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Research Article

Findings and Diagnostic WorkUp of Pediatric Focal Epilepsies and Nueroimaging

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Abstract

Approximately half of focal epilepsies in childhood that do not fall into a specific self-limited epilepsy syndrome will be due to a known underlying cause. Such children have a higher risk of developing drug-resistant epilepsy and a lower likelihood of epilepsy remission. The vast majority of known causes of focal epilepsy in childhood are structural causes, and thus high-quality, epilepsy protocol brain MRI is mandatory for all cases of focal epilepsy, excepting those meeting criteria for a clear self-limited epilepsy syndrome.

Structural causes can be divided into two groups: developmental, where brain development is abnormal, and acquired. More extensive malformations of cortical (e.g., hemimegalencephaly) development present at an earlier stage in life with developmental delay and epilepsy which is often drug resistant. Some of these infants can present initially with infantile spasms with or without focal seizures but then evolve to focal or multifocal epilepsy over time. Less extensive forms of developmental structural abnormalities such as focal cortical dysplasia (FCD) may present later in childhood or even in adulthood where drug-resistant epilepsy is common.

Among acquired causes, one of the most common etiologies is perinatal brain injury associated with intraventricular hemorrhage and subsequent periventricular leukomalacia in premature infants, and hypoxic–ischemic injury as the major injury in term infants. These infants often present with neonatal seizures which then may settle and recur either as focal seizures or as epileptic spasms. Other types of structural changes can be seen at any age and include stroke, trauma, tumors, for example, low-grade epilepsy-associated tumors including gangliogliomas and dysembryoplastic neuroepithelial tumors (DNETs), post-infectious brain injury after meningitis or encephalitis, or other brain injury. In children who have had a prior prolonged febrile seizure within the first years of life, hippocampal edema can be seen acutely which, over time, can evolve to mesial temporal sclerosis. This is an important etiology as it is often drug resistant and amenable to epilepsy surgery.

For some patients with developmental structural abnormalities of the brain, an underlying genetic cause can be found such as variants with a second brain somatic hit or postzygotic somatic mosaicism only. These include conditions such as Sturge–Weber syndrome, tuberous sclerosis complex, DEPDC5 (DEP domain containing 5, GATOR1 subcomplex subunit) and other genes affecting the mTOR (mammalian target of rapamycin) pathway, or the galactose transporter SLC35A2 (solute carrier family 35 member A2). These children can be considered for epilepsy surgery if it is deemed that seizures are arising from a specific focus.

Metabolic derangements are most commonly associated with generalized seizures as opposed to focal seizures although very young infants can present with focal seizures due to metabolic disorders. Immune-mediated epilepsies are relatively rare in children although autoimmune etiologies, especially anti-NMDA, are often associated with acute symptomatic seizures, especially when concurrent movement disorders are present. Infectious causes such as neurocysticercosis should be considered especially when the MRI is concordant with the clinical diagnosis.

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In this article findings about the diagnostic workup of pediatric focal epilepsies and neueroimaging are considered. Neuroimaging in epilepsy serves different purposes. Early neuroimaging is crucial in emergencies, when a seizure may occur as a secondary manifestation accompanying, for example, focal neurological deficits, cognitive alterations, headache, or fever. Seizures may also occur as a first manifestation of intracranial tumors, steering the patient toward oncological rather than a primarily epileptological diagnostic pathway and treatment. Neuroimaging is performed in the early stage of the diagnostic evaluation of epilepsy to establish a syndromic diagnosis and determine the underlying etiology of recurrent seizures.
Overall, however, imaging reveals an epileptogenic lesion in only around 20% of patients with new-onset seizures. In patients with focal onset seizures, this percentage reaches ~50%but depends on the level of technological sophistication and neuroimaging technique. In general, patients with epilepsy without an identifiable lesion on MRI (i.e., with causes other than structural lesions) have a better prognosis for seizure control with antiseizure medication. In patients with drug-resistant focal epilepsies, structural imaging plays an essential role in the planning of invasive recordings and epilepsy surgery. Presence of an epileptogenic lesion is a significant predictor of successful epilepsy surgery, although complete lesionectomy of the structural lesion alone is not always sufficient to achieve long-term seizure freedom. In contrast, non-lesional patients represent an especially challenging subgroup with comparably limited postsurgical seizure outcomes.

Keywords: *CT, computed tomography; EEG, electroencephalogram; EMU, epilepsy monitoring unit; ESI, EEG source imaging; LTM, long-term EEG monitoring; MEG, magnetoencephalography; MRI, magnetic resonance imaging; MSI, magnetic source imaging; PET, positron emission tomography; SPECT, single photon emission computed tomography. Epilepsy, EEG characteristics, PET scanning, Focal Epilepsy, Magnetic Resonance Imaging.*

INTRODUCTION

Epilepsy is a disorder of the cerebral cortex in which symptoms occur due to an excessive, abnormal, sudden, synchronous discharge of neurons. This abnormal, sudden brain stimulation is intermittent, usually short-term and self-limiting, lasting from a few seconds to a few minutes. Epilepsy should include the occurrence of at least two unprovoked seizures occurring more than 24 hrs apart.

Approximately 10.5 million children under age 15 have epilepsy worldwide.

The prevalence of pediatric patients with Focal epilepsy has been reported to be up to 2.99% in the literature.

Seizures can be categorized as partial or generalized: Seizures originating from a single location in the brain are considered to have a focal onset, known as a partial seizure. Seizure activity originating simultaneously from bilateral hemispheres is considered to have a generalized onset.

Partial seizures are further divided into simple and complex. Simple partial seizures involve a small portion or a focal area of the brain. Complex partial seizures start in one area and travel to another.

The international classification 2017 also divides partial seizures into two: Focal seizure with preserved awareness and focal seizure with impaired awareness. Focal epilepsy is considered to include epileptogenic seizures and to be the type of epilepsy most resistant to antiepileptic drugs.

The leading causes of this form of epilepsy in early childhood are defects acquired during the course of development, hypoxic-ischemic encephalopathy, perinatal infections, neonatal hypoglycemic brain injury, tuberous sclerosis.

Epilepsies due to focal structural lesions are common and easily diagnosed when there is an obvious lesion observed upon neuroimaging, but lesions are not necessarily always visible on conventional MR imaging. Instead, their presence might be inferred from a number of important clinical clues including seizure semiology, neurologic examination, interictal or ictal EEG characteristics, or PET scanning. In this volume, we have grouped the epilepsies based upon the interictal EEG characteristics. Here too, the interictal EEG can be the single most important screening test indicating the presence of a focal structural lesion. Focal slowing, focal attenuation, focal disruption of normal background rhythms, and focal pleomorphic interictal epileptiform discharges are all very suggestive features. Often, focal structural epilepsies will manifest with focal seizures, but not invariably. Diffuse and symmetric ictal manifestations are sometimes seen, in particular, in the very young.

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The converse is also true: focal seizures may be commonly seen in children who do not harbor a focal structural lesion. Therefore, the entire clinical picture, not just the seizure semiology, is important to consider when evaluating children with epilepsy for the presence of focal structural lesions. While the clinical manifestations of focal seizures dramatically change from infants to school-age children, these changes follow some clear rules and therefore are predictable.

AIMS AND OBJECTIVES

AIM: Magnetic Resonance Imaging findings and their association with Electroencephalograph Data in children with Focal Epilepsy.

