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Review article:

Childhood Epilepsy: Clinical Insights into Pathogenesis, Classification, and Management Strategies

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ABSTRACT

Epilepsy is a widespread neurological disorder among children, with around 10.5 million cases globally. Progress in diagnostic techniques, EEG, and brain imaging has enhanced the ability to classify and treat pediatric epilepsy more effectively.

The disorder manifests in varying degrees, from mild, manageable forms to severe epileptic encephalopathies that hinder cognitive and motor growth. This research examines the prevalence, underlying causes, classification systems, and therapeutic approaches for childhood epilepsy. Key factors contributing to the condition include genetic predisposition, disruptions in ion channels, and brain injuries during the perinatal period.

Although antiepileptic medications are the standard treatment, some patients benefit from specialized diets, neurostimulation, or surgical procedures. Beyond physical symptoms, epilepsy also significantly influences a child's emotional well-being, social interactions, and educational progress. The study emphasizes the need for timely detection, tailored treatment plans, and public education to enhance the lives of children with epilepsy.

Keywords: Epilepsy, EEG, AEDs, OSA, VNS.

INTRODUCTION

Epilepsy represents a neurological condition marked by a lasting susceptibility to recurrent seizures. Since one of the most widespread neurological disorders globally, it affects individuals across all age groups and ethnicities. While industrialized nations report lifetime epilepsy rates of 3-4%, developing countries face even higher prevalence rates. The condition carries substantial consequences for patients' social functioning, employment prospects, physical health, and psychological state. The 2010 Global Burden of Disease study ranked severe epilepsy as having the fourth most significant disability impact out of 220 evaluated health conditions [1].

Clinical evaluation forms the cornerstone of epilepsy diagnosis, supplemented by diagnostic aids including EEG recordings and neuroimaging techniques, particularly MRI. Current ILAE diagnostic criteria (2014) establish epilepsy when A patient experiences two or more unprovoked seizures separated by at least 24 hours

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(conventional definition). A single unprovoked seizure occurs with a ≥60% likelihood of subsequent seizures within 10 years. A recognized epilepsy syndrome is present [1].

The spectrum of epilepsy syndromes shows remarkable variability in clinical course. Some forms, like absence epilepsy of childhood, typically resolve during adolescence, while others, such as juvenile myoclonic epilepsy, persist throughout adulthood. Particularly challenging cases include West syndrome and Lennox-Gastaut syndrome, which often prove refractory to standard treatments and may necessitate specialized therapeutic approaches, including ACTH therapy or ketogenic dietary regimens [2].

Fig.1. illustrates the cumulative occurrence rates of febrile convulsions. [66]

EPIDEMIOLOGY OF CONVULSIVE DISORDERS

Neonatal Seizure

The occurrence of neonatal seizures in full-term infants varies from 1 to 8 cases per 1,000 live births [3-5]. Developed nations report lower rates compared to developing countries, with increased incidence observed among infants from lower socioeconomic backgrounds. While most industrialized countries have seen a decline in cases among term infants, this trend is not universal [4, 6, 7].

Febrile Seizures

Approximately 2-4% of children in the U.S. and Northern Europe will experience at least one febrile seizure before age 5 [8-11]. Male children demonstrate consistently higher susceptibility. Global prevalence patterns show significant geographical variation (Fig. 1).

Risk Factors for Febrile Seizures:

Among the identified risk factors for febrile seizures, genetic predisposition (family history of febrile convulsions or epilepsy) shows the strongest and most consistent association [12-15].

