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Research Article

Profiles of Patients based on different Criterias in Relation with Magnetic Resonance Imaging findings and their association with Electroencephalograph Data in children with Focal Epilepsy

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Abstract

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.

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.

By this article, profile of patients based on diagnosis is as follows: majority of them diagnosed with symptomatic focal epilepsy (70%), minimum number of them diagnosed with early childhood occipital lobe epilepsy (2%), some of them diagnosed with late childhood occipital lobe epilepsy (4%), some of them diagnosed with frontal lobe epilepsy (6%), some of them diagnosed with idiopathic focal epilepsy (12%), some of them diagnosed with temporal lobe epilepsy (6%). Profiles of patients based on different criterias e.g. demography, age group, gender of patients, birth of asphyxia, family history, microcephaly have also been examined.

Keywords: *Epilepsy, Pediatric Epilepcy, EEG characteristics, PET scanning, Focal Epilepsy, Magnetic Resonance Imaging, Patient Profile, Electroencephalograph Data.*

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.

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 influx (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 non-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 influence 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.

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

DIAGNOSTIC WORKUP OF FOCAL EPILEPSIES, NEUROIMAGING

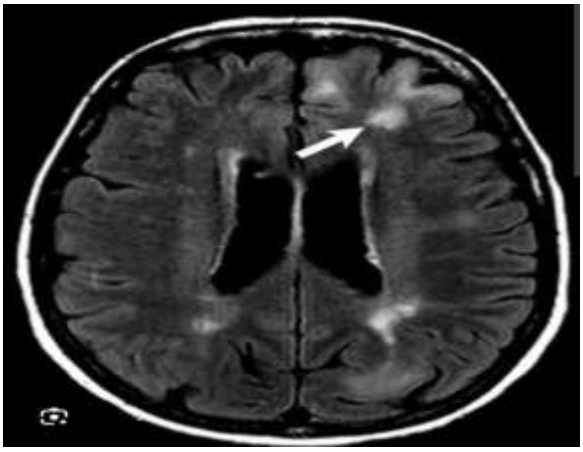
Significance and indications

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.

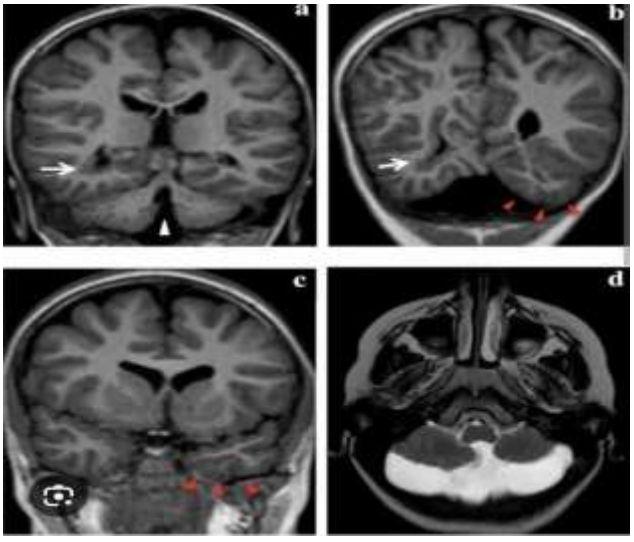
Overall, however, imaging reveals an epileptogenic lesion in only around 20% of patients with new-onset seizures. In patients with focal onset seizures, this percentage reaches ~50% but depends on the level of technological sophistication and neuroimaging technique. In general, patients with epilepsy 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.

