EPILEPSY

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Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "EPILEPSY Newsletter'' which contains latest and interesting news in the field of Neuropsychiatry.

This issue contains:

- **Prevalence of Sleep Disorders in Epilepsy Patients: A Case-Control Study**
- **Anterior Prefrontal EEG Theta Activity Reflects Memory and Executive Functions in Epilepsy Patients**
- **Impact of childhood trauma on psychogenic non-epileptic seizures**
- **Case Report on New-Onset Absence Status Epilepticus During Pregnancy**

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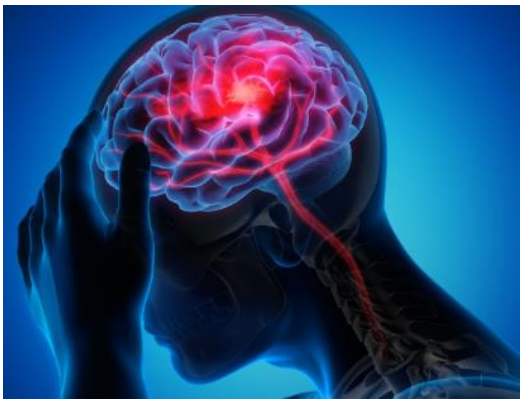
Prevalence of Sleep Disorders in Epilepsy Patients: A Case-Control Study

INTRODUCTION

Insomnia frequently affects individuals with epilepsy, with prevalence rates reaching up to 50%.[1] People with epilepsy experience sleep disturbances at nearly twice the rate of healthy individuals, with about one-third reporting sleep-related issues.[2] During non-rapid eye movement (NREM) stage 2 sleep, cortical excitability is believed to increase, making seizures more likely to occur, while the desynchronization seen in rapid eye movement (REM) sleep has an antiepileptic effect. Sleep deprivation can also trigger epileptic seizures, which may take place during sleep. This bidirectional relationship between epilepsy and sleep disturbances can contribute to comorbid psychiatric conditions such as anxiety and depression.[3] So, this study assessed the prevalence of sleep disorders in individuals with epilepsy through a questionnaire and to explore their association with epilepsy type, seizure frequency, and the use of antiepileptic medications.



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Psychiatric comorbidities, epilepsy, and sleep difficulties have a complicated relationship, and the existence of these comorbidities may seriously lower patients' quality of life.

METHODS

This case-control study included 100 epilepsy patients from our outpatient clinic who had no psychiatric or systemic conditions that could contribute to sleep disturbances and 50 healthy controls matched them. Participants completed several assessments, including the Epworth Sleepiness Scale, the STOP-Bang questionnaire for obstructive sleep apnea screening (OSAS), the REM Behavior Disorder-Hong Kong Questionnaire, the Swiss Narcolepsy Scale, the Restless Legs Syndrome (RLS) Diagnostic Form, the Beck Depression Scale, and the Beck Anxiety Scale. Multiple logistic regression analysis was used to study the relationship between epilepsy and sleep disturbances, anxiety, and depression, considering important and potential confounding factors such as age, gender, and antiepileptic medication type.

RESULTS

The rates of sleep disorders in both the epilepsy and control groups were described in Table 1. Excessive daytime sleepiness was observed in 30% of epilepsy patients, with half of them experiencing seizures within the past six months. The risk of OSAS was significantly higher in individuals with epilepsy, particularly among males, older patients, and those with left temporal lobe epilepsy (p=0.02). RLS was also more common in epilepsy patients, with a higher risk

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noted in females (p=0.04). Insomnia affected 33% of epilepsy patients, though no significant correlation was found between insomnia and factors such as age, gender, or the number of anti-seizure medications used. Additionally, moderate to severe depression was reported in 45% of epilepsy patients, a rate significantly higher than that of the general population (p=0.03). Anxiety was present in 50% of patients (p<0.01).

Table 1. Sleep disorders in the epilepsy and control groups

	Epilepsy patients (n=100)	Control (n=50)	p value
ESS	30	7	p<0.01
Insomnia	33	15	p=0.7
STOP-Bang Survey			
High risk	15	4	p<0.01
Medium risk	4	4	p<0.01
Low risk	81	44	p<0.01
Restless Leg Diagnosis Form	20	5	p<0.01
RBDQ-HK	12	1	p=0.01
Swiss Narcolepsy Scale	15	1	p=0.01
Beck Depression Scale			
Minimal	29	19	p>0.01
Mild	31	8	p<0.01
Moderate	14	-	p<0.01
Beck Anxiety Scale			
Minimal	20	15	p>0.01
Mild	21	5	p<0.01
Moderate	29	3	p<0.01