OBJECTIVES: -

1) To identify the prevalence of Electroencephalographic profile & Radiological spectrum of children with Focal epilepsy patients.
2) To correlate MRI findings and Electroencephalographic profile in children with Focal epilepsy.

REVIEW OF LITERATURE

PATHOPHYSIOLOGY OF EPILEPSY:

Epilepsy is suspected when there is repetition of seizures or an unprovoked seizure is accompanied by a probability of further seizures similar to the general recurrence risk after two unprovoked seizures. Children with epilepsy present multiple challenges to the clinician. The cause and clinical spectrum of epilepsy are extremely wide-ranging in children. Moreover, there are age-specific changes in the semiology/types of seizures that might be the result of the differences in the connectivity and functionality of different brain regions. Epilepsy is more than a repetition of seizure.

DEVELOPMENTAL FACTORS PREDISPOSING BRAIN TO SEIZURES:

Regardless of the underlying factors, such as gene mutation, structural brain abnormalities, or any type of brain injury, several intrinsic factors contribute to a higher susceptibility in the immature brain to generate seizures compared to the mature brain. The developmental changes of the neurotransmitter system in the brain seem to be key factors of the seizure susceptibility. Neurotransmission in early life is characterized by an enhancement of the excitatory system, while the inhibitory system is less efficient or exhibits a paradoxical excitatory effect. A change of the composition of the glutamate receptors during development results in changes in the brain's excitability. The subunit composition of the NMDA receptors in early life promotes extra synaptic localization, slow kinetics (NR2B), decreased calcium in flux (NR3), and decreased sensitivity to magnesium blockade (NR2D, NR3).

ION CHANNELS, MEMBRANES AND MOLECULES IN EPILEPSY:

SUSTAINED REPETITIVE FIRING (SRF) Sustained high-frequency repetitive fi ring is an important property of vertebrate and invertebrate neurons that correlates with the excitability state of the neuron. Many central nervous system (CNS) neurons exhibit SRF. Although no direct evidence has demonstrated the link between SRF and epilepsy, information from in vitro studies on isolated neurons may have some bearing on altered neuronal excitability and anticonvulsant action. SRF is a no synaptic property of neurons.

SUSTAINED REPETITIVE FIRING AND SODIUM CHANNEL REGULATION In studying SRF, several properties of neuronal excitability and seizure phenomenon have been recognized. Voltage-gated sodium channels are responsible for the rising phase of neuronal action potentials. Upon neuronal depolarization to action potential threshold, the sodium channel undergoes a conformational change that results in the channel opening for a few milliseconds from its closed (resting), nonconducting state to permit sodium flux. The channel inactivates within a few milliseconds to terminate sodium ion influx.

BENZODIAZEPINE RECEPTORS AND MEMBRANE EXCITABILITY In the 1970s, use of radioactively labeled benzodiazepine derivatives allowed the detection of specific nanomolar benzodiazepine receptor sites in brain membrane. These sites have a very high affinity for the benzodiazepines, binding in low (nanomolar) concentration ranges. Binding to these receptors is reversible, saturable, and stereospecific. Nanomolar benzodiazepine receptors have now been identified in the human brain, where they are widely distributed.

THE GABA SYSTEM, NEURONAL EXCITABILITY, AND SEIZURE ACTIVITY Gamma-aminobutyric acid is the major inhibitory neurotransmitter in the brain. It has been extensively characterized and plays a major role in regulating neuronal excitability by controlling chloride permeability. Specific binding sites for GABA molecules have been identified in the neuronal membrane. Although not all GABA receptors are linked to the chloride channel, a large proportion of these receptors are directly involved in regulating chloride channel function.

EXCITATORY TRANSMISSION Glutamate is the major excitatory neurotransmitter in the brain. Understanding the role of excitatory transmission and its over-activation in epilepsy is an important area for anticonvulsant drug development. Only since the early 1980s has the role of glutamate, aspartate, and other compounds that serve as

excitatory transmitters been clearly identified. Several important receptors, which respond to glutamate and other excitatory neurotransmitters, have been identified in the brain. The major glutamate receptors that regulate ion channels include the N-methyl-D-aspartate (NMDA), quisqualate or alpha-amino-5-methyl-3-hydroxy-4- isoxazole propionic acid (AMPA), and kainate channels. In addition, there are other, more recently identified subcategories of excitatory amino acid ion channels regulated by glutamate.

METABOTROPIC RECEPTORS Metabotropic receptors are glutamate- or excitatory amino acid-activated receptors that are not coupled to ion channels. These excitatory amino acid receptors are coupled to second messenger systems in the membrane that have an important role in regulating cellular metabolism and function.

CARBONIC ANHYDRASE INHIBITION Carbonic anhydrase (CA) is a major enzyme system that has been found to regulate GABA-mediated inhibitory potentials and therefore has important anticonvulsant or antiepileptic effects. GABA receptor ion channels are permeable to both chloride and bicarbonate ions.

NEUROMODULATORS There are many classes of neuromodulators, but two major classes have been widely studied that have significant implications in regulating seizure activity: adenosine and the monoamines. Both adenosine and monoamines in fluence seizures and epileptogenesis in several models of epilepsy. Adenosine is released during seizure activity and acts as an endogenous anticonvulsant.

CALCIUM REGULATION OF NEURONAL FUNCTION Calcium plays a major role in modulating the normal activity and function of the nervous system. One of its most widely recognized roles is modulating synaptic neurotransmission. A host of studies have demonstrated the importance of calcium in stimulus- secretion coupling. In addition to its important effects on neurotransmission, calcium plays a major role as a second messenger in neuronal and nonneuronal tissues.

Excitatory Amino Acid Receptors and Calcium Channels L-glutamate was proposed as an excitatory neurotransmitter over 30 years ago. Recently, excitatory amino acids (EAA) have been found to play important roles in epilepsy, neuronal excitability, and learning. The two main excitatory neurotransmitters currently known are glutamate and aspartate. Many pathways in the brain use these neurotransmitters, including hippocampal afferents and major cortical output tracts that are widely activated during convulsions.

FOCAL EPILEPSY:

Epilepsies due to focal structural lesions are common and easily diagnosed when there is an obvious lesion observed upon neuroimaging, but lesions are not necessarily always visible on conventional MR imaging. Instead, their presence might be inferred from a number of important clinical clues including seizure semiology, neurologic examination, interictal or ictal EEG characteristics, or PET scanning. In this volume, we have grouped the epilepsies based upon the interictal EEG characteristics. Here too, the interictal EEG can be the single most important screening test indicating the presence of a focal structural lesion. Focal slowing, focal attenuation, focal disruption of normal background rhythms, and focal pleomorphic interictal epileptiform discharges are all very suggestive features.

PATHOLOGY An international survey of surgical cases that was published in 2008 provides an excellent, though somewhat biased, view of the cause of focal structural epilepsy. The most common causes were cortical dysplasia followed by low grade tumors, which together account for more than 60% of the etiologies. Other causes in descending frequency were atropy/stroke, hippocampal sclerosis, non-specific gliosis, tuberous sclerosis complex, hypothalamic hamartoma, Sturge-Weber, Rasmussen, and vascular lesions. The commonest tumors were DNETs and gangliogliomas. The commonest location was the temporal, followed by the frontal, and there were significantly fewer parietal and occipital foci.