SLEEP-DEPENDENT EPILEPSY CONDITIONS:

While epileptic episodes may occur during any state of consciousness, several distinct seizure disorders show preferential manifestation during sleep cycles. The principal sleep-associated epileptic conditions include: Frontal lobe epilepsy with nocturnal predominance. The benign childhood seizure disorder is characterized by centrotemporal spikes. Occipital-onset epileptic syndrome of childhood. The acquired aphasia-epilepsy complex (Landau-Kleffner syndrome). Continuous spike-wave activity during slow sleep (CSWS) [16-21]. Furthermore, certain generalized seizure patterns demonstrate distinct circadian timing: Dawn-predominant generalized convulsive episodes, Morning myoclonic jerks (characteristic of adolescent-onset myoclonic epilepsy), and Generalized tonic-clonic events triggered by awakening

Focal Seizures: Frontal Lobe Epilepsy:

While focal seizures can manifest during both sleep and wake states, their clinical presentation differs based on the seizure origin. Notably, epileptic events emerging from sleep are clinically recognized as a characteristic feature suggesting a frontal lobe epileptogenic focus [20].

THE DYNAMIC INTERPLAY BETWEEN SLEEP AND SEIZURE DISORDERS

Sleep's Influence on Epileptic Manifestations

Sleep-State Dependent Seizure Patterns

Documented observations of nocturnal seizure activity trace back to classical antiquity, with early descriptions found in Hippocratic and Aristotelian medical texts [22]. Contemporary research reveals pronounced fluctuations in epileptiform event occurrence throughout various sleep cycles, with empirical measurements illustrated in Figure 2 [23].

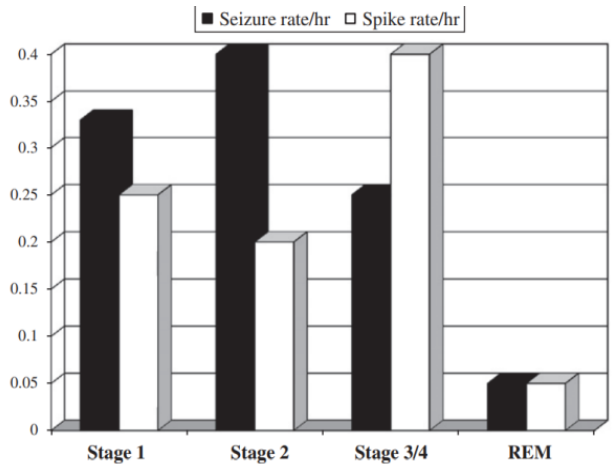

Fig.2. Temporal distribution of ictal and interictal activity during sleep stages. [67]

Effects of Sleep Restriction:

Pioneering EEG research conducted by Gibbs and Gibbs [24] established that sleep deprivation protocols significantly improve the detection of abnormal brain wave patterns associated with epilepsy. Subsequent comparative studies indicate that EEG recordings performed after sleep deprivation identify interictal epileptiform activity at rates 52% higher than standard sleep studies, regardless of the duration or depth of sleep obtained during the recording [25].

Mechanisms Influencing Sleep and Epilepsy

The physiological changes occurring during NREM sleep progression create conditions that enhance both seizure susceptibility and interictal discharge generation. While these effects are well-established, the particular sleep depth that most strongly promotes epileptiform activity shows considerable variation depending on individual factors, including patient age, specific epilepsy classification, and seizure subtype characteristics [23].

During NREM sleep, a state of synchronized neural activity emerges involving coordinated functioning between the brainstem's arousal networks, thalamic nuclei, and cortical pyramidal neurons. As NREM sleep deepens, two key neurochemical changes occur: diminished cholinergic neurotransmission and progressively stronger hyperpolarization of thalamocortical pathways. These electrophysiological modifications appear to lower seizure thresholds, creating favorable conditions for both interictal discharges and clinical seizure events.

In direct contrast, REM sleep and wakefulness represent physiologically distinct brain states that counteract these epileptogenic processes [30]. REM sleep demonstrates several protective mechanisms, including active suppression of thalamocortical synchronization [26], characteristic desynchronized EEG patterns, natural inhibition of epileptiform discharge propagation, and the typical skeletal muscle paralysis associated with this sleep stage.

