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REVIEW

Childhood epilepsy and sleep

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Abstract

Sleep and epilepsy are two well recognized conditions that interact with each other in a complex bi-directional way. Some types of epilepsies have increased activity during sleep disturbing it; while sleep deprivation aggravates epilepsy due to decreased seizure threshold. Epilepsy can deteriorate the sleep-related disorders and at the same time; the parasomnias can worsen the epilepsy. The secretion of sleep-related hormones can also be affected by the occurrence of seizures and supplementation of epileptic patients with some of these sleep-related hormones may have a beneficial role in controlling epilepsy.

Key words: Nocturnal frontal lobe epilepsy; Sleep; Parasomnias; Sleep-related hormones

Core tip: The relation of sleep and epilepsy was discovered many centuries ago. Some types of epilepsies have increased activity during sleep disturbing it; while sleep deprivation aggravates epilepsy. Both are integrating together; so that controlling epilepsy will improve the sleeping quality while consolidating sleep will ease controlling the seizures. Meticulous care of sleep pattern and quality in epileptic children has significant effects for diagnosis, efficacy of controlling seizure activity, and health-related quality of life. Adequate studying of the sleep related disorders and hormones may give a clue for new methods of better epilepsy control.

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INTRODUCTION

Epilepsy is a phenomenon of recurring seizures. A seizure is a result of an increased electrical activity in the brain which causes sudden change in the behavior of the affected person. One third of epileptic patients have seizures during the sleep. On the other hand; sleep disorders are more prevalent in epileptic children and they are common associated epilepsy co-morbidities[1]. This is due to the strong link between the physiology of sleep state and the principal pathological mechanisms of the epileptic seizures[2]. The possible association of epilepsy with sleep was known since long time. Description of episodes of epileptic seizures occurring during sleep was found in the extant writings of both Aristotle and Hippocrates. However, this interactive double-way effect shared between sleep and epilepsy was revealed only by the end of the 19th century by Gower who was interested by the effect of sleep/awakens cycle on generalized tonic-clonic epilepsy[3].

Over the last twenty years, there has been a vast growth in the awareness about the inherent relationship between sleep and epilepsy especially with the wide use of polysomnography and video electroencephalogram (EEG) monitoring. This complex relationship and interaction between sleep and epilepsy was found to be interrelated. This means that epilepsy disturbs sleep and sleep deprivation may aggravate epilepsy due to the decrease in seizure threshold thus forming a vicious circle[4]. About 45% of patients with medically refractory epilepsy experienced excess daytime sleepiness. However, presence of sleep fragmentation without a history of seizures or

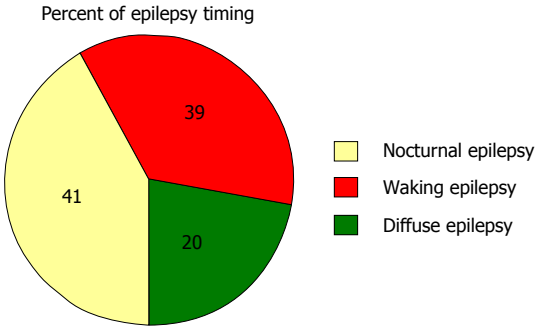

Figure 1 The ratio of the different epilepsy timing.

antiepileptic medication is suggesting that sleep disintegrity may be an intrinsic component of certain types of epilepsy[5].

EFFECT OF SLEEP ON EPILEPSY

Epileptic electrical activities may appear only during sleep

There is a considerable difference in the amount of baseline brain rhythmicity between the states of sleep and wakefulness which could explain that different seizure varieties begin specially in certain sleep states[6]. Almost two-thirds of seizures happen between 8:00 PM and 8:00 AM and about 20% of seizures occurred during sleep (nocturnal epilepsy), 41% occurs during the day (waking epilepsy) and the rest occur during day or night (Figure 1).

Nocturnal seizures most often occur in the early morning near the end of the sleep period (5 AM to 6 AM) and less frequently 1 to 2 h after sleep onset, while diurnal seizures frequently occur in the early morning and late afternoon[7]. Pavlova *et al*[8] showed that frontal lobe seizures occurred more frequently between 12 midnight and 12 midday while temporal lobe seizures, occurred more often between 12 midday and 12 midnight.

