1-2/mount	VA,LEV	Normal
8	M	5.5	2	Atonic, PMS	3-4/mount	FB, CBZ	Bilateral pyramidal sign, microcephaly
9	M	11	3	PMS,GTCS	1-2/mount	CBZ,LEV	Normal
10	M	8	1.5	GTCS	2-3/mount	VA, LEV	Normal

Abbreviations:
F =Female
M = Male
Freq = Frequency
GTCS = Generalized tonic-clonic seizures
PMS = Partial motor seizures
FB =Phenobarbital
VA = Valproic asid
CBZ = Carbamazepin
LEV = Levetirasetam
TPM =Topiramate

Table 1-b. Demographic and clinical findings of control group.

Cases	Sex	Age, year	Neurological Findings
1	F	5	Normal
2	F	6	Normal
3	F	7	Normal
4	M	9	Normal
5	M	7	Normal
6	M	10	Normal
7	M	8	Normal
8	M	4	Normal
9	M	9	Normal
10	M	12	Normal

Table 2. Polysomnography Results of CSWS Patients

Patient	TST (min.)	S1 %	S2 %	S3+S4 %	R %	Non-scored (min.)	Sleep onset (min.)	Apnea-Hipopne Index	Periodic Leg Movements	CAP %
1	284	2.5	29.6	66.9	1	193	11.5	0	0	56
2	368.3	4.1	34.3	48	13.6	0	4	0	0	48
3	459.1	2.5	29.8	58.2	9.5	36	5.5	0	0	40
4	488.4	3.4	38.8	54.4	3.5	26	3	0	0	43
5	441.8	13.1	59.1	27.8	0	0	2	0	0	42
6	177.2	2.3	40.1	57.6	0	256	19	0	0	54
7	290.2	3.3	33	57.1	6.6	227	6	0	0	49
8	468	3.4	11.6	81.3	3.6	0	5.5	0	0	39
9	221.2	1	13.9	85.3	0	215	3	0	0	46
10	409.5	8.2	28.3	47.9	15.6	0	6	0	0	50

Abbreviations:
TST: Total sleep time
S1: Non-REM sleep stage 1
S2: Non-REM sleep stage 2
S3+S4: Non-REM sleep stage 3 + Non-REM sleep stage 4
R: REM sleep stage
CAP: cyclic alternating pattern

Table 3. Polysomnography Results of Control Group

Control	TST (min.)	S1 %	S2 %	S3+S4 %	R %	Non-scored (min.)	Sleep onset (min.)	Apnea-Hipopne Index	Periodic Leg Movements	CAP %
1	324.1	6.5	52.2	37.5	3.9	0	3	0	0	33
2	427	5.5	17.2	63.9	13.3	0	4	0	0	24
3	450	9.8	21	47	22.2	0	5	0	0	26
4	389.4	8.3	31.1	39.8	20.8	0	7	0	0	28
5	454	10.7	47.7	37.6	4	0	5	0	0	30
6	298.9	3	34	48	15	0	2	0	0	21
7	345.8	3.4	29.8	56.9	9.9	0	4	0	0	22
8	438.1	5	28.1	58.3	8.6	0	5.5	0	0	20
9	398	4	27.8	48.4	19.8	0	4	0	0	26
10	411.9	6	31.6	40.7	21.7	0	5	0	0	28

Table 4. The Duration of Sleep Stages in Patient and Control Groups

Groups	S1	S2	S3+S4	REM
	Mean ± S.D.	Mean ± S.D.	Mean ± S.D.	Mean ± S.D.
Patient n=10	4,38 ± 3,6	31,85 ± 13,4	58,5 ± 16,64	5,34 ± 5,8
Control n=10	6,22 ± 2,6	32,05 ± 10,7	47,8 ± 9,3	13,84 ± 7,03
p value	p=0.096	p=0.097	p=0.096	p=0.03