SLEEP DISRUPTION IN EPILEPSY: ETIOLOGICAL FACTORS:

Patients with epilepsy frequently experience sleep disturbances stemming from multiple interrelated causes. Primary contributors include suboptimal sleep hygiene practices, comorbid sleep disorders such as insomnia or sleep apnea, and circadian rhythm abnormalities. Importantly, epileptic seizures themselves exert significant disruptive effects on sleep architecture and continuity, regardless of whether the events occur during wakefulness or sleep periods [27] (comprehensive analysis available in "Epilepsy's Impact on Sleep" section).

Antiepileptic drug (AED) therapy demonstrates complex, bidirectional influences on sleep quality that extend beyond its primary seizure-controlling mechanisms. These pharmacological effects may either improve or impair various sleep parameters through distinct pathways.

The cumulative impact of these sleep disruptions manifests clinically through excessive daytime somnolence, exacerbation of seizure frequency, and marked deterioration in overall quality of life, as visually summarized in Figure 3.

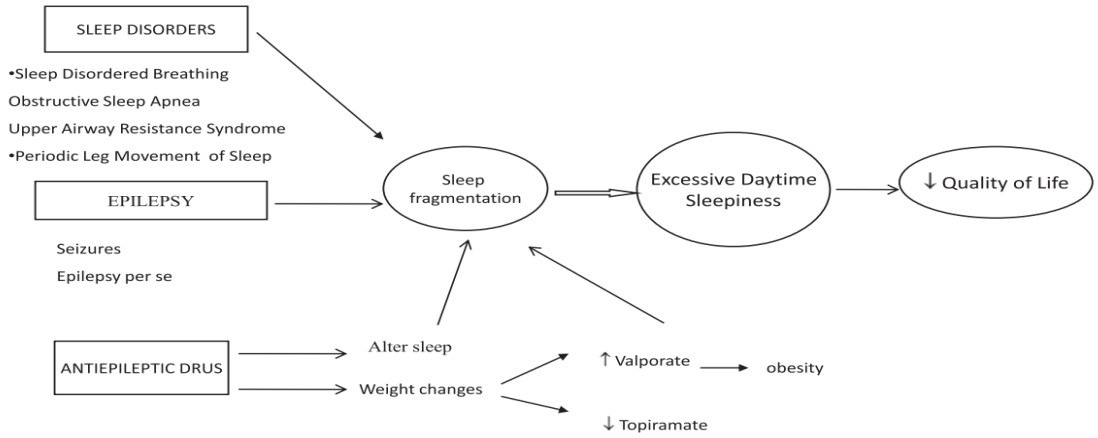

Fig.3 illustrates the multifactorial origins of sleep disturbances in pediatric epilepsy populations [67].

RESPIRATORY COMPLICATIONS ASSOCIATED WITH EPILEPTIC DISORDERS

Obstructive Sleep Apnea (OSA)

OSA represents a clinically significant yet frequently undiagnosed comorbidity in epileptic patients. Notably, therapeutic management of OSA may yield substantial improvements in seizure control, including potential reduction in seizure frequency or complete remission in some cases [28].

Central Sleep Apnea (CSA)

The pathophysiology of CSA during epileptic events involves multiple contributing factors [29]. This bidirectional relationship demonstrates complex interdependence: CSA may precipitate seizure activity through hypoxia-mediated lowering of seizure threshold [30]. Conversely, epileptic seizures may induce CSA episodes[29]. CSA may also manifest as a terminal event in seizure sequences [31].

Impact of Epilepsy Treatment on Sleep Patterns:

Pharmacological management of epilepsy exerts significant modulatory effects on sleep architecture and quality, with various anti-epileptic drugs (AEDs) demonstrating distinct influences independent of their seizure-control properties. Clinical observations reveal that AEDs may produce paradoxical effects, ranging from sedative to stimulant responses, potentially inducing either excessive drowsiness or sleep initiation difficulties [32]. Particular drug classes exhibit specific sleep-related effects: Benzodiazepines and barbiturates may alter respiratory physiology through Excessive relaxation of upper airway musculature, Impaired chemoreceptor sensitivity to blood gas fluctuations [33]. These diverse pharmacological impacts on sleep parameters are systematically categorized in Table 1