ESS: Epworth Sleepiness Scale, RBDQ-HK: REM Behavioral Disorder-Hong Kong Questionnaire, REM Sleep Behavior Disorder Questionnaire

DISCUSSION

Increased daytime sleepiness is a common complaint experienced by patients with epilepsy, and it is particularly subjective. According to reports, persons with epilepsy experience increased daily sleepiness at a rate of 10 to 47.5.[4] Thirty percent of the patients in the present study had an ESS score greater than 10. Patients with epilepsy who were at high risk for OSAS also had significantly higher anxiety scores in the present study (p=0.02). This finding is in line with previous research, which found that patients with epilepsy and OSAS had higher levels of seizure fear, which may raise their scores on the Beck fear Scale.[5]

CONCLUSION

The interplay between sleep disorders, psychiatric comorbidities, and epilepsy is intricate, with these conditions collectively having a significant impact on patients' quality of life. Further comparative studies with more homogenous patient groups are necessary to gain a deeper understanding of this complex relationship.

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Anterior Prefrontal EEG Theta Activity Reflects Memory and Executive Functions in Epilepsy Patients

INTRODUCTION

In epilepsy, theta EEG power in the anterior prefrontal cortex offers a simple, easily accessible, and objective electrophysiological assessment of memory and executive function.

Cognitive function deficits are common in various neurological and psychiatric disorders, impacting overall performance and quality of life. Due to limited treatment options for these impairments, there is a growing interest in developing targeted and innovative therapeutic interventions that focus on specific brain regions and neural activity. Electroencephalography (EEG) provides a non-invasive method for identifying neural activity associated with cognitive functions. Certain frequency bands within the EEG spectrum can act as biomarkers for cognitive abilities.[1] For instance, increasing resting spectral power in the upper alpha frequency range using neurofeedback training has been linked to enhanced cognitive performance.[2] EEG characteristics, including spectral power in the delta, theta, and alpha frequency ranges, are linked to specific cognitive functions. This study involved EEG recordings and proposed that theta activity in the anterior prefrontal cortex would correspond to patient performance on cognitive tasks. Theta frequency activity in the frontal cortex is known to be crucial for memory and executive functions, but only during certain tasks.[1] This study aimed to identify a consistent EEG pattern across various frequency bands and cortical regions that could distinguish epilepsy patients with cognitive impairments from those with normal cognitive functions.

METHODS

Eighty-six epilepsy patients with confirmed electrographic seizures were enrolled in this study while undergoing monitoring in the epilepsy unit at Mayo Clinic. Researchers utilized the Cambridge Neuropsychological Test Automated Battery (CANTAB) to assess memory and executive function in these individuals. EEG signals recorded during specific tasks were analyzed to identify distinct frequency bands and brain regions associated with varying levels of cognitive performance—categorized as impaired, normal, or superior—based on age and gender. A two-way ANOVA was used to evaluate spectral features between good and bad performers in each task.

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RESULTS

Patients with impaired memory and executive function (z-score <-1) exhibited consistently lower EEG power in the theta frequency band within the anterior prefrontal cortex. This reduction was observed across all four behavioral measures—executive, visual, spatial, and working memory functions—and was specifically localized to the frontal pole electrode sites (Nz, Fpz, Fp1, and Fp2). A summary of the effects of patient group differences on anterior theta power was provided in Table 1.

Table 1. Summary of patient group effects on the anterior theta power

	Nz				Fp1		Fpz		Fp2			
	Visual memory	Spatial memory	Working memory	Executive	Visual memory	Executive	Visual memory	Executive	Visual memory	Spatial memory	Working memory	Executive
Good versus poor	0.4520	0.0001	0.0014	$<10^{-5}$	$<10^{-5}$	0.0358	$<10^{-5}$	$<10^{-5}$	0.9969	0.3752	0.0188	$<10^{-5}$
Good versus normal	0.0170	$<10^{-5}$	0.0002	$<10^{-5}$	$<10^{-5}$	0.1765	$<10^{-5}$	0.0791	0.1680	0.0057	0.0051	0.0252
Normal vs poor	0.0221	0.1225	0.5193	0.8016	0.0715	0.9816	0.6289	0.3304	0.0120	0.0160	0.6112	0.2574