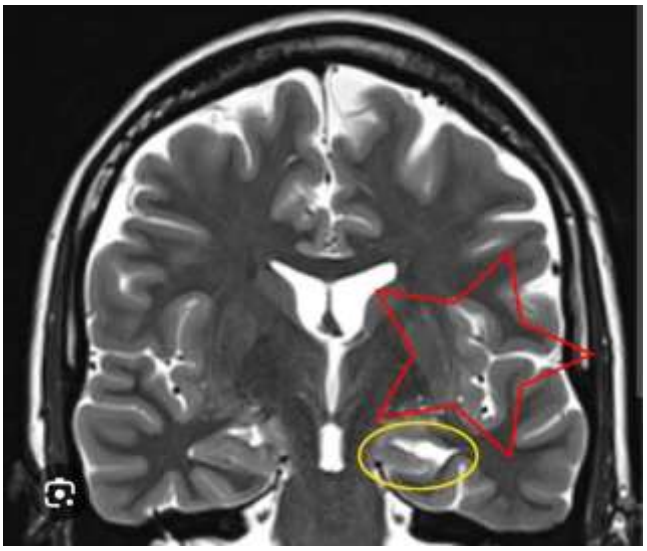
Structural imaging: MRI And CT

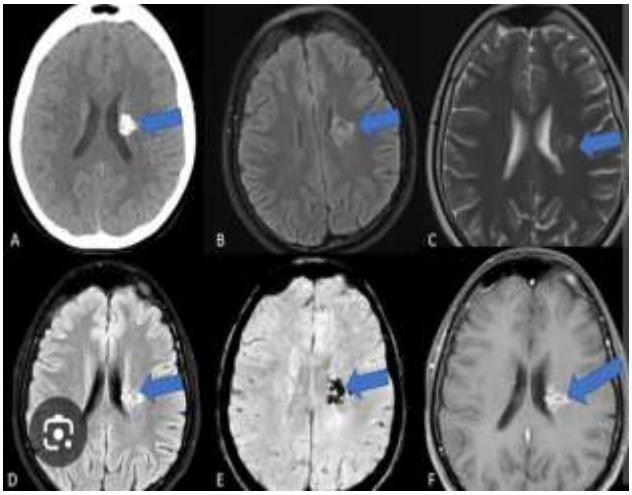
CT is recommended in emergency patients with a first seizure and is widely available worldwide. It is sensitive to bleedings, but also small calcified and bone lesions as well as skull defects (e.g., in patients with encephalocele). However, due to better differentiation of brain tissues, all patients 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.



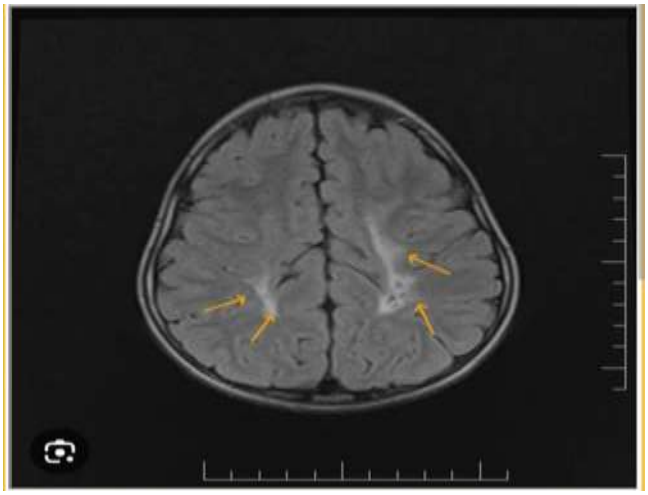
TUBEROUS SCLEROSIS



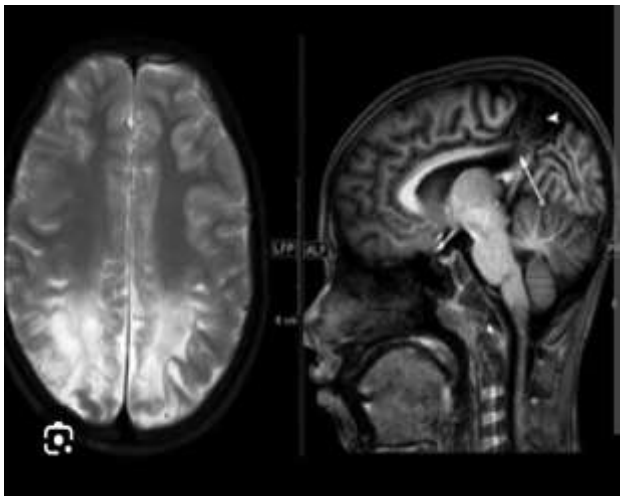




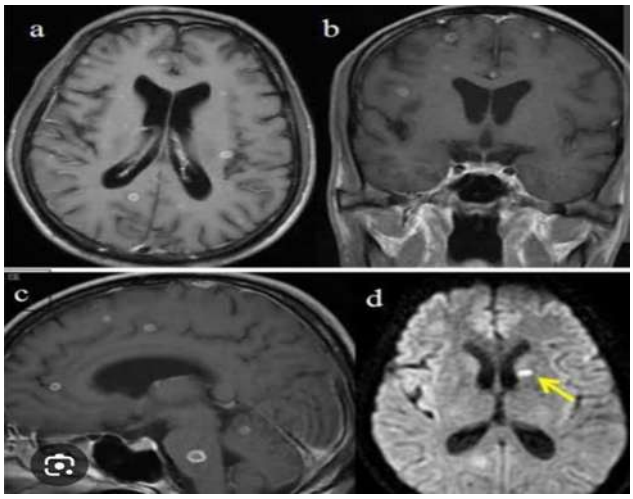
CALCIFICATION



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 (The below table). 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.