Table 1: for complete details about how epilepsy medications affect sleep. [67]

Drug	Sleep complaint	Sleep efficiency	TST	SL	Arousals	% Stage 1	% Stage 2	% SWS	% REM
Carbamazepine	S	↑	0	↓	↓	0	0	↑	↓/0
Ethosuximide	I	↓	?	?	↑	↑	0	↓	↑
Gabapentin	S	↑	↑	?	↓	↓	0	↑	↑
Lamotrigine	I	0	0	0	0	0	0	?	↑
Levetiracetam	S	0	0	0	0	0	↑	↓	0
Phenobarbital	S	↓	0	↓	↓	↑	↑	0	↓
Phenytoin	S	↓	0	↓	↓	↑	↑	↓	0
Tiagabin	I	?	?	?	?	?	?	?	?
Topiramate	S	?	?	?	?	?	?	?	?
Valproate	S	0	0	0	↑	↓	0	↑	↓
Vigabatrin	S	?	?	0	?	?	?	?	?
Zonisamide	I	?	?	?	?	?	?	?	?

TST = total sleep time; SL = sleep latency; SWS = slow wave sleep; REM = rapid eye movement; S = sleepiness; I = insomnia; ↑ = increase;↓ = decrease; 0 = no change; ? = unknown.

Vagus Nerve Stimulation (VNS) Therapy, as an FDA-approved adjunctive treatment for refractory epilepsy, demonstrates notable respiratory effects during sleep. Clinical evidence indicates this intervention may exacerbate preexisting obstructive sleep apnea (OSA) through modulation of respiratory patterns. Ketogenic Diet (KD) Therapy, The high-fat, low-carbohydrate ketogenic diet, employed for medication-resistant epilepsy cases, presents complex metabolic influences. While its precise anticonvulsant mechanisms remain incompletely understood, recent research suggests potential interactions between KD-induced metabolic states and circadian rhythm regulation [34].

SEIZURE CHARACTERISTICS ACROSS CEREBRAL PALSY SUBTYPES:

As presented in Table 2, children with cerebral palsy exhibited three predominant seizure types with nearly equal frequency: generalized tonic-clonic, focal (partial), and myoclonic seizures. Several patients experienced combinations of these seizure types. The study documented rare occurrences of astatic seizures (in two cases), which coincided with myoclonic episodes. Notably, no cases of absence seizures (either typical or atypical variants) were observed in this patient population [34].

Table 2 for seizure type distribution in cerebral palsy subtypes [67].

Seizure Type	*SPQ (n = 53)*	*SPD (n = 20)*	*SPH (n = 22)*	*DYS (n = 5)*	*HYPO (n = 3)*	*Mixed (n = 2)*	*Overall (n = 105)*
GTC	19 (35.1%)	9 (45%)	7 (31.8%)	1 (20%)	3 (100%)	1 (50%)	40 (38.1%)
SP	3 (5.7%)	3 (15%)	1 (4.5%)	1 (20%)	0	1 (50%)	9 (8.6%)
CP	6 (11.3%)	1 (5%)	6 (27.3%)	0	0	0	13 (12.4%)
P-SG	7 (13.2%)	3 (15%)	3 (13.6%)	0	0	0	13 (12.4%)
MJ	9 (17%)	3 (15%)	1 (4.5%)	2 (40%)	0	0	15 (14.3%)
IS	14 (26.4%)	5 (25%)	3 (13.6%)	1 (20%)	0	0	23 (22%)
Others	0	1 (5%)	0	0	0	0	1 (1%)

CP = complex partial; DYS = dyskinetic; GTC = generalized tonic clonic; HYPO = hypotonic; IS = infantile spasms; MJ = myoclonic jerks; P-SG = partial with secondary generalization; SP = simple partial; SPD = spastic diplegia; SPH = spastic hemiplegia; SPQ = spastic quadriplegia.