Epileptic Spasms with Asymmetric Features Spasms can be recognized by their tendency to recur in clusters, many times in an almost periodic fashion, with a fairly constant interval between some of the individual spasms. Spasms have a quick or myoclonic component at the start, followed by a brief sustained posture (tonic phase), followed in turn by a relaxation. Spasms that are asymmetric, that occur in a child with hemiparesis or other focal pathology, or that are associated with marked interhemispheric asymmetries on EEG could indicate a focal process. As an incidental note, focal electrographic seizures may occur in conjunction with infantile spasms, but one should be cautious about overinterpreting their localizing value unless they are very consistent and there is other supportive evidence to suggest a focal process. The EEG accompaniment of spasms often contains diffuse electro decrements, even if they are preceded by clear focal seizures.

Versive Pronounced and sustained lateral version of the eyes (versive seizure) is rarely encountered as an ictal manifestation in infants or young children. When present, version is another indicator of a focal seizure.

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Tonic postures, both symmetric and asymmetric, are seen with focal seizures. It is surprising to observe how often symmetric tonic postures can occur as a manifestation of a focal seizure in infants, and also how unreliable asymmetries of tonic postures can be in localizing ictal onsets. It is possible that these tonic postures are generated in deeper brainstem or subcortical structures and are not direct manifestations of the ictal discharges. Most common etiologies of focal epilepsy syndromes in children.

Self-limited focal epilepsy syndromes	Focal epilepsy of known cause	Focal epilepsy of unknown cause
Self-limited *neonatal* epilepsy (*KCNQ2*) Self-limited *infantile* epilepsy (*PRRT2*, *SCN2A*, *SCN8A*) Self-limited epilepsy with autonomic seizures Self-limited epilepsy with centrotemporal spikes	Structural, developmental (malformations of cortical development; possible genetic origin: e.g., *DEPDC5* and *TSC1/TSC2*) Structural and/or acquired Perinatal brain injury Hypoxic–ischemic injury Stroke Trauma Tumor Vascular malformation Post-infectious Hippocampal injury/FS Immune-mediated (anti-NMDAR)	

Self-limited focal epilepsy syndromes

Specific self-limited focal epilepsy syndromes have been recently defined by the ILAE in both neonates and infants[5] and children. The most common types are outlined below.

Self-limited neonatal epilepsy usually begins in the 1st week of life with focal tonic or clonic seizures in an otherwise healthy baby. There may be a positive family history of similarly affected neonates. Most of these babies will have a pathogenic variant in *KCNQ2* (potassium voltage-gated channel subfamily Q member 2). This epilepsy syndrome typically resolves by 6 weeks of age.

Self-limited infantile epilepsy begins after the neonatal period but usually before 18 months of age. Focal seizures consist of behavioral arrest, impaired awareness, versive, or focal clonic movements. These seizures often occur in brief clusters where several seizures occur over a few days and, rarely, they can evolve to bilateral tonic–clonic seizures. Infants have normal development and imaging and nearly all cases resolve by 3 years of age. Several genetic variants have been found for this syndrome, mainly *PRRT2* (proline-rich transmembrane protein 2) but also *SCN2A* (sodium voltage-gated channel alpha subunit 2) and *SCN8A* (sodium voltage-gated channel alpha subunit 8).

Self-limited epilepsy with autonomic seizures (SeLEAS; formerly known as Panayiotopoulos syndrome) most commonly begins in preschool-aged children. Seizures often occur shortly after falling asleep and have prominent autonomic features, in particular recurrent retching. The seizure frequency is low but about a quarter of patients can have prolonged seizures. Remission typically occurs within 1–2 years of onset. Imaging and genetic studies are typically normal.

Self-limited epilepsy with centrotemporal spikes (SeLECTS) presents in school-aged children with focal seizures affecting the lower face with prominent drooling, dysarthria, and facial twitching. Seizures are usually seen shortly after falling asleep or shortly before awakening and during sleep, when they may evolve to bilateral tonic–clonic seizures. EEG shows characteristic high-amplitude centrotemporal epileptiform discharges. The majority of patients' seizures resolve by age 12 years, and nearly all patients' seizures remit by age 16 years. Imaging and genetic studies are typically normal.

Focal epilepsy of unknown cause but which do not meet criteria for a self-limited focal epilepsy syndrome

Approximately half of children with focal epilepsies that begin in childhood but do not meet criteria for one of the self-limited syndromes will have no known cause found despite careful evaluation including brain MRI. Overall, these patients have a very favorable prognosis with regard to seizures, with one population-based long-term study showing that 81% of patients achieve seizure freedom and 68% can be weaned off antiseizure medication(s). Favorable outcome is particularly likely if the child has a normal development and a normal neurologic examination.

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Focal epilepsy of known cause

Approximately half of focal epilepsies in childhood that do not fall into a specific self-limited epilepsy syndrome will be due to a known underlying cause. Such children have a higher risk of developing drug-resistant epilepsy and a lower likelihood of epilepsy remission. The vast majority of known causes of focal epilepsy in childhood are structural causes, and thus high-quality, epilepsy protocol brain MRI is mandatory for all cases of focal epilepsy, excepting those meeting criteria for a clear self-limited epilepsy syndrome.

Structural causes can be divided into two groups: developmental, where brain development is abnormal, and acquired. More extensive malformations of cortical (e.g., hemimegalencephaly) development present at an earlier stage in life with developmental delay and epilepsy which is often drug resistant. Some of these infants can present initially with infantile spasms with or without focal seizures but then evolve to focal or multifocal epilepsy over time. Less extensive forms of developmental structural abnormalities such as focal cortical dysplasia (FCD) may present later in childhood or even in adulthood where drug-resistant epilepsy is common.

Among acquired causes, one of the most common etiologies is perinatal brain injury associated with intraventricular hemorrhage and subsequent periventricular leukomalacia in premature infants, and hypoxic–ischemic injury as the major injury in term infants. These infants often present with neonatal seizures which then may settle and recur either as focal seizures or as epileptic spasms. Other types of structural changes can be seen at any age and include stroke, trauma, tumors, for example, low-grade epilepsy-associated tumors including gangliogliomas and dysembryoplastic neuroepithelial tumors (DNETs), post-infectious brain injury after meningitis or encephalitis, or other brain injury. In children who have had a prior prolonged febrile seizure within the first years of life, hippocampal edema can be seen acutely which, over time, can evolve to mesial temporal sclerosis. This is an important etiology as it is often drug resistant and amenable to epilepsy surgery.

For some patients with developmental structural abnormalities of the brain, an underlying genetic cause can be found such as variants with a second brain somatic hit or postzygotic somatic mosaicism only. These include conditions such as Sturge–Weber syndrome, tuberous sclerosis complex, DEPDC5 (DEP domain containing 5, GATOR1 subcomplex subunit) and other genes affecting the mTOR (mammalian target of rapamycin) pathway, or the galactose transporter SLC35A2 (solute carrier family 35 member A2). These children can be considered for epilepsy surgery if it is deemed that seizures are arising from a specific focus.

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DIAGNOSTIC WORKUP OF FOCAL EPILEPSIES

The following sections will address the diagnostic workup of focal epilepsies including neuroimaging, neurophysiology, and neuropathology. The below table summarizes the auxiliary workup modalities in the management of patients with focal epilepsies.

Summary of auxiliary workup modalities in the management of focal epilepsies.