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			

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

DIAGNOSTIC WORKUP OF FOCAL EPILEPSIES, NEUROPHYSIOLOGY

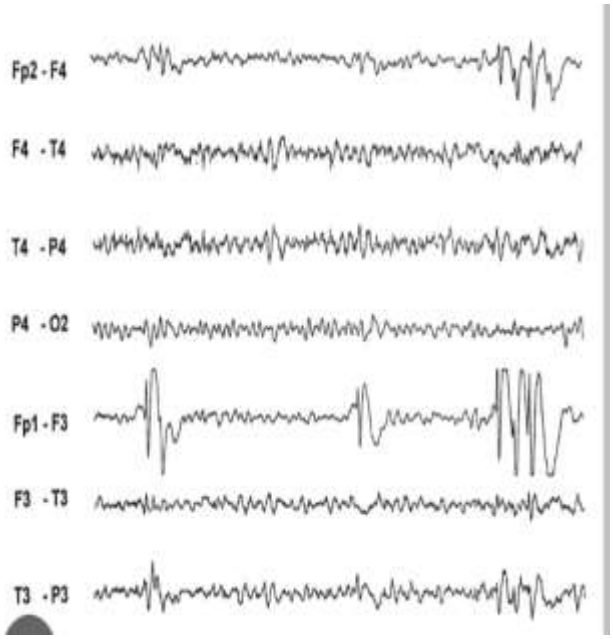
Confirmation of focal epilepsy

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.

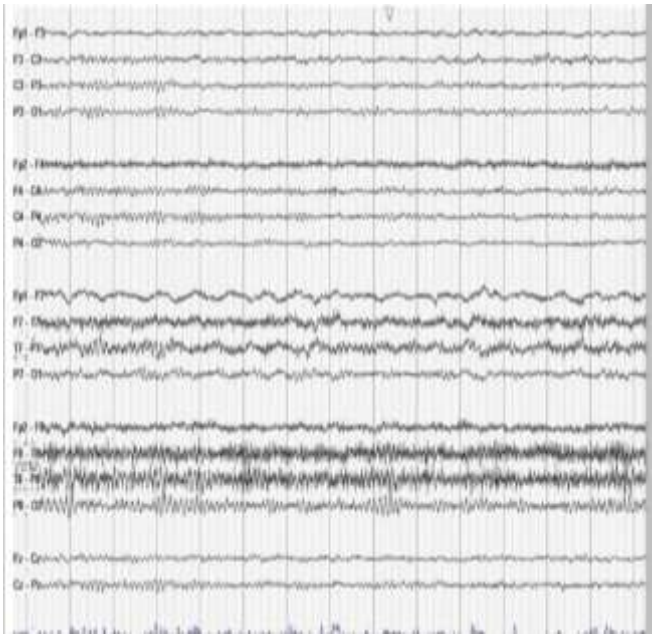
First, EEG is a low-amplitude, continuous and dynamic bio signal that requires considerable expertise for interpretation. The EEG signal recorded at the scalp reflects the summed potentials generated in a relatively large area (≥6–10 cm2) of the cortex at the cerebral convexity and, therefore, represents 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

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

Profile of patients (n=50). Table 1 shows the demography of patients with epilepsy.

Table 1: Demography of patients with epilepsy (n=50)