Table 5. CAP Scores in Patient and Control Groups

Groups	CAP	CAP A	CAP B
	Mean ± S.D.	Mean ± S.D.	Mean ± S.D.
Patient n=10	46,7 ± 5,76	67,8 ± 6,66	32,2 ± 6,66
Control n=10	25,8 ± 4,13	59,5 ± 6,7	41,5 ± 8,2
p value	p=0.001	p=0.015	p=0.015

Abbreviations:
CAP: Cyclic alternating pattern
CAP A: Cyclic alternating pattern phase A
CAP B: Cyclic alternating pattern phase B

DISCUSSION

It is already known cortical as exitability and synchronization must be present for epileptogenesis. Although sleep disorders are frequent, children with epilepsy may be at increased risk, for both biological and social reasons. Epileptiform discharges may be activated by sleep[9], epilepsy[28] and antiepileptic drugs[19] may alter sleep architecture, leading to daytime somnolence.

Generally, interictal spikes increase at the beginning of sleep, reach its maximum during deep N phases and return to a level

of slightly lower than that in wakefulness during REM, and this leads to the activation of epileptiform discharges[33]. Sleep spindles and slow waves observed during NREM stage of sleep occur due to the synchronization[14,32].

Due to unique the as spesific changes of NREM period, it play a significant role in triggering epileptic neurons[24]. Another significant aspect of relation between sleep and epilepsy is that thalamic and cortical physiological as mechanisms underlies sleep spindles among electrophysiological patterns and spike-wave discharges among epileptic patterns[13].

Most of experimental and clinical studies had shown that many epileptic paroxysmal events occur during slow-wave sleep[35]. Epileptic activity may be observed during REM period of sleep (9 %), but it is very low in comparison with that of NREM period (41 %)[8]. When patients with CSWS fall asleep, bilateral and widespread slow waves appear. As sleep spindles and K-complexes are not distinguished, NREM cannot be discriminated differentiated. In our study, it was also observed that 60 % sleep phase on-scored epochs were rate present. In REM phase, bioelectrical status appearance in EEG disappears; should it be more frequent in frontal region, widespread spike waves rarely occur. They significantly decrease in REM sleep and wakefulness. Following full wakefulness, this electronic status generally disappears. In records obtained after wakefulness of our patients, significant decrease in pathological waves was observed[35,38]. It is known that epileptic patients have sleep disorders with different symptoms and at various degrees[41]. As as a result of studies conducted on children and adolescents, incidence of sleep respiratory disorder was reported to range between 1 and 3 percent[29]. In our study, although a patient had complaint of snoring, no sleep respiratory disorder could be found including control group.

Particularly language problems and memory disorders, behavioral and psychiatric disorders are experienced by patients with CSWS. Decrease in sleep efficiency, frequent wakening, fragmented sleep and slow-wave sleep as well as decrease in REM sleep were reported in polysomnographic studies conducted on children and adults describing short-term memory deficits. Both epilepsy and antiepileptic medication can cause as these clinical conditions[23].

In the present study, children with epilepsy with nighttime arousal difficulties were found to have reduced REM sleep . Compared with for control children, the proportion of REM in our study sample was reduced from 13.84 to 5.34% of total sleep time[15]. In our study, REM sleep was found to statistically decrease in comparison with that of control group ($p<0.05$).

In addition, sleep abnormalities have been associated with the increase in frequency of epileptiform discharges and the reduction of seizure thereshold[7].

Although sleep disorders are more prevalent in pediatric epilepsy, surprisingly few studies have been reported in this area. Cortesi et al showed that children with idiopathic epilepsy had significantly more sleep problems than controls[6]. In another study which compared 79 schoolchildren with epilepsy with healthy controls, higher rates of sleep disorders were seen in epileptic children[37]. To our as knowledge, this present study represents the first attempt to evaluate sleep patterns of children with CSWS in regards of macro and microstructure of sleep as encountered during PSG.