Neuroimaging	Neurophysiology	Neuropathology
CT (emergency patients with first seizure, widely available) MRI (recommended for all patients with epilepsy; ideally HARNESS protocol with or without MRI post-processing) Interictal PET Ictal SPECT Functional imaging (spatial distribution of functional cortex; MRI and/or MEG)	Routine EEG Routine EEG, sleep-deprived Ambulatory EEG Inpatient long-term video-EEG monitoring (LTM/EMU) ESI/MSI	Standardized operational procedure (SOP) for inspection, distribution, processing of epileptogenic brain tissue Routine light microscopic assessment with hematoxylin–eosin or cresyl violet or luxol-fast-blue stainings Antibodies to identify aberrant immunoreactivity patterns of disease-specific protein epitopes

Abbreviations: CT, computed tomography; EEG, electroencephalogram; EMU, epilepsy monitoring unit; ESI, EEG source imaging; LTM, long-term EEG monitoring; MEG, magnetoencephalography; MRI, magnetic resonance imaging;

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MSI, magnetic source imaging; PET, positron emission tomography; SPECT, single photon emission computed tomography.

DIAGNOSTIC WORKUP OF FOCAL EPILEPSIES, NEUROIMAGING

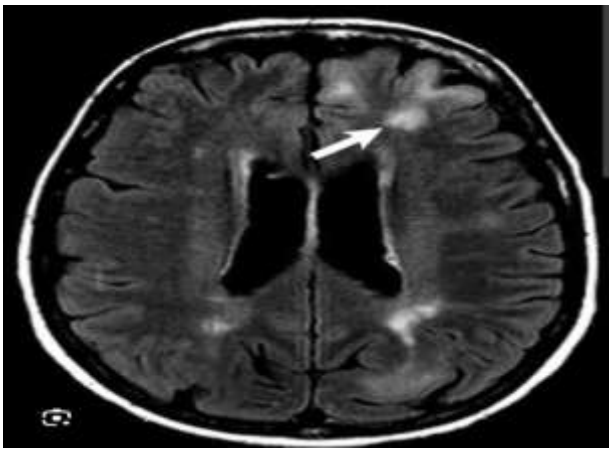
Significance and indications

Neuroimaging in epilepsy serves different purposes. Early neuroimaging is crucial in emergencies, when a seizure may occur as a secondary manifestation accompanying, for example, focal neurological deficits, cognitive alterations, headache, or fever. Seizures may also occur as a first manifestation of intracranial tumors, steering the patient toward oncological rather than a primarily epileptological diagnostic pathway and treatment. Neuroimaging is performed in the early stage of the diagnostic evaluation of epilepsy to establish a syndromic diagnosis and determine the underlying etiology of recurrent seizures.

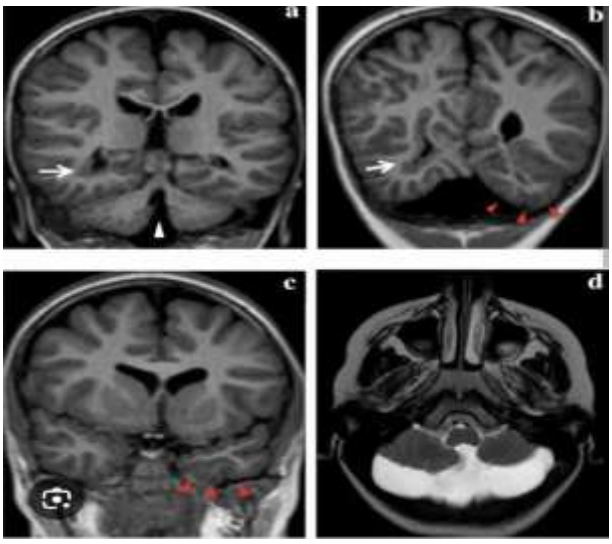
Overall, however, imaging reveals an epileptogenic lesion in only around 20% of patients with new-onset seizures. In patients with focal onset seizures, this percentage reaches ~50% but depends on the level of technological sophistication and neuroimaging technique. In general, patients with epilepsy without an identifiable lesion on MRI (i.e., with causes other than structural lesions) have a better prognosis for seizure control with antiseizure medication. In patients with drug-resistant focal epilepsies, structural imaging plays an essential role in the planning of invasive recordings and epilepsy surgery. Presence of an epileptogenic lesion is a significant predictor of successful epilepsy surgery, although complete lesionectomy of the structural lesion alone is not always sufficient to achieve long-term seizure freedom. In contrast, non-lesional patients represent an especially challenging subgroup with comparably limited postsurgical seizure outcomes.

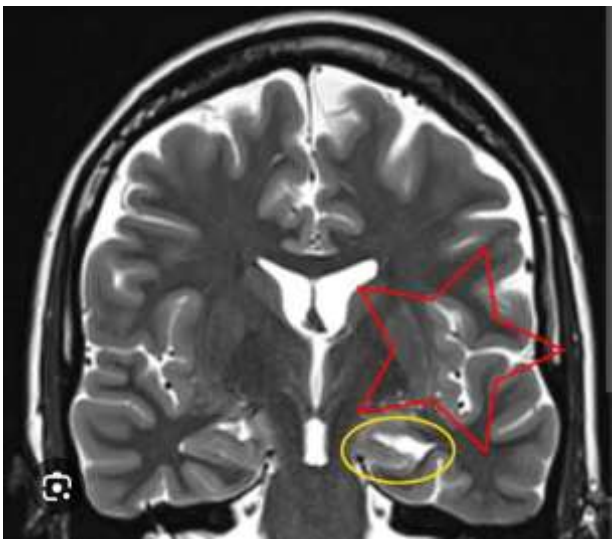
Structural imaging: MRI And CT

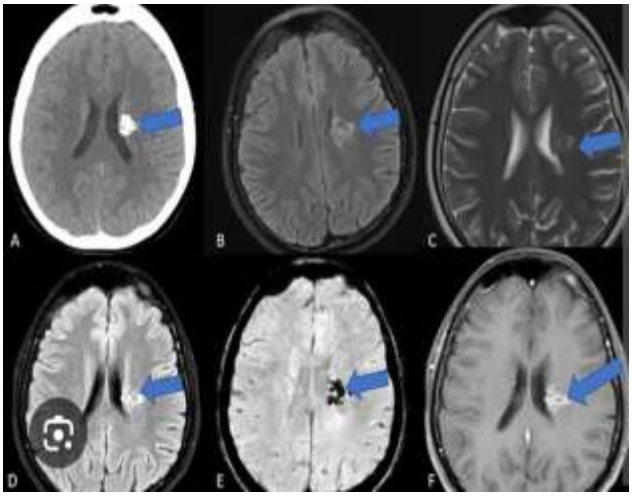
CT is recommended in emergency patients with a first seizure and is widely available worldwide. It is sensitive to bleedings, but also small calcified and bone lesions as well as skull defects (e.g., in patients with encephalocele). However, due to better differentiation of brain tissues, all patients with focal epilepsy should undergo at least a 1.5 T MRI using a protocol optimized for the evaluation of epilepsy, while the application of CT is limited to special situations. Patients should undergo repeated MRI if previous imaging did not follow an optimized protocol. Furthermore, children with focal seizures and unrevealing MRI performed below the age of 1 year should undergo repeated MRI.