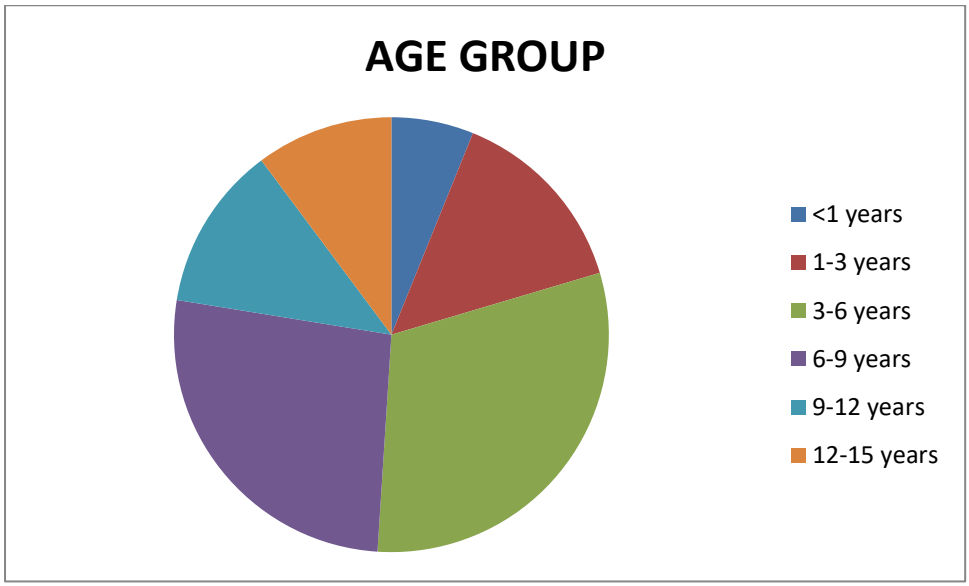
		No.	%
Age Group	Upto 1 yr.	3	6.0%
	>1 to 3 yrs.	7	14.0%
	>3 to 6 yrs.	15	30.0%
	>6 to 9 yrs.	13	26.0%
	>9 to 12 yrs.	6	12.0%

	>12 to 15 yrs.	5	10.0%
Sex	Male	38	76.0%
	Female	12	24.0%
	Total	50	100.0

PROFILE OF PATIENTS BASED ON AGE:

Out of the total patients, majority of them have ages 3-6 years(30%), least number of them had ages <1 year(6%), 9-12years(12%), 12-15 years(10%), 6-9 yrs(26%), 1-3 yrs(14%). Figure 1 shows the profile of patients based on age.

Figure 1: Age group of patients (%, n=50)



Based on Sex:

Majority of them were male (76%), remaining of them were female (24%). Figure 2 shows the profile of patients based on sex.

Figure 2: Gender of patients (%, n=50)

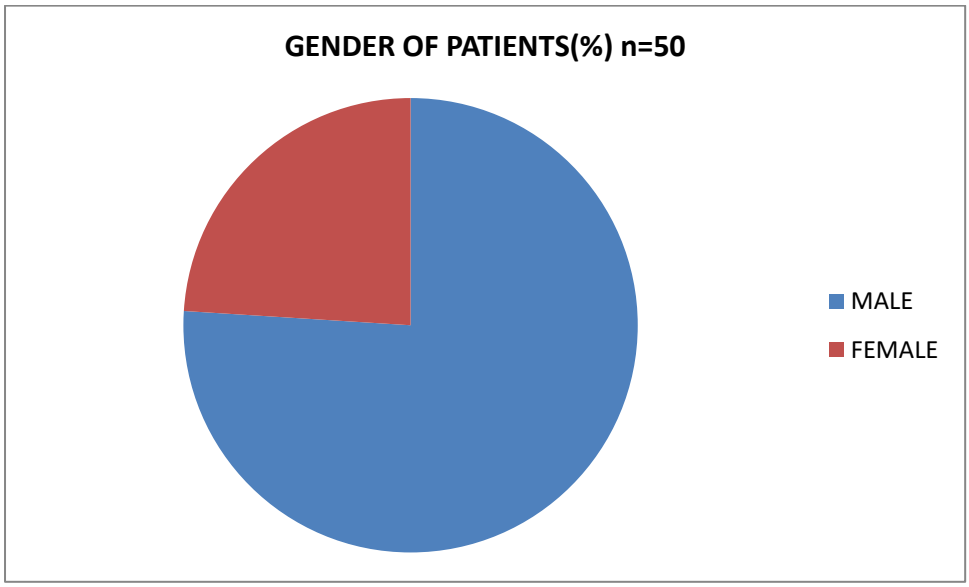
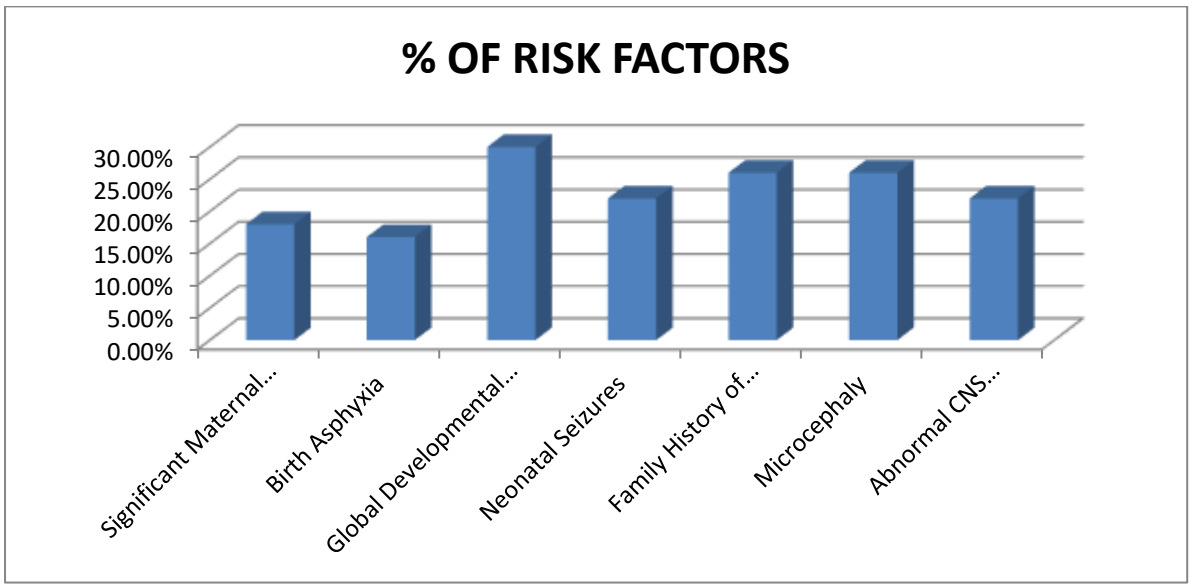