TREATMENT OUTCOMES AND MEDICATION MANAGEMENT:

Seizure Control and AED Discontinuation:

Effective seizure management was achieved in 58.1% of pediatric cases, while only 5.7% (n=6) successfully discontinued antiepileptic medication. Treatment response varied significantly by cerebral palsy subtype: Hemiplegic patients showed a 75% seizure control rate, Quadriplegic and diplegic patients demonstrated 50% control efficacy [35]

Polytherapy Utilization:

Combination therapy was necessary for 38.1% (n=40) of cases, with 26.6% (n=28) requiring dual-drug regimens 11.5% (n=12) necessitating triple-drug protocols. Comparative analysis of clinical parameters between mono- and polytherapy groups is presented in Table 3.

Table 3 for mono-versus polytherapy comparisons [67].

Characteristics	*Polytherapy (n = 40) (%)*	*Monotherapy (n = 65) (%)*
Mean age (mo)	51.1	51.13
History of birth asphyxia	13 (32%)	27 (41.5%)
Neonatal seizures	6 (15%)	6 (9%)
Seizure onset (mean) (mo)	17.9	19.5
Microcephaly	30 (75%)	47 (72%)
CT abnormalities	26 (65%)	38 (58.5%)
Unilateral	10 (25%)	17 (26.2%)
EEG abnormalities	31 (77.5%)	43 (66.2%)
Bilateral	31 (77.5%)	36 (55.4%)
Mean SQ	41.4	47.4
Mental retardation	32 (80%)	46 (71%)

P = not significant for all.
CT = computed tomography; EEG = electroencephalogram; SQ = social quotient.

CT SCAN ABNORMALITIES IN CHILDREN WITH CEREBRAL PALSY

Brain CT scans detected abnormalities in 61% of cases (64 children), with bilateral findings present in 74% of these abnormal scans (48 children) and focal abnormalities in 26% (17 children). The frequency of CT abnormalities varied by cerebral palsy type, being most common in hemiplegic and diplegic forms (75% each) compared to quadriplegic cases (57%). In hemiplegic patients, 60% of abnormal CTs showed unilateral changes. In contrast, bilateral abnormalities dominated in diplegic (93%) and quadriplegic (84%) patients.

ELECTROENCEPHALOGRAPHY FINDINGS

Electroencephalogram (EEG) recordings revealed abnormalities in 70.5% of cases (74 children). The observed abnormalities included generalized patterns (44 cases), focal abnormalities (13 cases), and focal discharges with secondary bilateral synchrony (17 cases). Additional findings included rhythm slowing (13 patients) and asymmetrical activity (3 patients).

The distribution of EEG abnormalities varied by cerebral palsy subtype:

Generalized abnormalities predominated in quadriplegic and diplegic patients. Focal abnormalities were present in 7 of 16 hemiplegic cases with abnormal EEGs. Rhythm

slowing occurred most frequently in quadriplegic and hemiplegic patients [35].

Pediatric Temporal Lobe Epilepsy: Clinical and Diagnostic Features

Temporal lobe epilepsy in children presents with age-dependent clinical manifestations. Infants and toddlers (0-3 years) often demonstrate nonspecific seizure patterns that resemble generalized epilepsy, with limited localizing features that become more distinct as children mature [36-41]. Between ages 3-6 years, clearer lateralizing signs emerge, including contralateral dystonic posturing and eye deviation (75-100% lateralizing accuracy), along with ipsilateral localization in 80% of early nonversive head turns [36, 37, 42-44]. Older children (>6 years) typically exhibit adult-like semiology, with frequent epigastric auras in mesial temporal cases, though they experience fewer prodromal symptoms than those with generalized seizures [42, 45].

Two particularly notable pediatric variants include abdominal epilepsy, characterized by episodic abdominal pain with vomiting and altered consciousness [45], and autonomic manifestations, most commonly ictal tachycardia occurring in up to 98% of cases with similar frequency across age groups [46].