TUBEROUS SCLEROSIS







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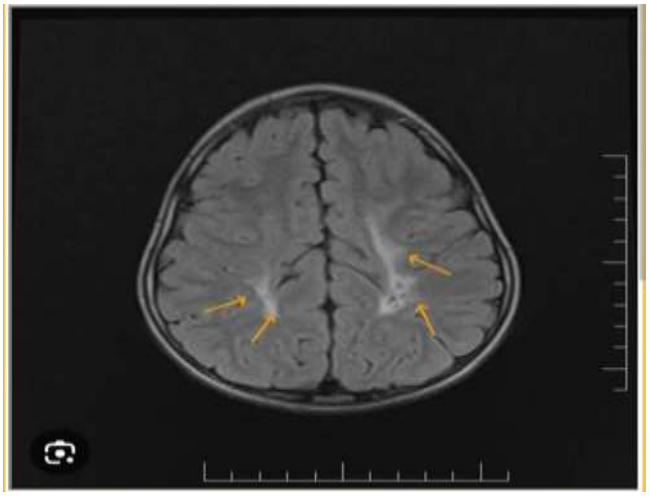
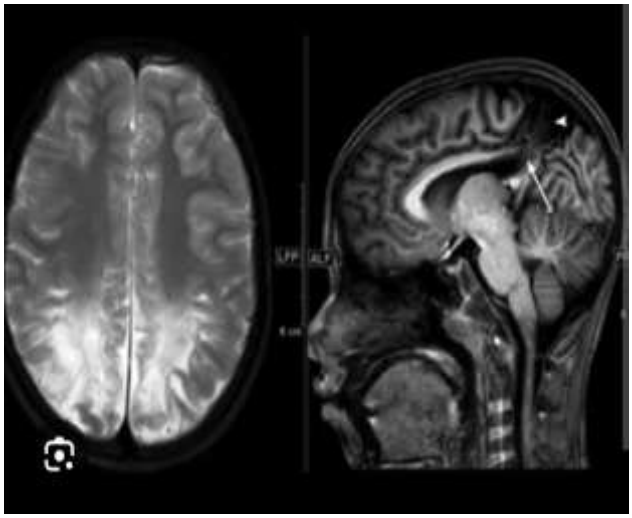
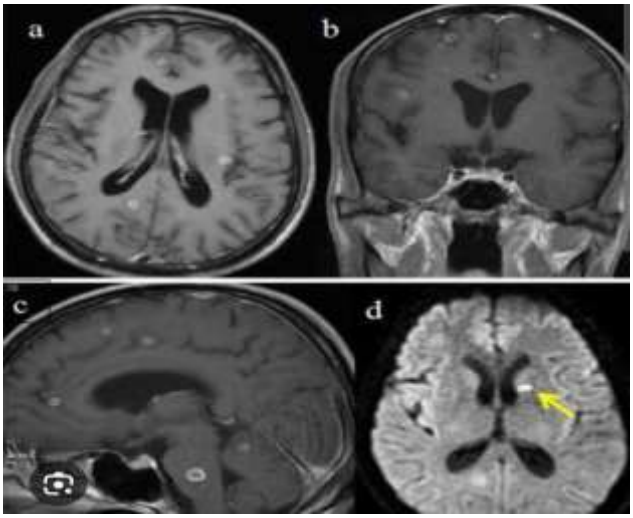


PERIVENTRICULAR LEUKOMALACIA



OCCIPITAL GLIOSIS



TUBERCULOMA

MRI: The HARNESS protocol

The ILAE Neuroimaging Task Force has recently recommended the "HARNESS" MRI protocol ("Harmonized Neuroimaging of Epilepsy Structural Sequences"), which allows detection of the complete range of epileptogenic lesions, while limiting the required scanning time. This protocol includes three mandatory and two optional sequences. HARNESS leverages the advantages of isotropic 3D MRI acquisition without interslice gap to allow flexible re-slicing. A high resolution of 1 mm or better is suggested to reduce partial volume effects. In combination, this eliminates the need for pathology-specific sequences and angulations. For inspection of the hippocampus, a 2D T2-weighted series angulated perpendicularly to the long axis of the hippocampus with increased intra-slice resolution is suggested. Two optional series provide additional sensitivity in specific cases: gadolinium-enhanced 3D T1 to assess tumors, vascular malformations, and infectious processes, and susceptibility-weighted SWI/T2*, which is sensitive to iron deposits, blood products, and calcifications. The protocol is recommended for field strengths of 3 T however the sequences can also be obtained with 1.5 T. Even when acquired with an optimal HARNESS protocol, the MRI interpretation should consider the context of the EEG findings, semiology, and other clinical information. Experience of the examiner is also fundamental, and an MRI reported as normal might really harbor a subtle structural abnormality not initially identified by the interpreter, in particular FCDs.

Mandatory and optional sequences of the HARNESS MRI-protocol.

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Name	Weighting	Resolution	Advantages
Mandatory sequences			
Magnetization-prepared rapid gradient echo (MPRAGE, Siemens), Spoiled gradient echo (SPGR, GE), Turbo field echo (TFE, Phillips)	T1	3D 1 × 1 × 1 mm, no interslice gap	Allows for reformatting to different angulations and view planes, visualization of brain anatomy and morphology
3D fluid attenuation inversion recovery (FLAIR)	T2-FLAIR	3D 1 × 1 × 1 mm, no interslice gap	Allows for reformatting to different angulations and view planes, fluid attenuation enhances visibility of lesions such as FCD, hippocampal sclerosis, and tubers
Coronal spin echo (acquisition plane perpendicular to long axis of the hippocampus)	T2	2D 0.4 × 0.4 × 2 mm	High in-plane resolution and coronal view plane provides optimal visualization of hippocampal internal structure
Optional sequences			
Gadolinium-enhanced MRI	T1	3D	Contrast agent improves assessment of tumors, vascular malformations, or infectious processes
Susceptibility-weighted imaging	T2*/SWI	3D	Sensitive to iron deposits, blood products and calcifications, e.g., for assessment of cavernomas

The visual inspection of digitally stored 3D MRIs (usually in DICOM format), acquired with the smaller isotropic voxel size as possible, should be done using a platform that allows multiplanar reformatting. There are several options of user-friendly DICOM viewers that run on most desktop computers and can be incorporated into the routine of epileptologists.

MRI post-processing

In patients with subtle lesions, visual analysis of MRI may not be sufficient. Post-processing techniques may offer solutions by computationally analyzing morphological MRI features, such as sulcal depth, contrast of the gray-white matter junction, volumes, and cortical thickness, among others. A comparison to healthy controls then provides objective metrics of structural alterations potentially providing evidence for a subtle lesion. The use of artificial intelligence approaches in parallel to the visual analyses may enhance further the detection of very subtle FCDs in previously negative MRIs.

PET and SPECT

Positron emission tomography (PET) and single photon emission computed tomography (SPECT), imaging methods that are mainly used in the presurgical evaluation context, provide additional localizing information especially in selected (e.g., non-lesional) cases. Interictal PET, currently largely fluorodeoxyglucose (FDG)-PET, can reveal decreased glucose metabolism, which may be observed in the seizure onset zone. SPECT evaluates regional cerebral blood flow, which is increased during a seizure. If the radioactive SPECT tracer is injected at seizure onset or shortly thereafter, an ictal SPECT may localize the seizure onset zone. Subtraction of ictal-interictal SPECT co-registered with MRI (SISCOM) has shown increased specificity relative to interictal SPECT alone and is less informative especially in extratemporal lobe epilepsy.