Table 2 shows the profile of patients based on risk factors.

Table 2: Profile of patients based on risk factors. (n=50)

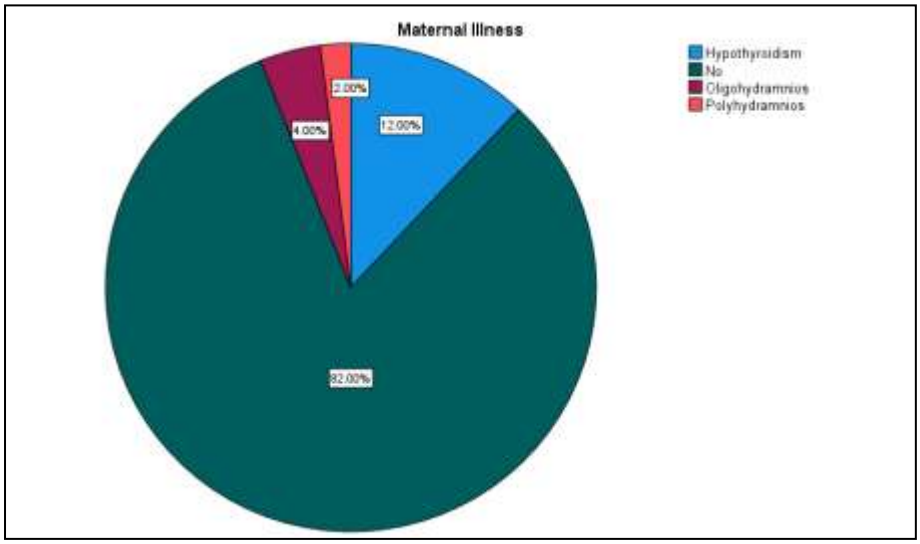
		No.	%
Maternal Illness	Hypothyroidism	6	12.0%
	Oligohydramnios	2	4.0%
	Polyhydramnios	1	2.0%
	No	41	82.0%
Birth Asphyxia	No	42	84.0%
	Yes	8	16.0%
Global Developmental Delay	Absent	35	70.0%
	Present	15	30.0%
Neonatal Seizures	No	39	78.0%
	Yes	11	22.0%
Family History of Seizures	No	37	74.0%
	Yes	13	26.0%
Microcephaly	Absent	37	74.0%
	Present	13	26.0%
CNS Examination	Normal	39	78.0%
	Abnormal	11	22.0%
	Total	50	100.0



PROFILE BASED ON MATERNAL ILLNESS:

Majority of them had antenatal history of hypothyroidism (12%), some of them had history of oligohydramnios (4%), some had history of polyhydramnios (2%). Figure 3 shows the profile of patients based on maternal illness.