Sleep consists of repetitive cycles each lasting about 90 min, advancing through non-rapid eye movement (NREM) stages to rapid eye movement (REM). These two neurophysiological conditions that distinguish sleep (NREM and REM) exert contradictory effects on interictal state and clinical conditions. Many researches showed occurrence of generalized discharges and clinical seizures mainly in NREM sleep. This is the reason that NREM may be considered as a natural "epileptogenic agent"[9]. NREM sleep can enhance interictal epileptiform discharges in both partial and generalized seizures while REM sleep limits spread of epileptic discharges outside the area that started seizure activity and allows the localization of the primary epileptogenic area as seen in temporal lobe seizures. The seizure activating role of NREM sleep has been attributed to increased neuronal synchronisation within thalamo-cortical projection neurons with robust activation of epileptic ictal and interictal activity. Most sleep-related seizures start during sleep stage Ⅱ. Meanwhile, few seizures occur during stages of slow wave sleep (SWS) (stages Ⅲ/Ⅳ) and fewer or none occur during stage of REM sleep. Seizures originating from frontal lobe start during sleep more often than that arise from temporal lobe, a finding that is of clinical significance[5,10-12].

The seizures epileptic activities increased during sleep were illustrated in a number of epileptic conditions and distinguished by uncommon clinical seizures but with significant cognitive impairment. The syndrome of continuous spike-wave discharges during sleep (CSWS) is a typical example of such epileptic conditions. Other sleep augmented epilepsies include benign focal epilepsy of childhood with centro-temporal spikes, Lennox Gastaut syndrome and frontal lobe epilepsies (either supplementary sensorimotor area (SSMA) or autosomal dominant nocturnal frontal lobe epilepsies). Other epilepsies have a tendency to occur upon awakening like Juvenile myoclonic epilepsy; petite male epilepsy; and epilepsy with grand mal seizures on awakening[5]. There are other neurological disorders seen in children that are associated with sleep-activated EEGs. These disorders include Landau-Kleffner syndrome (LKS); pervasive developmental disorder with regression; childhood disintegrative disorder; congenital aphasia (developmental language disorder); and transient cognitive impairment[13].

The epileptic condition termed as CSWS describes clinical epileptic syndromes seen within a condition called electrical status epilepticus in sleep (ESES). In this condition, there is a characteristic electroencephalographic pattern with significant activation and increase in epileptiform discharges during sleep. Some cases of LKS may present only with electrographic seizures, together with the unique ESES which could represent some overlap between CSWS and LKS. However, children with the condition of CSWS who have more global regression will suffer from more challenging epilepsy and the focus of the electric epileptic activity located frequently in fronto-temporal or frontocentral areas. On the contrary, children with LKS develop an acquired aural agnosia, less seizures, and their foci of electric epileptic activity located in the posterotemporal areas. Occurrence of ESES needs a high level of clinical attention and suspicion because slow-wave sleep should be documented and recorded to confirm this condition. Severity of ESES can differ over time in the same patient or differ from patient to another and clinical condition does not necessarily show direct correlation with spike wave index in all the times. However, the prognosis of LKS is significantly superior to CSWS. Treatment of ESES is not only by controlling the seizures; but refinement and improvement of the continuous epileptiform discharge must be done to get good neuropsychological outcome[14,15].

Benign childhood epilepsy with centrotemporal spikes (BECTS) is a type of focal idiopathic epilepsy that is more common in childhood and was previously known as Rolandic epilepsy. It comprises three quarters of benign childhood partial epilepsies, and is characterised by striking ictal clinical manifestations and EEG abnormalities. The typical clinical symptoms include hemifacial convulsions which tend to generalize in sleep. The interictal

EEG shows normal base activity and epileptiform activity, characterized by high amplitude spikes mainly in the central or mid-temporal regions (centrotemporal spikes or rolandic spikes) which are exaggerated by drowsiness or sleep. There is no intellectual defect or cerebral lesion with good prognosis. The seizures usually remit by the age of 15 years[16]. The Sleep style is not notably impaired in BECTS; however; cyclic alternating pattern (CAP) studies show a decrease of NREM instability, mainly in sleep stage Ⅱ. This is most likely related to disturbance of the physiological synchronization mechanisms (needed for the generation of slow-wave components of CAP) by the centro-temporal spikes[17].