DISCUSSION

It was established that non-invasive EEG can serve as a predictor of cognitive performance. Basic measures, such as the amplitude and latency of specific event-related potential components, have been shown to distinguish between strong and weak memory performance, as subjectively assessed in both younger and older adults.[3] Recent studies suggest that theta activity may serve as a diagnostic marker for cognitive impairment in epilepsy.[4] Researchers conducted a study to examine EEG spectral power and theta phase coherence in pediatric patients with common genetic neurodevelopmental disorders associated with cognitive impairment. These measures were assessed both at rest and during a working memory task, with comparisons made to an age- and sex-matched control group of typically developing children. Results showed that children with cognitive impairment exhibited increased resting-state slow-wave power, a lower peak alpha frequency, and greater theta power. Additionally, they demonstrated heightened frontoparietal theta phase coherence during the working memory task. However, these differences were no longer significant after adjusting for baseline resting-state activity.[5]

CONCLUSION

Theta EEG power in the anterior prefrontal cortex serves as a straightforward, accessible, and objective electrophysiological indicator of memory and executive function in epilepsy. These findings highlight its potential as a clinical biomarker for diagnosing, monitoring, and treating cognitive deficits, particularly with the development of targeted neuromodulation therapies.

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Impact of childhood trauma on psychogenic non-epileptic seizures

INTRODUCTION

The onset of PNES and the mental health issues that PNES patients experience are linked to childhood trauma.

Psychogenic non-epileptic seizures (PNES) are sudden, involuntary episodes that mimic epileptic seizures but result from intricate biopsychosocial factors rather than abnormal brain activity.[1] PNES is frequently mistaken for epilepsy and occurs in approximately 20–30% of patients seen at epilepsy centers.[2] Although the neurobiological mechanisms underlying PNES are not yet fully understood, the psychological and social risk factors have been well documented. Childhood trauma, along with experiences of abuse and life adversity, has been identified as a significant contributor to PNES.[1] Patients with PNES reported experiencing childhood trauma more frequently across all categories in the Childhood Trauma Questionnaire compared to those with epilepsy.[3] This study examined the prevalence of childhood trauma in PNES patients using standardized assessment tools and defined cutoff scores for exposure. Additionally, researchers investigated the relationship between childhood trauma and current psychiatric symptoms as well as clinical characteristics.

METHODS

This case-control study included 35 PNES patients and 34 control participants. PNES patients were recruited consecutively from the epilepsy monitoring unit, where their diagnosis was confirmed through 24-hour video-electroencephalogram (VEEG) monitoring. A diagnosis required the occurrence of at least one typical seizure-like event without accompanying epileptiform activity. PNES was categorized into three types based on semiology: psychogenic minor motor seizures, psychogenic major motor seizures, and unresponsive seizures. Childhood trauma and psychiatric symptoms were assessed using the Childhood Trauma Questionnaire-Short Form (CTQ-SF), the Dissociative Experiences Scale (DES), and the Symptom Checklist-90 (SCL-90).

RESULTS

The PNES group had a significantly higher rate of parental separation (48.6%) compared to the control group (23.5%, P=0.03). PNES patients also scored significantly higher on the CTQ-SF and its Physical Abuse (PA) and Physical Neglect (PN) subscales compared to controls. Based on cutoff scores, PNES patients showed a higher prevalence of PA and PN. Additionally, they reported more psychiatric symptoms on the SCL-90 and exhibited greater dissociative symptoms, with a median DES score of 25 versus 13.9 in controls (P=0.004) (Table 1). A two-variable model in PNES patients explained 54% of the variance in the overall SCL-90 score, indicating that higher Sexual Abuse (SA) scores and a greater number of trauma types were associated with increased SCL-90 scores. Among controls, the DES score correlated with the number of trauma types (Sβ=0.366, P=0.024) and SA (Sβ=0.360, P=0.026). Regression analyses revealed that higher SA scores (Sβ=0.494, P=0.001) were linked to later PNES onset, while a history of parental separation (Sβ = −0.365, P=0.013) was associated with earlier onset. Seizure frequency was only correlated with DES scores (Sβ=0.384, P=0.023), accounting for 12.1% of the variance.