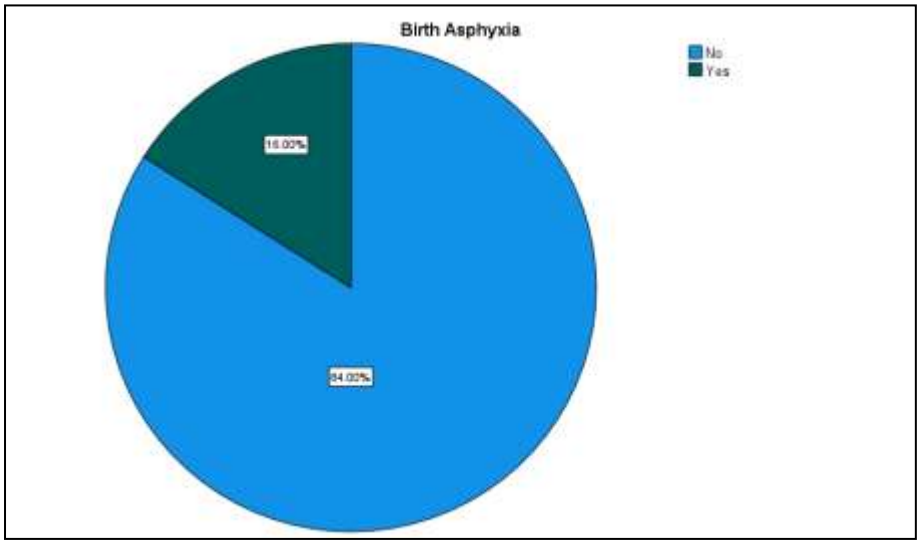
Figure 3: Maternal illness of patients (%, n=50)



PROFILE OF PATIENTS BASED ON BIRTH ASPHYXIA.

least of them had history of birth asphyxia, majority of them did not have (84%) Figure 4 shows the profile of patients based on Birth asphyxia.

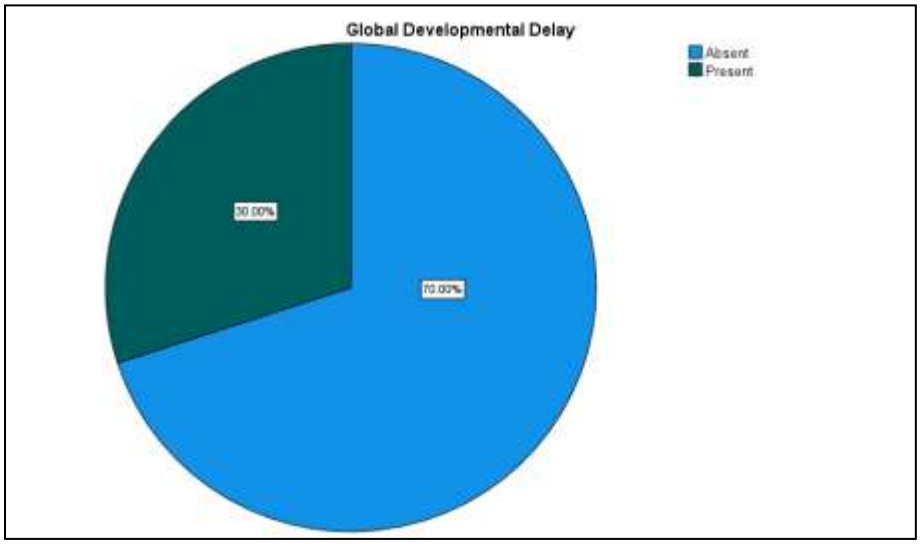
Figure 4: Birth asphyxia of patients (%, n=50)



PROFILE OF PATIENTS BASED ON GLOBAL DEVELOPMENTAL DELAY.

least of them had history of global developmental delay (30%), majority of them did not have (70%) Figure 5 shows the profile of patients based on Global developmental delay

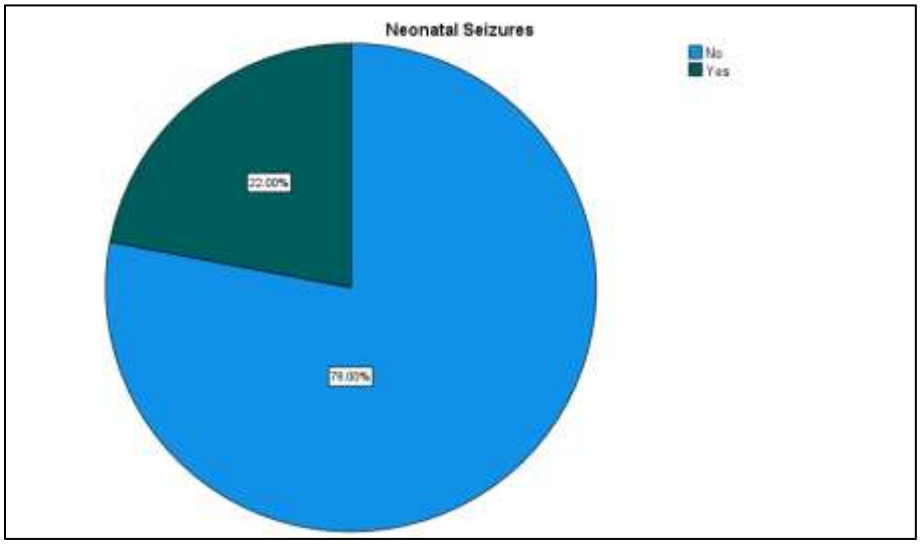
Figure 5: Global developmental delay in patients (%, n=50)