Frontal lobe epilepsy is another commonly occurring type of partial epilepsy which originates from the frontal lobe of brain. These seizures show a tendency to arise preferentially during sleep. This kind of epilepsy typically occurs in a cluster of short fits with a rapid start on and off. Some of the common symptoms of this disorder include sudden battering movements during sleep, with the head jerks to one side, and the upper limbs rise with it into a brief, frozen state[5]. Behavioral automatisms are common and include rocking, bicycle pedaling movements and repetitive hand movements. There are two unique main clinical types of epilepsy that originate from the frontal lobe: the first one is that originates from SSMA, and the second one is nocturnal frontal lobe epilepsy (NFLE). BECTS is considered by some authors as a type of frontal lobe epilepsy[18]. The SSMA epilepsy describes the epilepsy that originates from the SSMA located at the mesial aspect of the superior frontal gyrus (area 6) and extends to the dorsal aspect of the lobe convexity. The seizure activities occur principally during sleep. Seizures arising from SSMA characterized by being of brief duration (10-40 s) with occurrence of quick onset of asymmetric tonic posture affecting both extremities. Consciousness is often preserved despite that speech arrest and vocalization are common[13]. These seizures also tend to occur in groups and can be badly disabling. Interictal epileptic sharp waves are frequently found at the midline, with maximum activity at the vertex or just close to the midline in the fronto-central area. The EEG pattern during the fit is characterized by occurrence of a high amplitude transient slow or sharp wave at the vertex, followed by low amplitude fast activity or an electrodecremental style[19].

Autosomal dominant type of NFLE is a rare inherited form of epilepsy that occurs in families. Its onset can start in infants or in adults. It is clinically characterized by occurrence of groups of nocturnal motor seizures, which are usually repetitive in the same manner and of short duration (5 s to 5 min). The seizure may take the form of simple arousals from sleep or as surprising, dramatic and unusual attacks of hyperkinetic fits with tonic or dystonic features. Aura may occur in some individuals with this type. It is frequent to have maintained consciousness during the attacks. A few patients may have fits during the daytime[20]. EEG monitoring during the epileptic fits may be normal or may be fogged up by movement artifact. If present, the characteristic rhythmic pattern of the fits usually takes the form of sharp waves or rhythmic spikes with a rate of 8-11 Hz. There are some data suggesting that these seizures may be initiated with K-complexes[21].

Epileptic electrical activities affected by sleep

Presence of interictal electrical spike activity is one of the characteristic features of epilepsy[22]. Conventionally, sleep-deprivation induces sleep that can be used to trigger interictal electrical spike activity on scalp EEG or magnetoencephalography during evaluation of patients who will be subjected to epilepsy surgery[23]. Earlier researches proved the preferential occurrence of some seizures types during sleep and demonstrated the link between sleep and triggering of epileptic activity on EEG[24-26]. Seizures can be triggered by the synchronized NREM sleep while can be suppressed by desynchronized REM sleep. The association between epileptiform activity and NREM sleep is obviously revealed in the syndrome of continuous spikes in slow-wave sleep and the LKS. The sleep EEG is a helpful diagnostic tool of epilepsy. It also useful to localize the epileptic foci, as new epileptic foci can appear in sleep. REM sleep may aid to reveal the narrowest localization of the primary focus[5].

Previous researches also showed that children with different types of epilepsies (including focal epilepsy, LKS, and CSWS) had greater frequency and topographic field of interictal spike activity during sleep spindle than during wakefulness[27,28]. Earlier studies revealed presence of CAP between the excitatory A phase with K-complex and slow-waves during sleep (which could increase the frequency of interictal epileptic discharges) and the inhibitory B phase with low-voltage fast waves, together with sleep spindles alone[24,29]. However, Asano *et al*[30] showed that sleep could affect the overall frequency and severity of interictal spike frequency but not its topographic distribution. They proved that the topography of spike frequency was rather comparable between wakefulness and sleep with spindles in children who suffered from focal epilepsy. They assumed that sleep with spindles may lower the threshold of triggering the diffuse rather than focal activity of the interictal spike. They found also that both antiepileptic medication and postictal state may affect interictal spike frequency as the post-ictal period was associated with increased frequency of the interictal spike frequency but not simply after tapering antiepileptic medication[31].