Functional imaging

Functional imaging with fMRI or magnetoencephalography (MEG), also primarily used in the presurgical evaluation context, provides information about the spatial distribution of functional cortex (e.g., motor and sensory cortex) as well as language-related areas. The aim of epilepsy surgery is to ultimately achieve improvement in one's quality of life. Consequently, permanent functional deterioration from the effect of recurrent seizures must be avoided. However, individual functional anatomy is variable and epileptogenic lesions may lead to considerable reorganization, especially when they occur early in life. Functional imaging thus contributes to evaluating the viability of surgical therapy and aids

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planning of resection extents. Additional information from functional multimodal imaging improves MRI detection of subtle lesions.

DIAGNOSTIC WORKUP OF FOCAL EPILEPSIES, NEUROPHYSIOLOGY

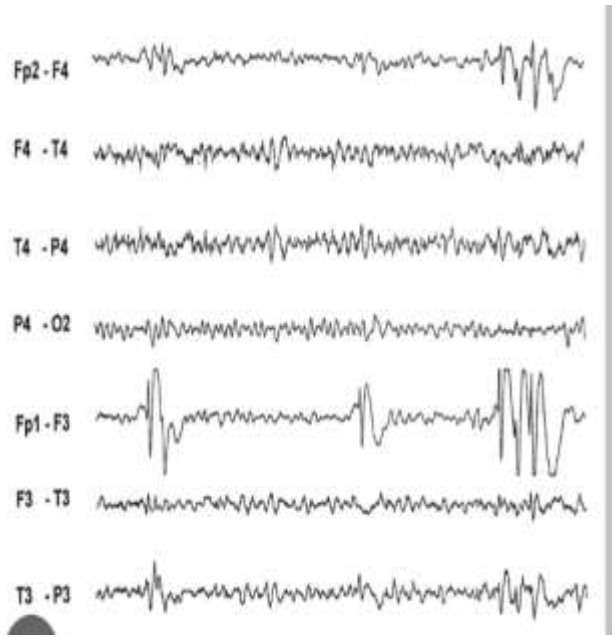
Confirmation of focal epilepsy

Once a diagnosis of focal epilepsy is considered, EEG can be used to confirm or refute clinical suspicions, and in some cases establish an electroclinical diagnosis: a clinical diagnosis supported by EEG. Several factors, however, limit the use of EEG as an auxiliary test in the diagnosis of focal epilepsy.

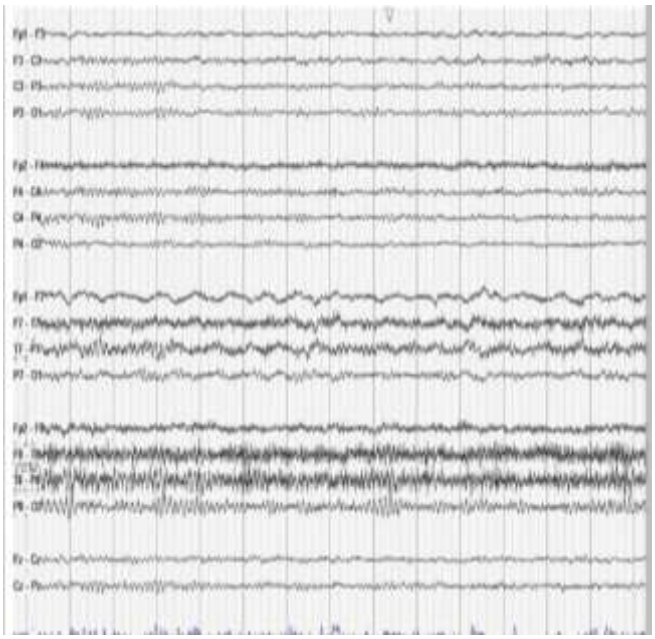
First, EEG is a low-amplitude, continuous and dynamic biosignal that requires considerable expertise for interpretation. The EEG signal recorded at the scalp reflects the summed potentials generated in a relatively large area (≥6–10 cm2) of the cortex at the cerebral convexity and, therefore, represents only a small portion of brain activity of clinical interest. Multiple non-brain sources further lower the signal to noise ratio (SNR). This low SNR is partly responsible for high inter-observer variability. Furthermore, there is a risk of over- or under-reading EEG findings. The former constitutes misinterpreting normal variants, artifacts, or other clinically irrelevant transients as epileptiform. The latter constitutes dismissing a subtle but true epileptiform abnormality. Even experienced EEG readers may still have a practice style of interpreting with “high sensitivity” or with “high specificity.” “Conservative” EEG reading has been recommended to achieve high specificity and avoid over-reading with resultant epilepsy misdiagnosis.

Second, epileptiform abnormalities can occur in the EEG of up to 4% of school-aged children in the absence of epilepsy, that is, on average 1 child in every classroom of 30 children if everyone were to undergo an EEG without indication. This fraction increases in children with comorbid learning disabilities, attention deficit hyperactivity disorder, and autism spectrum disorder.

In clinical practice, therefore, the diagnostic yield of an EEG depends on the prior (or pre-test) probability. According to Bayes' theorem for calculating conditional probabilities, a clinician should update prior information (e.g., is focal epilepsy present in this patient?) whenever new and relevant information is provided (e.g., the EEG is abnormal and contains temporal epileptiform discharges). In clinical practice, evidence and information are presented sequentially, not all at once. For example, a neurologically healthy toddler with a spell of brief unresponsiveness and an EEG with one focal epileptiform discharge may still not have epilepsy. The same EEG, however, could be supportive of an epilepsy diagnosis when the pre-test probability is higher: a new event occurs, or a more detailed history is provided by an eyewitness in the same patient. The EEG has important limitations, and the results should be interpreted within the appropriate clinical context of the patient.



FOCAL SPIKE WAVE



FOCAL SLOWING

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The diagnostic yield of EEG can be improved in various ways. If the first routine EEG is normal, consider obtaining a repeat routine EEG that captures sleep and, ideally, is recorded following sleep deprivation. Ambulatory EEG is a good tool to capture major "spells," but it can be unrevealing when brief subtle events occur. In addition, the quality of ambulatory EEG recordings may deteriorate over time which can prevent assessment of interictal findings. Children, uncooperative patients, and prolonged recordings may be susceptible to artifact obscuring meaningful interpretation. Inpatient long-term video-EEG monitoring in a dedicated epilepsy monitoring unit is the gold-standard to diagnose, classify, quantify, and characterize patients referred for uncontrolled seizures when the event of interest is captured. Prolonged high-quality assessment of both background and interictal patterns provide supportive information in conjunction with ictal EEG.

Interictal EEG also has an important role in the diagnosis of epilepsy. When seizures are highly suspicious from a clinical standpoint, the diagnosis of epilepsy is a clinical one that can be made irrespective of the presence or absence of an epileptiform EEG. In these cases, however, EEG can be helpful in classifying epilepsy—focal, generalized, or combined generalized and focal, or unknown—with respective treatment implications. If spells are of moderate clinical suspicion for epileptic seizures, the presence of interictal epileptiform abnormalities on EEG establishes the diagnosis of epilepsy. The absence of interictal epileptiform abnormalities on EEG though does not rule out the diagnosis of epilepsy. When spells are of low clinical suspicion, the presence of interictal epileptiform abnormalities on EEG may increase the likelihood of epilepsy.