Electroencephalographic evaluation benefits from sleep-activated studies with supplemental sphenoidal or inferotemporal electrodes [47-50]. Characteristic interictal findings include anterior temporal spikes/sharp waves and temporal intermittent rhythmic delta activity (TIRDA), often with prominent ear electrode involvement (Fig.4).

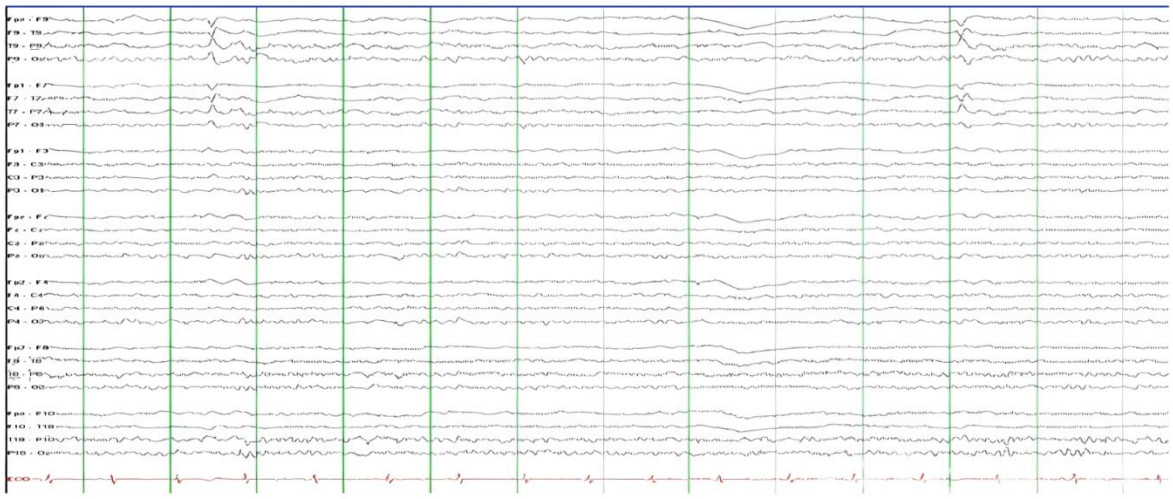

Figure 4. The electroencephalographic tracing demonstrates interictal epileptiform discharges localized to the left temporal region in an adolescent male patient with left mesial temporal sclerosis (recording parameters: sensitivity 10 μV/mm, paper speed 30 mm/sec) [67].

Ictal EEG Characteristics and Clinical Management of Temporal Lobe Epilepsy in Children

EEG Findings During Seizures:

While the temporal lobe demonstrates particular susceptibility to seizure activity, interpreting pediatric EEG recordings presents unique challenges. The ongoing maturation of neural circuits in children often results in less localized ictal and interictal patterns, even in clear temporal lobe epilepsy cases. Young patients with focal lesions may exhibit generalized or multifocal discharges, while anterior temporal seizures can initially manifest with widespread EEG changes that later localize [51].

Diagnostic Considerations:

Temporal lobe epilepsy in children occurs within several clinical contexts: Structural abnormalities (developmental or acquired), Mesial temporal sclerosis (frequently following prolonged febrile seizures), recognized as a distinct syndrome in contemporary classification systems [52]

THERAPEUTIC APPROACHES

Pharmacological Treatment

Initial management typically involves antiepileptic medications, though complete seizure control often proves challenging. Current evidence shows no single agent demonstrates superior efficacy for focal seizures, though newer medications (oxcarbazepine, levetiracetam, lamotrigine) generally offer better tolerability than traditional options. Clinicians should remain aware of potential cognitive effects (topiramate, zonisamide) and behavioral changes (levetiracetam) associated with certain newer agents [53].

Surgical Options

Patients unresponsive to medical therapy require prompt surgical evaluation, given: Limited success of continued medication trials (3-11% seizure freedom), Significant long-term medication risks (metabolic, endocrine, skeletal), Demonstrated benefits of timely surgical intervention [54, 55].