EFFECT OF EPILEPSY ON SLEEP

Aadequate, sufficient and good healthy quality of sleep is an integral and important but unfortunately frequently missed component of general health. It is predominantly vital to the proper management of epileptic patients. Ineffective or inadequate sleep is common in epilepsy patients. Their sleep could be disturbed by natures of their seizures, presence of coexisting sleep disorders and

sometimes by seizure medications. The epilepsy may affect all the sleep parameters including night time difficulties, and parent/child interaction during the night. It also can induce sleep fragmentation, parasomnias, and daytime drowsiness. This can result in memory dysfunction; and considerable impairment of daytime functioning, and quality of life. The patients may experience daytime tiredness and impaired attention and arousal for years without knowing the reasons[6]. de Weerd *et al*[32] showed that the prevalence of sleep disturbance was twice more common in patients with partial epilepsy compared with controls and most parameters of sleep were extensively troubled. They had already significant impairment of the quality of life, and which is further compounded by sleep disturbances. Stores *et al*[33] assessed the sleep disturbances in 79 school children with epilepsy by parental questionnaires. They found higher incidence of sleep disorders in epileptic children (mainly poor quality sleep as well as anxieties about sleep) compared with normal controls. They also found associations between disturbed daytime behavior and sleep problems, mostly poor sleep quality among epileptic children aged 5-11 years. The more the frequency of the seizure is; the more the anxieties about sleeping are.

Children suffering from epilepsy were scoring higher for poor sleep quality, about sleep anxiety, and breathing disorders than controls. Cortesi *et al*[34] evaluated 89 children with idiopathic epilepsy for comparisons with 49 siblings and 321 healthy control children using parental questionnaire to assess sleep problems. Their findings indicated that attention to behavioral problems and sleep was important in clinical management of children with idiopathic epilepsy. When seizures occur during sleep, the seizures may awake the patients from sleep which could be mistaken with insomnia. Epileptic patients are frequently not aware of the seizures that occur during sleep. Epileptic children had more problematic sleep disorders than did the controls. These sleep disorders were correlated with how much seizure were frequent, the age of the child, duration of illness, presence of paroxysmal activity on EEG, and behavioral problems. Disturbance of the sleep architectures have been recorded in epileptic patients. This disturbance took different forms. It may appear as interruption and unsteadiness of the protective REM phase of sleep with shortening of REM phase duration. The child also may complain taking long time to go to sleep or from increase frequency of waking up after sleep onset and increased number of arousals, awakenings, and stage shifts[35]. In addition, the type of epilepsy may have a role in sleep disturbances. For example; children with partial epilepsy with secondary generalization or idiopathic epilepsies may suffer more problems and less tolerance to sleep fragmentation than observed in children with generalized tonic-clonic epilepsy or those with symptomatic/cryptogenic seizures. There was a strong association between nocturnal EEG abnormalities (*e.g.*, paroxysmal activity) and the frequency and severity of sleep disturbance which give us a clue that the epileptiform discharges could have an important role in arousal disorders of sleep[34].

The sleep disturbances occurring in epileptic children may be in part due to the effect of drugs used to treat epilepsy. However, the anti-epileptics do not share the same effects on the sleep. Some drugs may have damaging effects on the sleep, predominantly benzodiazepines and barbiturates and others has less damaging effects like phenytoin and, possibly, carbamazepine (CBZ). On the other hand; other drugs may improve the sleep quality especially gabapentin. CBZ is well known to increase SWS[36]. Polytherapy may aggravate sleep related awakening due to parasomnia which may affect both intrinsic and extrinsic features of sleep. Children on more than one antiepileptic drug suffered worse sleep habits than children on one antiepileptic drug[37]. Ketogenic diet treatment despite decreasing the sleep time but it is able to improve sleep quality in epileptic children having drug-resistant epilepsy[38]. Table 1 showed the effects of anteepileptics on sleep. Epilepsy is rarely a syndrome purely of seizures-rather, it is usually accompanied by other cognitive, behavioral, and emotional changes. Epilepsy-associated co-morbidities and other health troubles may also affect sleep. Epileptic children are more risky to develop depression, anxiety, migraine headaches, and obesity which in turn may affect sleep pattern[39].