Table 1. Comparison of the scale scores between PNES patients and controls

Variables	PNES patients (n = 35)	Healthy controls (n=34)	P value
CTQ-SF	40.0 (35.0–46.0)	36.5 (32.3–41.3)	0.031[a]
EA	7.0 (6.0–10.0)	6.5 (5.0–8.3)	0.265[a]
EA ≥ 9	12 (34.3)	8 (23.5)	0.325[b]
PA	6.0 (5.0–8.0)	5.0 (5.0–5.3)	0.006[a]
PA ≥ 8	11 (31.4)	3 (8.8)	0.020[b]
SA	5.0 (5.0–6.0)	5.0 (5.0–5.0)	0.078[a]
SA ≥ 6	10 (28.6)	4 (11.8)	0.083[b]
EN	12.0 (9.0–14.0)	11.0 (6.8–13.5)	0.389[a]
EN ≥ 10	22 (62.9)	19 (55.9)	0.555[b]
PN	9.0 (7.0–11.0)	7.0 (6.0–9.0)	0.015[a]
PN ≥ 8	26 (74.3)	15 (44.1)	0.011[b]
Number of trauma types	2.0 (1.0–3.0)	1.5 (0.8–2.0)	0.017[a]
SCL-90	2.0 (1.6–2.6)	1.3 (1.1–1.6)	0.000[a]
SOM	1.8 (1.6–2.3)	1.2 (1.1–1.4)	0.000[a]
O-C	2.2 (1.8–2.9)	1.6 (1.3–2.1)	0.001[a]
INT	1.8 (1.6–2.3)	1.3 (1.1–1.6)	0.000[a]
DEP	2.0 (1.4–2.7)	1.3 (1.1–1.5)	0.000[a]
ANX	2.3 (1.7–3.0)	1.3 (1.1–1.8)	0.000[a]
HOS	2.5 (1.5–3.0)	1.3 (1.0–1.8)	0.000[a]
PHOB	1.9 (1.1–2.7)	1.1 (1.0–1.5)	0.000[a]
PAR	1.7 (1.3–2.2)	1.2 (1.0–1.7)	0.005[a]
PSY	1.5 (1.3–2.1)	1.1 (1.0–1.4)	0.000[a]
DES	25.0 (17.9–36.8)	13.9 (5.5–24.6)	0.004[a]
([a] Independent-samples Mann-Whitney U Test; [b] Chi-square test)			

DISCUSSION

This study found that individuals with PNES had a higher rate of childhood trauma compared to healthy individuals, reinforcing the link between early-life trauma and PNES. Previous research also identified psychological trauma as a key contributing factor among PNES patients.[4] In the PNES group, certain risk factors for childhood trauma were identified. Many PNES patients reported experiencing early separation from their parents, which was linked to

greater childhood trauma and more severe Emotional neglect (EN). The majority indicated that their parents were migrant workers who prioritized financial stability over family time. This disruption in parent-child relationships contributed to suboptimal childhood development.[5]

CONCLUSION

Patients with PNES experienced higher levels of childhood trauma, including PA, PN, and possibly SA. Such trauma plays a role in both the onset of PNES and the deteriorating mental health of affected individuals. Early separation from parents due to migration may contribute to childhood trauma and should be considered within the context of societal changes. Enhancing sex education and social support, particularly in rural areas, is essential. Overall, this study underscores the need for child protection efforts, given the lasting impact of childhood trauma.

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Case Report on New-Onset Absence Status Epilepticus During Pregnancy

INTRODUCTION

Being the first documented case of de novo ASE brought on by pregnancy, the current case emphasizes its importance.

Absence status epilepticus (ASE), a form of status epilepticus (SE), is mainly marked by impaired mental status, ranging from slight cognitive and responsiveness deficits to a stuporous state. While ASE is commonly associated with individuals who have idiopathic or genetic epilepsy syndromes (I/GGE), it can also emerge de novo in rare cases. Late-onset de novo ASE may be triggered by factors such as chronic alcohol use, benzodiazepine withdrawal, or metabolic disorders, particularly in older adults.[1] New-onset status epilepticus is rarely observed in pregnant women without a prior history of seizures. When it does occur, it is often linked to factors such as pregnancy-induced hemodynamic changes, eclampsia, or cerebral thrombotic events.[2] This case describes a newly developed ASE during pregnancy and explores the potential underlying mechanisms.