EEG IN EPILEPSY DIAGNOSIS

Electroencephalography remains fundamental for distinguishing epileptic from non-epileptic events. Characteristic epileptiform patterns include various spike and wave complexes, polyspikes, hypsarrhythmia, and specific ictal patterns, along with benign childhood variants [56,57].

IEDs are relatively frequent in healthy children aged between 1 and 15 years, with a prevalence of approximately 1–2% [58]. They are most often benign focal epileptiform discharges of childhood, typically appearing in the occipital, centrotemporal, and frontal brain regions [59] (Fig.5).

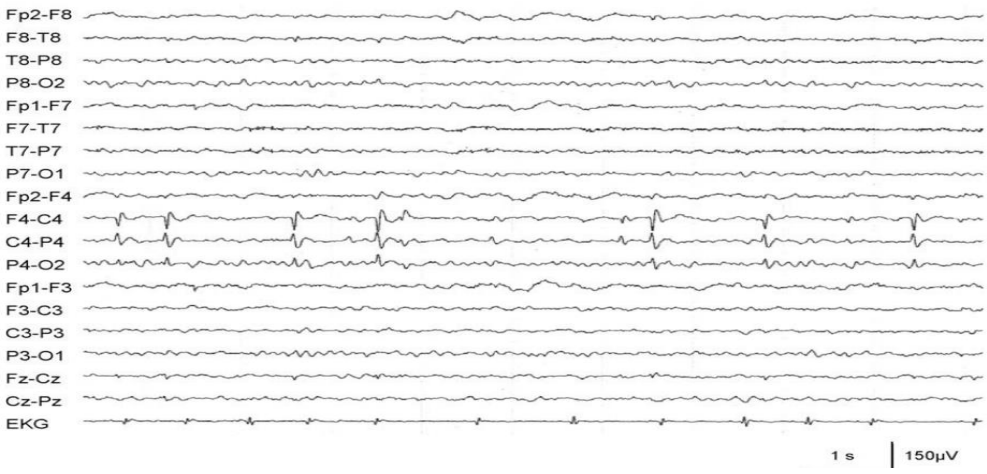

Fig.5. Right central benign epileptiform discharges of childhood shown in a bipolar longitudinal EEG recording. The patient is an 8-year-old child who experienced infrequent generalized tonic–clonic seizures during sleep, which were preceded by clonic movements of the left side of the face [67].

Localization of Epileptogenic Zones Through EEG Patterns

Distinct electroencephalographic signatures correlate with specific epilepsy syndromes, serving as valuable markers for identifying seizure onset zones. These syndrome-specific patterns are systematically categorized in Table 4, which demonstrates their diagnostic utility in localizing epileptogenic foci.

Table .4. Common patterns of interictal epileptiform discharges observed on EEGs in patients diagnosed with specific epilepsy syndromes or underlying causes. [67]

EEG pattern	Epileptic syndrome/etiology
Anterior temporal spikes	Mesial temporal lobe epilepsy
Generalized 3-Hz spike–wave complexes	Absence epilepsy
>4-Hz spike–wave complexes, generalized polyspikes	Juvenile myoclonic epilepsy
Generalized slow spike–wave complexes	Lennox–Gastaut syndrome
Regional (extratemporal) polyspikes	Focal cortical dysplasia
Hypsarrhythmia	West syndrome

Electroclinical Features of Generalized Epilepsies

The 3-Hz spike-wave complex represents the characteristic EEG signature of absence epilepsy, demonstrating both diagnostic and prognostic significance [60]. This epileptiform pattern shows particular pharmacological responsiveness, with optimal therapeutic outcomes achieved using valproate, lamotrigine, or topiramate. Conversely, carbamazepine and tiagabine may paradoxically exacerbate absence seizures.