PROFILE OF PATIENTS BASED ON HISTORY OF NEONATAL SEIZURE:

Least number of them had history of neonatal seizures (22%), most of them did not have (78%). Figure 6 shows the profile of patients based on Neonatal seizures.

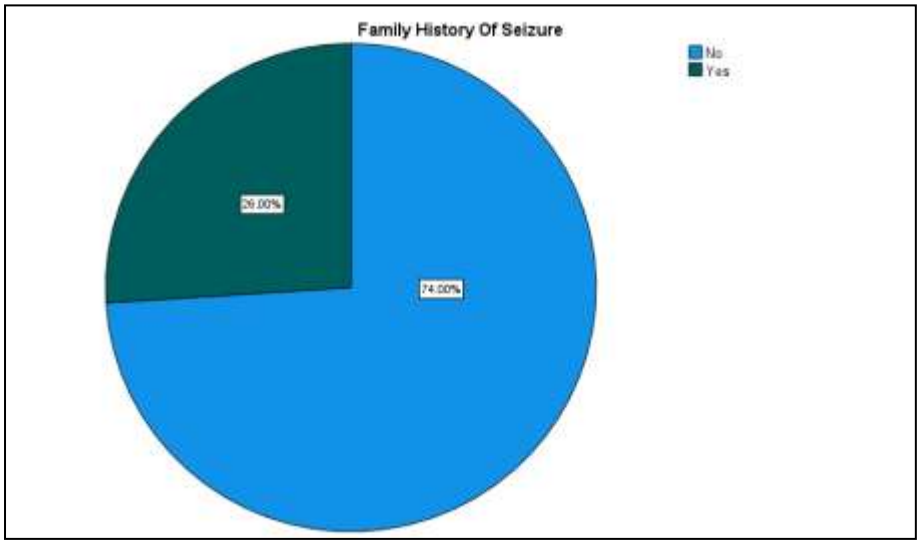
Figure 6: Neonatal seizures in patients (%, n=50)



PROFILE OF PATIENTS BASED ON FAMILY HISTORY OF SEIZURE:

Least number of them had family history of seizure(26), majority of them did not have(74%). Figure 7 shows the profile of patients based on Family history of seizures.

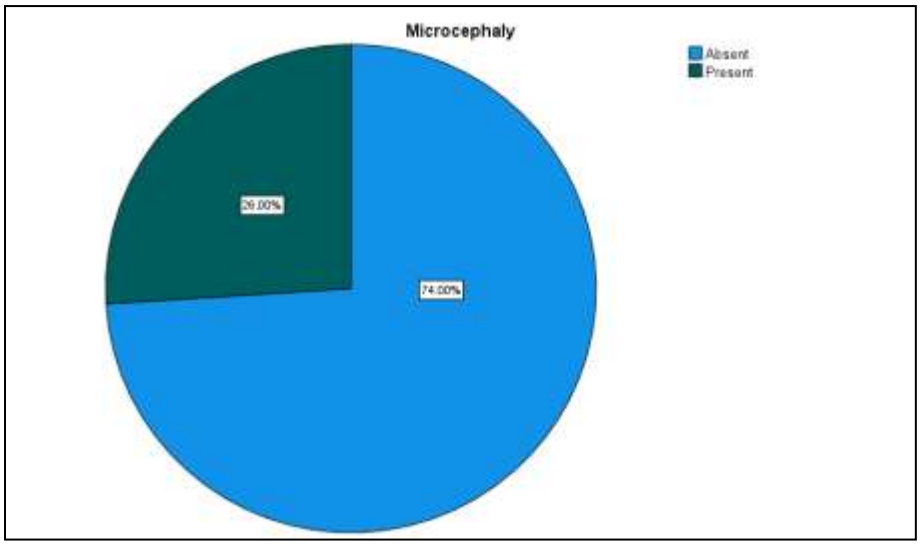
Figure 7: Family history of seizures in patients (%, n=50)



PROFILE OF PATIENTS BASED ON PRESENCE OF MICROCEPHALY:

Least number of them had microcephaly(26%), most of them did not have(74%). Figure 8 shows the profile of patients based on Family history of Microcephaly.