Considerable sleep impairment in epileptic children could negatively affect their quality of life and impair their seizure control. So, it is of utmost importance to improve all aspects of sleep and should be taken into consideration when treating an epileptic child as sleep can be affected by frequency of seizure occurrence, their location and the balance between the benefits and side effects of antiepileptic used. The concerned treating physician should proactively evaluate sleep and treat any sleep disturbances as part of the comprehensive care of epileptic patients[6]. Many researchers showed marked improvement in sleep quality by proper controlling of nocturnal seizures which improved sleep efficiency, decreased arousals, and associated with increases in duration of REM sleep[40].

EPILEPSY AND PARASOMNIA

Parasomnia is a category of sleep disorders which involve abnormal and unusual behaviours, perceptions, movements, feelings, or dreams that could occur in any sleep phase while going to sleep, sleeping, between individual sleep stages, or during the stage of arousal from sleep. Parasomnias were classified into two main categories of parasomnias according to the sleep state of origin. The first category occurs during stage 3 (or 4) of NREM sleep and called NREM parasomnias or SWS. The second category is REM parasomnias which occur between wakefulness and REM sleep. The NREM parasomnias are further subdivided into disorders of arousal and disorders of sleep-wake transition and include confusional arousals; sleep walking (somnambulism), sleep talking

Table 1 Effects of anti-epileptic drugs on sleep

Antiepileptic	Effect on sleep
Benzodiazepines and barbiturates[70,71]	Reduce sleep latency Decrease the amount of REM sleep Benzodiazepines reduce slow wave sleep Increase incidence of OSA
Phenytoin[70,72,73]	Increases light sleep Decreases sleep efficiency Most studies show decreased REM sleep
Ethosuximide	May cause sleep disturbances, and night terrors
Carbamazepine[72,74,75]	Reduction in REM sleep particularly with acute treatment Effective in treatment of restless legs syndrome sleep disorder
Valproate[73]	May increase stage 1 sleep Could worsen OSA through weight gain
Lamotrigine[75,76]	May cause decreases in slow wave sleep May cause insomnia and sleep disturbance
Gabapentin, pregabalin, and tiagabine[76-81]	Effective in treatment of restless legs syndrome Enhance slow wave sleep and sleep continuity Gabapentin is effective in treatment of restless legs syndrome Pregabalin may cause insomnia and abnormal dreams
Levetiracetam[79,80]	Has little effect or an increase in sleep continuity and slow wave sleep
Zonisamide, rufinamide, oxcarbazepine, and topiramate[82,83]	Have no known effects on sleep and sleep disorders May cause insomnia

REM: Rapid eye movement; OSA: Obstructive sleep apnea.

(Somniloquy); bruxism (teeth grinding), night terrors, restless legs syndrome; and periodic limb movement syndrome. On the other hand; REM associated parasomnias include behaviour disorders of REM sleep, night mares and catathrenia. Catathrenia consists of breath holding and expiratory groaning during sleep, is different from both sleep talking and obstructive sleep apnea (OSA). Because of the higher frequency of arousal disorders, nightmares and bruxism in families with frontal lobe epilepsy; Bisulli *et al*[40] suggested an intrinsic link between parasomnias and NFLE and an abnormal (possibly cholinergic) arousal system as a common pathophysiologic mechanism. Despite being benign disorder, frequent or unusual episodes of parasomnias may occasionally be confused with epilepsy, predominantly NFLE[41]. In cases of diagnostic dilemma video-EEG monitoring is required. Even this, however, may not result in a proper diagnosis, as interictal and ictal EEG findings are frequently unremarkable or nonspecific in both parasomnias and NFLE[42].