The clinical correlation of 3-Hz generalized spike-wave discharges depends substantially on discharge duration: Episodes exceeding 4 seconds typically manifest as clinically evident absence seizures. Shorter bursts may preserve some responsiveness, albeit with slowed reaction times (Fig.6). Various generalized epilepsy syndromes demonstrate specific EEG signatures, as detailed in Table 4 [61]. The discharge begins with a more prominent amplitude in the left frontal area. Typically, generalized spike–wave complexes show a shifting dominance between the left and right frontal regions. Although these discharges were present, the patient initially maintained the ability to respond to auditory cues. However, as the discharge progressed, response times became slower, eventually leading to a failure to respond to the final stimulus at the end of the discharge.

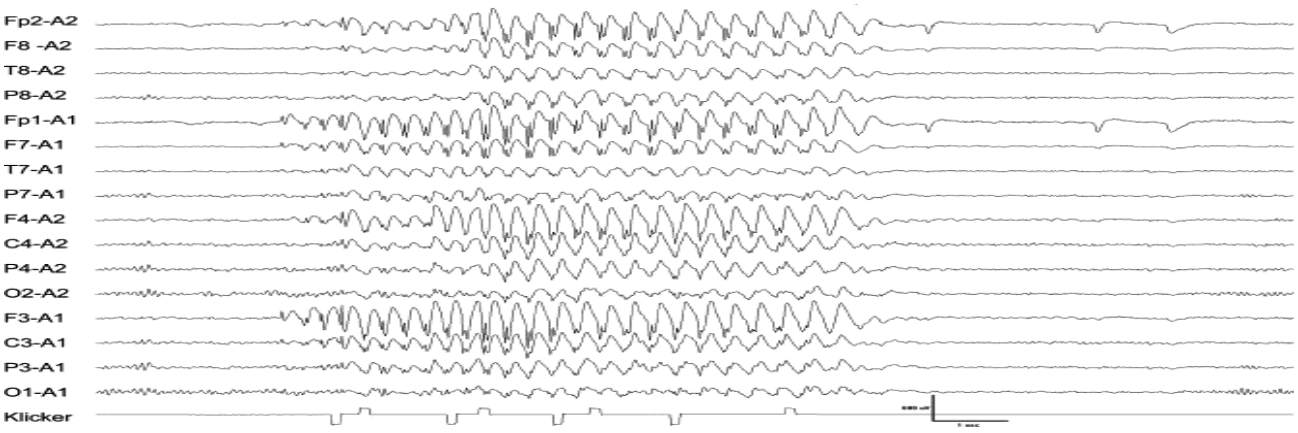

Fig.6. Generalized spike–wave complexes recorded with an ipsilateral ear reference during wakefulness in a 19-year-old individual diagnosed with juvenile absence epilepsy.

Localization in Focal Epilepsies:

Accurate identification of epileptiform discharges plays a pivotal role in determining the epileptogenic zone, especially for surgical candidates [62]. Historical EEG studies first established the association between anterior temporal interictal discharges and temporal lobe epilepsy [63]. While regional EEG slowing lacks specificity for epilepsy diagnosis, rhythmic delta activity (particularly temporal intermittent rhythmic delta activity/TIRDA) provides valuable localizing information in confirmed epilepsy cases [64]. Notably absent in non-epileptic controls, TIRDA's clinical significance has evolved since its original attribution solely to temporal lobe epilepsy [65].

CONCLUSION

Childhood epilepsy is a multifaceted neurological condition that demands a comprehensive, multidisciplinary approach for successful management. Affecting approximately 10.5 million children worldwide, it poses notable medical and social challenges. Recent advancements in diagnostic tools, including EEG and neuroimaging, have enhanced the accuracy of epilepsy classification and guided better treatment strategies. The underlying causes are diverse, ranging from genetic predispositions to brain injuries and infections. Although antiepileptic medications (AEDs) remain the primary treatment option, alternative therapies such as the ketogenic diet, neurostimulation, and surgical interventions are valuable for managing drug-resistant cases. Beyond medical concerns, epilepsy significantly influences the psychological, social, and educational aspects of a child's life. Therefore, early detection, individualized treatment plans, and robust support networks are essential for optimizing outcomes

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