CASE PRESENTATION

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A 22-year-old primigravid woman at 13 weeks of gestation visited the outpatient clinic due to recurrent fainting episodes over the past two days. These episodes were marked by noticeable pallor, loss of awareness, and involuntary movements affecting all limbs, each lasting up to 10 seconds. Her family noted that she appeared confused, had reduced spontaneous speech, and had shown decreased engagement in daily activities since the onset of these episodes. Neurological examination revealed apathy and significantly reduced spontaneous movement. She provided only single-word responses and had difficulty comprehending complex questions and instructions. Additionally, she exhibited temporal disorientation, though no focal motor or sensory deficits were identified. During the assessment, she experienced staring spells lasting up to 10 seconds. Her blood pressure was within the normal range. Routine electroencephalography (EEG) showed continuous, generalized synchronous paroxysms of 3 Hz spike-wave complexes (Fig. 1A), with intermittent persistence and occasional muscle artifacts. She was treated with an intravenous infusion of 10 mg of diazepam, followed by a daily dose of 1,000 mg of levetiracetam. By the next day, her speech had returned to normal, and she was fully alert. On the second day of hospitalization, a follow-up EEG revealed slow-wave paroxysms in the theta-delta frequency, occurring occasionally over a low-amplitude background, primarily in the bilateral frontal regions (Fig. 1B). With her clinical and EEG findings normalized, she was discharged on levetiracetam (1,000 mg/day). Two months post-discharge, she remained seizure-free, and her EEG demonstrated normal background activity with posteriorly located alpha rhythm (Fig. 1C). She delivered a healthy baby at 38 weeks of gestation without further seizures or pregnancy-related complications. During her three-month postpartum follow-up, she remained seizure-free while continuing levetiracetam treatment.

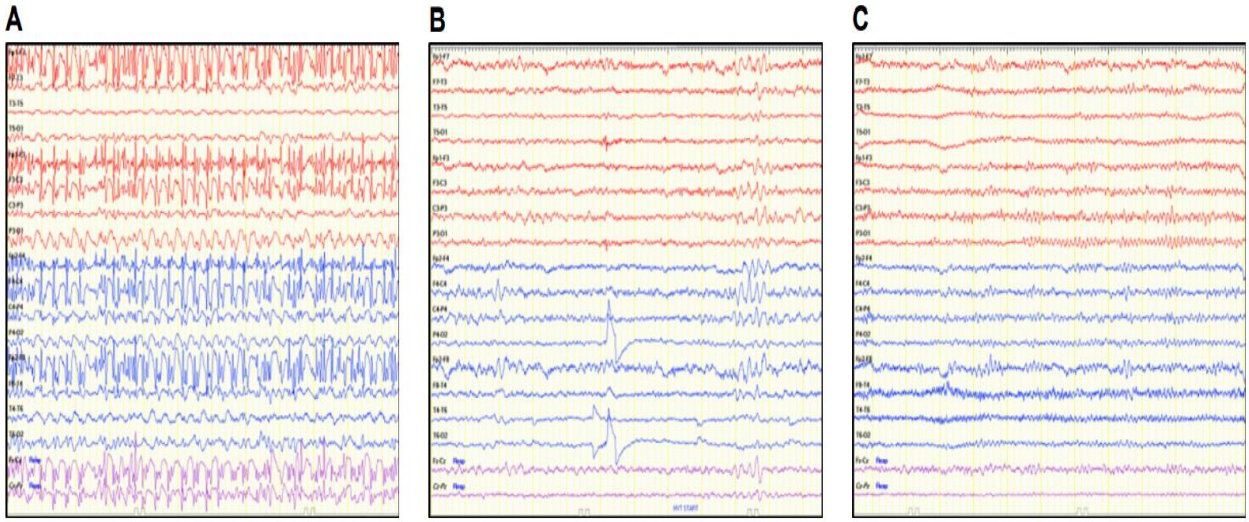

Figure 1. Electroencephalography (EEG) findings of the patient. (A) EEG shows continuous, generalized synchronous paroxysms of 3 Hz spike-wave complexes on day 1. (B) Control EEG demonstrates the suppression of the generalized synchronous activity on day 2. (C) Control EEG in the second month reveals normal background activity.

DISCUSSION

This was the first documented occurrence of de novo ASE during pregnancy. When a pregnant woman presents with a new-onset seizure, eclampsia should be among the primary conditions to be ruled out. In this case, eclampsia was excluded based on the fact that status epilepticus developed before the 20th week of gestation and the patient's blood pressure remained within normal limits.[3] Experiencing a first-time seizure during pregnancy is uncommon. Studies indicate that around 2.3–2.4% of women diagnosed with epilepsy have their initial seizure during pregnancy.[4,5] In this case, the researchers considered ASE to be a potential initial manifestation of I/GGE, specifically juvenile absence epilepsy (JAE), despite the absence of prior seizures. While JAE is usually diagnosed between ages 9 and 13, rare cases of later onset in young adulthood have been documented.[6]

CONCLUSION

The case report highlighted the uncommon nature of ASE in epilepsy practice and the rarity of de novo cases. This case is particularly significant as, to the best of the researcher's knowledge, it represents the first documented instance of de novo ASE induced by pregnancy.

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