MATERIALS AND METHODS

- **Study setting:**

This study will be conducted in BYL Nair hospital in Pediatric department where the patient will be enrolled as cases from Pediatric OPD, Pediatric general ward, Pediatric Neurology Epilepsy OPD and Neurodevelopmental OPD.

- **Study design:**

This Retrospective study will be carried out at the Pediatric Neurology, Epilepsy and Neurodevelopmental centre in a tertiary care hospital in a metropolitan city in india over a period of 6 months from last 5 years DATA. The study will be initiated after the approval from institutional review board and data from the past records will be enrolled

- **Study duration: 6 months**

Study population: This study will be conducted in BYL Nair hospital in Pediatric department where the patient will be enrolled as cases from Pediatric OPD, Pediatric general ward, Pediatric Neurology Epilepsy OPD and Neurodevelopmental OPD.

- **Inclusion criteria:**
  1) Children aged from birth to 18 years of both sex will be included in study.
  2) Children with Focal epilepsy previously diagnosed will be included in the study.
- **Exclusion criteria:**
  1) Those children with generalized epilepsy will be excluded.
  2) Those children with acute symptomatic seizure occurring due to fever or with any metabolic abnormalities like hypoglycaemia, hypocalcemia and dyselectrolemia are excluded.
  3) Children with acute cerebral insults like stroke, acute intoxication, head injury, acute encephalitis, CNS infections were excluded from the study.

SAMPLE SIZE CALCULATION:

- The prevalence of focal seizure from the literature is 2.99%.
- P= 2.99%
- Q=97%
- D=5%
- N= 46.4
- Round off to 50
- Final sample size- 50
- Formula: $N=4pq/d^2$

Methodology:

- This study is a Retrospective study in the Pediatric Neurology, Epilepsy and Neurodevelopmental centre at the tertiary care hospital in a metropolitan city.
- After obtaining clearance from the institutional ETHICS COMMITTEE review board, children diagnosed previously with Focal epilepsy with both EEG and MRI done will be enrolled as cases in the study.
- The investigator will fill a detailed case record form from the past records. A detailed medical and neurological history, baseline details including age, sex demographic details, family structure(joint/nuclear) will be noted.
- Detailed General examination and Central nervous system examination will be recorded.
- All the DATA will be recorded in the case record form and entered into master chart by the investigator.

STATISTICAL ANALYSIS:

- The data will be analysed using statistical package for social sciences software, version 25.0(SPSS)
- Results will be presented in the form of tables and graphs.
- Descriptive statistics will be applied to examine Electroencephalogram, clinical and radiological findings.
- It includes frequency, percentage, mean and standard deviations.

RESULTS

Table 1 shows findings in focal seizure.

Table 1: Findings in focal seizure (n=50)

		Focal seizures	
		No.	*%*
Age Group	Upto 1 yr.	3	6.0%
	>1 to 3 yrs.	7	14.0%
	>3 to 6 yrs.	15	30.0%
	>6 to 9 yrs.	13	26.0%
	>9 to 12 yrs.	6	12.0%
	>12 to 15 yrs.	5	10.0%
Sex	Male	38	76.0%
	Female	12	24.0%
History	Birth Asphyxia	8	16.0%
	Neonatal Seizures	11	22.0%
	Family History of Seizure	13	26.0%
	Global Developmental Delay	15	30.0%
	Microcephaly	13	26.0%
	Abnormal CNS Examination	11	22.0%

Table 2 shows Correlation of MRI and EEG findings.

Table 2: Correlation of MRI and EEG findings (n=50)

		EEG					
		Normal		Abnormal		Total	
		No.	%	No.	%	No.	%
MRI	Normal	5	10%	7	14.0%	**12**	24.0%
	Abnormal	5	10.%	33	66.0%	**38**	76.0%
MRI	Total	**10**	20.0%	**40**	80%	50	100.0%

Fischer's test: χ^2=7.214, p=0.014

Spearman's correlation: r=0.380, p=0.007

EXPLANATION:

- -Majority of them had abnormal EEG with abnormal MRI changes(66%), minimum number of them had normal EEG and abnormal MRI brain changes(10%), some of them had normal MRI brain and normal EEG changes(10%), some of them have abnormal EEG and normal MRI brain changes(14%).

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Figure 1 shows MRI and EEG findings in patients.

Figure 1: MRI and EEG findings in patients (%, n=50)

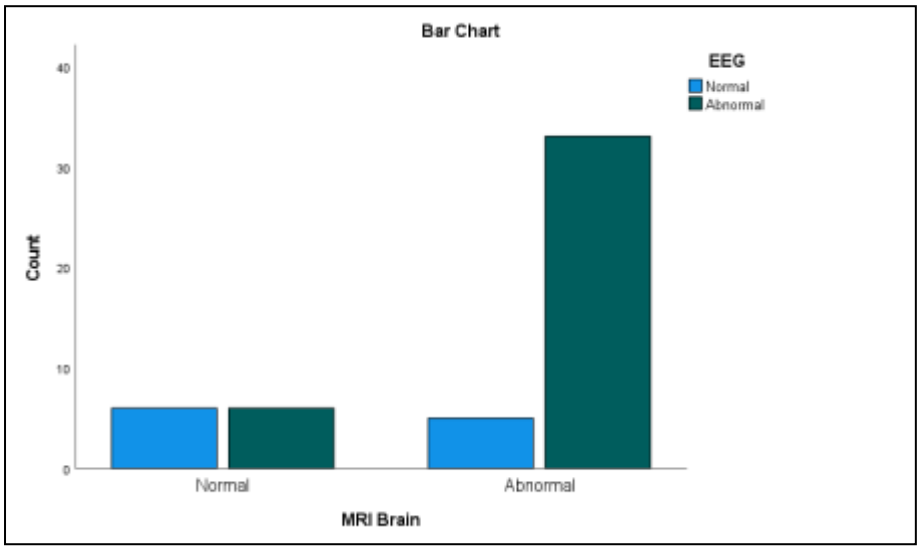

Table 3 shows EEG and MRI abnormalities in focal seizure.

Table 3: EEG and MRI abnormalities in focal seizure (n=50)

		Focal seizures	
		No.	*%*
EEG abnormality	Abnormal findings	40	80%
	Focal Epileptiform Activity	40	80%
	Generalised Epileptiform Activity	1	2.0%
	Frontal Epileptiform Activity	25	50.0%
	Occipital Epileptiform Activity	24	48.0%
EEG ABNORMALITY	Parietal Epileptiform Activity	21	42.0%
	Temporal Epileptiform Activity	22	44.0%
	Focal Slow Wave	19	38.0%
	Focal Spike Wave	33	66.0%
	Focal Spikes and Polyspikes	16	32.0%
	Rhythmic Delta Activity	6	12.0%
	Other Discharges	3	6.0%
MRI Brain abnormality	Abnormal findings	38	76.0%
	Focal Cortical Dysplasia	5	10.0%
	Hippocampal Sclerosis	3	6.0%
MRI ABNORMALITY	Heterotropia	5	10.0%
	Congenital Malformation	7	14.0%
	Focal Gliosis	11	22.0%
	Encephalomalacia/Leukomalacia	14	28.0%
	Space Occupying Lesion	1	2.0%
	Basal Ganglia Involvement	1	2.0%
	Calcification	3	6.0%
	Miscellaneous Group	0	0.0%

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PROFILE OF PATIENTS BASED ON EEG CHANGES:

Out of total cases majority of them had abnormal EEG changes(80%), out of them majority of them had focal epileptiform activity(80%), least number of them had generalized epileptiform activity(2%), some of them had frontal epileptiform activity(50%), some had occipital epileptiform activity(48%), some had parietal epileptiform activity(42%), some had temporal epileptiform activity(44%), some had focal slow wave(38%), some had focal spike wave(66%), some had focal spikes and polyspikes(32%), some had rthymic delta activity(12%).