In the largest study to date assessing sleep in Angelman Syndrome, Walz et al.studied 339 individuals, assessing sleep patterns using a standardized measure derived from the Internatıonal Classification of Sleep Disorders. More than half of their sample displayed a variety of abnormal sleep patterns, with more than 40% of the

respondents describing problems with sleep initiation and duration. In the prior study, difficulty with sleep initiation (48%) and a decresad need for sleep (42%) were the most frequently reported sleep abnormalities, and these were also the most frequently reported sleep in our study with similar rates (sleep initiation, 48%; decreased need for sleep, 49%)[42].

İt is stil unclear as to whether more severe epilepsies are causing the sleep disturbances or if poor sleep hygiene is exacerbating the epilepsies.

These presence of behavioral problems in children with epilepsy has been linked to abnormal sleep patterns[37]. Daytime behavioral complaints reported in children with epilepsy may also be due in part to prolonged subclinical arousals and disturbed arousal mechanisms during sleep, resulting in sleep fragmentation and poor quality of sleep[43]. Epilepsy appears to have important secondary effects on both the quality and the architecture of sleep[17]. Alterations in total sleep time and percentage of time in sleep stages, sleep latency and spontaneous awakenings have been reported in children with epilepsy[36]. Epileptic discharges even in the absence of seizures, may be cause sleep disruption, preventing normal progression through sleep stages.

Sleep disorders may also play a role in some of the cognitive problems reported in children with epilepsy, and therefore, a more extensive neuropsychological battery focused on problems of inattention, as well as a larger sample size, would also provide further means of evaluation.

CAP is a physiological periodic EEG rhythm which is examined within terms of arousal during sleep and it occurs in approximately 25-45 % of sleep. Increase in percent of CAP within sleep reflects deterioration of sleep stability. However, it is known that many pathologies pertaining to sleep occur during phase A of CAP[25]. In previous studies, seizures and interictal epileptiform discharges are known to occur more easily particularly in Stage-1 and Stage-2 among sleep stages, whereas it may be speculated that REM stage is a rather difficult stage for secondary generalization[27]. In general, CAP had shown that several stages of sleep are efficient for occurrence of epileptiform discharges[16,24].

Studies conducted on CAP had a recent increase and they provide us information about microstructure of sleep. As it may be regarded as a disadvantage that most studies were conducted on patients with epilepsy, a homogenous group could not be formed and number of cases were not high enough, those deficits will disappear as number of studies increases. In a study, Eisensehr et al evaluated 10 Lennox Gestaut Syndrome patients, examined epileptic changes, their relation with CAP in whole night polysomnographs and observed an increased activity in phase A of CAP in comparison with that of control group[12]. In a study conducted by Parrino et al, where researchers examined cases with primary generalized epilepsy and frontotemporal focus lesion, it was shown that interictal discharges increased in phase A of CAP and Phase B of CAP had a strong inhibition effect[19]. Similarly, in another study where 56 patients with nocturnal partial seizure were examined, it was reported that seizures were concentrated during NREM period and there was an increase in phase A of CAP. Moreover, in same study, they concluded that fragmented sleep observed in epilepsy patients triggered seizures and increase in awake period deteriorated integrity of sleep[25].

In our study, CAP and Phase A of CAP in patients with was found significantly different than that of control group that this finding is compatible with literature.

CONCLUSION

CSWS is a group of patients characterized by observation of epileptic activity during sleep. In this study, we aimed to define whether primary sleep disorder in general

population is associated with and to examine variability of sleep stages throughout night.

We found that sleep EEG records of patients with CSWS were more pathologic than that of control group and that integrity of sleep is deteriorated in patient group. However, we could not find any pathology related with primary sleep disorder. According to the extent of our investigations in the literature, this study is one of a few study in terms of assessing patients with CSWS by polysomnographic method. We believe that this study may clear up a way for future studies which may make contributions.

In conclusion, children with epilepsy have greater sleep problems than their non-epileptic sibling. Questions about disturbed sleep should be part of the routine evalation of children with epilepsy. Further studies are needed to clarify the etiology of the sleep disturbances in children with epilepsy. These should include physiological measures (polysomnography) to characterized sleep architecture and document sleep-disordered breathing and assessment of child behavior and parental discipline sytle.

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