EPILEPSY AND OSA

OSA is a common problem in epileptic patients. It presents in about 10% of unselected adult epilepsy patients, 20% of children with epilepsy and up to 30% of drug-resistant epilepsy patients. OSAs results in sleep disruption and deprivation which worsens seizure control and induces drug resistance. It causes repeated brief cessation of breathing during night, sometimes leads to cerebral hypoxemia that could trigger seizure activity. Many researches propose increased incidence of nocturnal seizures during the lighter stages of sleep. OSA is associated with greater incidence of transitory stages of light sleep due to sleep fragmentation which act as a seizure-provocative factor. On the other side, OSA can occur as a side effect of certain epilepsy therapies. For example; vagus nerve stimulation which is used to control the seizures in children with refractory epilepsy above the age of 12 years may enhance OSA in patients with preexisting OSA[43,44]. Barbiturates and benzodiazepines can cause respiratory depression, smooth muscle relaxation and hypotonia of upper airway muscles making them more collapsible. Other antiepileptics (such as valproate, gabapentin, CBZ, pregabalin, and vigabatrin) can induce weight gain as an adverse side effect and hence can promote OSA[45]. Malow *et al*[46] studied the effect of treating the co morbid OSA with medically refractory epilepsy in 35 adult patients using continuous positive airway pressure (CPAP). They found that proper managements of OSA in epileptic patients notably helps in seizure control.

EPILEPSY AND SLEEP-RELATED HORMONES

Many physiological hormonal changes in neuroendocrine system occur in the epileptic patients especially that encountered in the sex hormones. The mechanisms of these neuroendocrine changes are not yet fully elaborated. The hypothalamo-pituitary-adrenal axis function could be impaired acutely during the seizure or may be affected chronically on the long run in epileptic patients. Melatonin is a powerful chronobiotic secreted from the pineal gland; helps to maintain normal circadian rhythms and is used to treat sleep disorders. Bazil *et al*[47] found that patients with intractable epilepsy have low baseline melatonin levels that increase dramatically following seizures. Night-time melatonin intake proved to be helpful in alleviating epileptic activity in children specially

myoclonic types and nocturnal epilepsies. However; it is unclear whether this alleviating effect was through improving quality of sleep or through more specific neuroprotective function[48,49]. On the other hand, some studies reported that melatonin can decrease seizure threshold, and hence could increase seizure activity[50,51]. Gupta *et al*[52] showed that daily doses of 3-9 mg of melatonin could have a valuable effect through its effects on antioxidant enzyme levels and through improving the quality of life in epileptic children.

Some studies demonstrated increase of certain hormones immediately after the seizure. The serum levels of prolactin (PRL) and sex-related hormones (as luteinizing hormone, and follicle-stimulating hormone) were found to increase in the postictal stage in epileptic patients with either generalized tonic-clonic or partial seizures[53]. PRL shows more consistent postictal changes than other hormones. Serum PRL showed transient increase following epileptic seizures. The main reason for such increase in postictal PRL level is probably due to involvement of the temporal lobes and limbic system. This increase could be used to discriminate between true epileptic and psychogenic fits. It is advisable to measure serum PRL with in 10 to 20 min after the fit and is probably a useful measure to differentiate between a grand mal epilepsy seizure, complex partial seizure or psychogenic non-epileptic seizures. In contrast, if the serum PRL level is measured after 6 h from the seizure, then it may indicate the baseline PRL level of the patient. Nonetheless, serum PRL assay cannot differentiate between true epileptic seizures and loss of consciousness due to syncope. At the same time, normal levels of PRL cannot exclude epilepsy or confirm presence of psychogenic seizures because of low PRL assay sensitivity[54,55]. Meanwhile, there are a considerable numbers of studies with conflicting results about PRL increase in epilepsy[56,57].