PROFILE OF PATIENTS BASED ON MRI CHANGES:

Out of total cases, majority (76%) of them had abnormal MRI findings. Among them, majority of them had encephalomalacia/leukomalacia, least number of them had space occupying lesion (2%) and basal ganglia involvement(2%), some of them had hippocampal sclerosis(6%), some had focal cortical dysplasia(10%), some of them had heterotropia(10%), some of them had congenital malformation(14%), some of them had focal gliosis(22%), some had calcification(6%).

Table 4 MRI and EEG abnormalities in different sub-groups.

MRI and EEG abnormalities in different sub-groups (n=50)

Table 4: MRI and EEG abnormalities in different sub-groups (n=50)

		Normal EEG(total 10 cases)		**EEG Abnormality(total 40 cases)**			**MRI Abnormality(total 38 cases)**			**MRI normal cases(12 cases)**	
		N	*%*	*N*	*No.*	*%*	*N*	*No.*	*%*	*N*	*%*
	Hypothyroidism	1	10%	5	5	12.5%	6	6	15.7%	0	0%
Risk factors	Oligohydramnios	0	0%	2	2	5.%	2	2	5.2%	0	0%
	Polyhydramnios	0	0%	1	1	2.5%	1	0	0.0%	1	8%
	No	9	90%	32	32	80.%	30	30	78.9%	11	91%
	Birth Asphyxia	1	10%	7	7	17.5%	8	8	21.0%	0	0%
	Neonatal Seizures	1	10%	10	10	25%	11	11	28.9%	0	0%
	Family History of Seizure	2	20%	10	10	25%	8	8	21.0%	5	41%
	Global Developmental Delay	2	20%	13	13	32.5%	13	13	34.2%	2	16%
	Microcephaly	2	20%	11	11	27.5%	12	12	31.5%	1	8%
	Abnormal CNS Examination	2	20%	9	9	22.5%	10	10	26.3%	1	8%
EEG	Normal			-	-	-	5	5	13.1%	6	50%
	Abnormal			-	-	-	33	33	86.8%	6	50%
EEG abnormality	Focal Epileptiform Activity	0	0%	40	40	100.0 %	33	33	86.8%	7	58%
	Generalised Epileptiform Activity	0	0%	1	1	2.5%	1	1	2.6%	0	0%
	Frontal Epileptiform Activity	0	0%	25	25	62.5%	23	23	60.5%	2	16%
	Occipital Epileptiform Activity	0	0%	24	24	60%	20	20	52.6%	4	33%
	Parietal Epileptiform Activity	0	0%	21	21	52.5%	21	21	55.2%	0	0%
	Temporal Epileptiform Activity	0	0%	22	22	55%	20	20	52.6%	2	16%
	Focal Slow Wave	0	0%	19	19	47.5%	13	13	34.2%	6	50%
	Focal Spike Wave	0	0%	33	33	82.5%	28	28	73.6%	5	41%
	Focal Spikes And Polyspikes	0	0%	16	16	40%	14	14	36.8%	2	16%
	Rhythmic Delta Activity	0	0%	6	6	15%	5	5	13.1%	1	8%
	Other Discharges	0	0%	3	3	7.5%	3	3	7.8%	0	0%
MRI Brain	Normal	5	50%	7	7	17.5%	-	-	-		
	Abnormal	5	50%	33	33	82.5%	-	-	-		
MRI abnormality	Focal Cortical Dysplasia	0	0%	5	5	12.5%	5	5	13.1%	0	0%
	Hippocampal Sclerosis	0	0%	3	3	7.5%	3	3	7.8%	0	0%
	Heterotropia	1	10%	4	4	10%	5	5	13.1%	0	0%
	Congenital Malformation	1	10%	6	6	15%	7	7	18.4%	0	0%
	Focal Gliosis	0	0%	11	11	27.5%	11	11	28.9%	0	0%
	Encephalomalacia/Leukomalacia	2	20%	12	12	30%	14	14	36.8%	0	0%
	Space Occupying Lesion	0	0%	1	1	2.5%	1	1	2.6%	0	0%

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	Basal Ganglia Involvement	0	0%	1	1	2.5%	1	1	2.6%	0	0%
	Calcification	1	10%	2	2	5%	3	3	7.8%	0	0%
Diagnosis	Frontal Lobe Epilepsy	0	0%	3	3	7.5%	1	1	2.6%	2	16%
	Idiopathic Focal Epilepsy	5	50%	1	1	2.5%	0	0	0.0%	6	50%
	Occipital Lobe Epilepsy (Early Childhood)	0	0%	1	1	2.5%	0	0	0.0%	1	8%
	Occipital Lobe Epilepsy (Late Childhood)	0	0%	1	2	2.5%	0	0	0.0%	2	16%
	Symptomatic Focal Epilepsy	5	50%	30	30	75%	35	35	92.1%	0	0%
	Temporal Lobe Epilepsy	0	0%	3	3	7.5%	2	2	5.2%	1	8%

Conclusion

We observed multiple abnormalities on neuroimaging in pediatric epileptic patients, and the information given might help in directing medical or surgical management. Despite the fact that EEG and MRI are the best diagnostic equipment for evaluating patients presenting with seizures, our results did not demonstrate a correlation between EEG and neuroimaging findings. Rather, our findings showed that EEG information should not be the only condition for subsequent MRI evaluation. A large sample-sized population or prospective trial study should be used to confirm our study results.

MRI has been shown to play an important role in the management of pediatric seizure patients along with other modalities that include the use of EEG. It should be able to aid caregivers in reaching an accurate diagnosis, which is essential to decide the necessary care for their patients. MRI is considered the main imaging modality to establish a correct diagnosis and hence help in achieving a good prognosis.

A normal EEG does not rule out the presence of any pathology, including tumor formation. Thus, EEG findings are not a good predictor of MRI results. The presence of abnormalities in MRI was demonstrated to be significantly higher in cases with abnormal EEG. Therefore, MRI examinations should be performed as soon as possible in cases with multifocal IED. This will be important for clinical diagnosis, treatment, and follow-up of epileptic children with multifocal IED.

LIMITATIONS AND FUTURE DIRECTIONS

A. LIMITATIONS:-

- Our study has small sample size, which cannot be extrapolated to population.
- As our study is retrospective observational study, we nor able to follow up these patients afterward to know further clinical progression.
- Many of the patients done MRI with less than 3 tesla machines. Some clinical entities like cortical dysplasia needs 3 tesla MRI, this makes many MRI reports normal in cases with focal epilepsy.

B. FUTURE DIRECTIONS:-

- Large multicentre trial needed to use data for population.
- Many other factor can be included in study such as no.
- 3 Tesla MRI should use.

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