Despite some hormonal changes other than PRL were observed during the postictal stage, there was no strong evidence of their usefulness for postictal hormonal testing[58,59]. PRL releasing neuropeptide (PrRP) is a peptide with strong PRL-releasing effect from the anterior pituitary cells. Its receptors present not only in the hypothalamic-pituitary axis but also present outside it suggesting that it could have other roles. Lin *et al*[60] found that PrRP has the ability to modify the function of the reticular thalamic nucleus and triggering of non-convulsive absence seizures. These finding open the door for new therapeutic methods able to alleviate sleep disorders and absence seizures. Zhang *et al*[61] showed that adrenocorticotropic hormone (ACTH) and cortisol serum levels showed significant fluctuations in epileptic patients: decreasing below the usual sleep-level shortly before epileptic seizures, rising during epileptic seizures and far above the average wake-level after epileptic seizures. In contrast; Gallagher[62] showed that patients with temporal lobe epilepsy secrete ACTH at higher rates and in greater amounts than normal subjects.

El-Khayat *et al*[63] found that the post provocation growth hormone (GH) levels as well as that of insulin-like growth factor 1 were significantly lower in children and adolescents with idiopathic epilepsy than in the control. This can explain the impaired physical growth pattern observed in the affected children. On the other hand, status epilepticus has a paradoxical GH effect. Lindbom *et al*[64] showed that regulation of GH was significantly altered as a result of the long-standing epileptic activity encountered in status epileptics. Ghrelin is a peptide hormone, which affects both endocrine function and sleep. Berilgen *et al*[65] showed that the mean serum ghrelin level was significantly higher in epileptic patients than the control. This increase in serum ghrelin level might contribute in prolongation of NREM sleep in those epileptic patients, thus may participate in seizure provocation. On the other hand, increase serum ghrelin levels could induce physiological changes in the hormonal secretions or function through its impacts on GH, and in that way play a supplementary role in seizure provocation.

Hormonal changes that occur during puberty could enhance the appearance or remission of certain types of epilepsy. A good example of this hormonal effect is juvenile myoclonic epilepsy which typically develops around the onset of puberty. On the other hand, the childhood absence seizures often remit during puberty[66].

SLEEP HYGIENE IN EPILEPTIC CHILDREN

Adequate sleep helps the overall health and helps to prevent seizures, and improve memory, learning and concentration. Sleep disruption can cause increasing sleepiness during daytime, deterioration of the seizures control, and poor life quality. Proper sleep disorders screening in epileptic children and appropriate interventions will help to improve the quality of life and adequate seizure control[67].

Epileptic patients are in a real need for proper sleep hygiene practice which is more reasonable in epileptic patients than in normal subjects. The need for adequate sleep hygiene practice in epileptic patient appeared as a result of their habitual prevention from exercising many of the activities that could trigger seizure activity or worsen the course of epilepsy or its complications[68].

For a better sleep hygiene, the child is better to have fixed sleeping and awakening times every day including weekends. Naps are better to be avoided if possible. The epileptic patient should be encouraged to have a regular exercise, particularly aerobic exercise, is good not only for the sleep or epilepsy control but also for overall health. However, the child should avoid stimulating or strenuous exercise especially in the evening times (should be avoided no less than 5 h before bedtime).

The child should avoid exhausting, exciting, annoying, or anxiety provoking activities at the bed time, in bed or in the bedroom (watching movies, learning, revising the checkbook, *etc.*). Quiet, relaxing activities and breathing techniques could help the child to feel asleep. Stimulating drinks like cola, coffee, tea, or other caffeinated beverages should not be taken after about noon. He should

not have chocolate by the evening time. The child should keep away from drinking many liquids in the evening to avoid disturbing the sleep by needing the toilet. Sedating anti-epileptics should be lessened if possible during the day, and activating anti-epileptics should be used when suitable. Weight gain should be avoided in patients with sleep apnea. Obese patients with sleep apnea should avoid using anti-epileptics that may promote weight gain; instead they should be encouraged to use anti-epileptics that cause weight loss. CPAP is still the treatment of choice for sleep apnea[69].

CONCLUSION

Epilepsy and sleep are integrating together; controlling epilepsy will improve the sleep quality while consolidating sleep will make it easier to control the seizures. Meticulous care of sleep habits in epileptic children has important effects in epilepsy diagnosis and control as well as quality of life. Adequate studying of the sleep related disorders and hormones may give a clue for new methods of better epilepsy control.

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