Figure 8: Microcephaly in patients (%, n=50)



PROFILE OF PATIENTS BASED ON NEUROLOGICAL EXAMINATION:

Minimum of them had abnormal neurological examination findings(22%), most of them had normal neurological finding(78%). Figure 9 shows the profile of patients based on neurological examination.

Figure 9: CNS examination findings in patients (%, n=50)

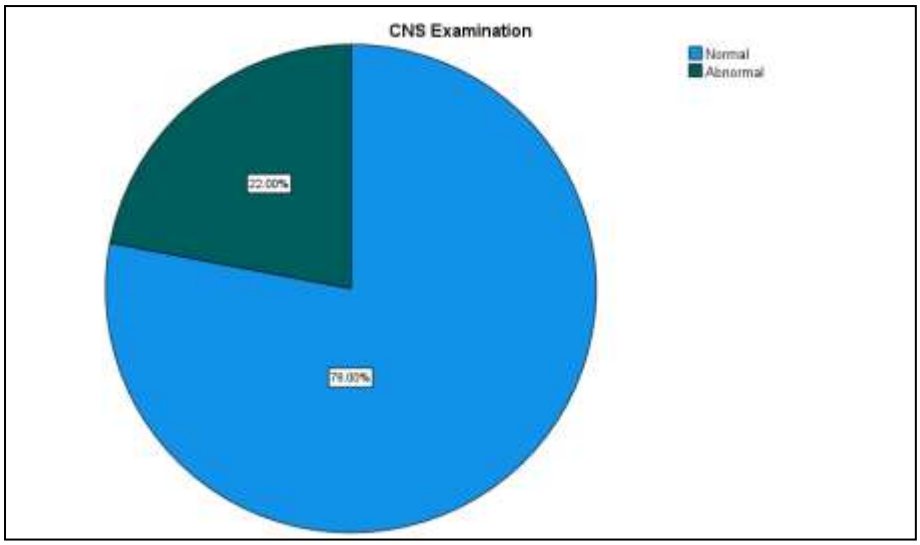


Table 3 shows the EEG findings of patients with epilepsy.

Table 3: EEG findings of patients with epilepsy (n=50)

		No.	%
EEG	Normal	10	20.0%
	Abnormal	40	80.0%
Total		50	100.0

PROFILE OF PATIENTS BASED ON EEG CHANGES:

Majority of them had abnormal EEG changes (80%), minority of them had normal EEG findings (20%). Figure 10 shows the profile of patients based on EEG findings.

Figure 10: EEG findings in patients (%, n=50)

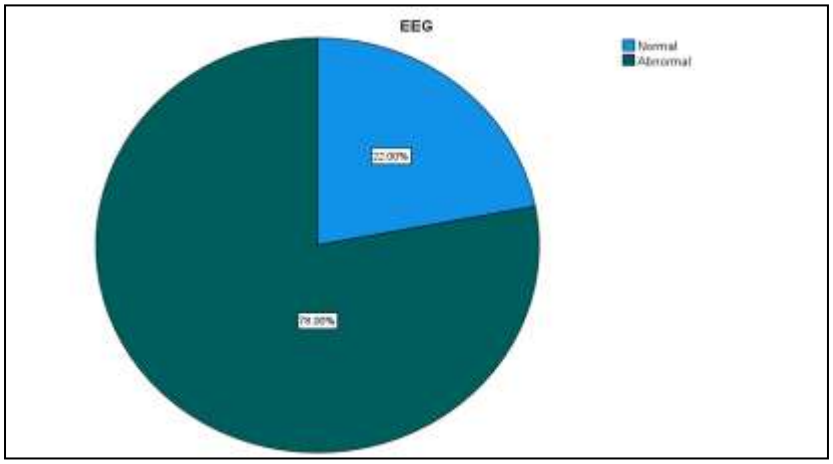


Table 4 shows MRI findings of patients with epilepsy.

Table 4: MRI findings of patients with epilepsy (n=50)

		No.	%
MRI	Normal	12	24.0%
	Abnormal	38	76.0%
Total		50	100.0

PROFILE OF PATIENTS BASED ON MRI BRAIN CHANGES:

Majority of them had abnormal MRI brain findings (76%), minority of them had normal MRI brain findings (24%).%). Figure 11 shows the profile of patients based on MRI Brain findings.

Figure 11: MRI findings in patients (%, n=50)

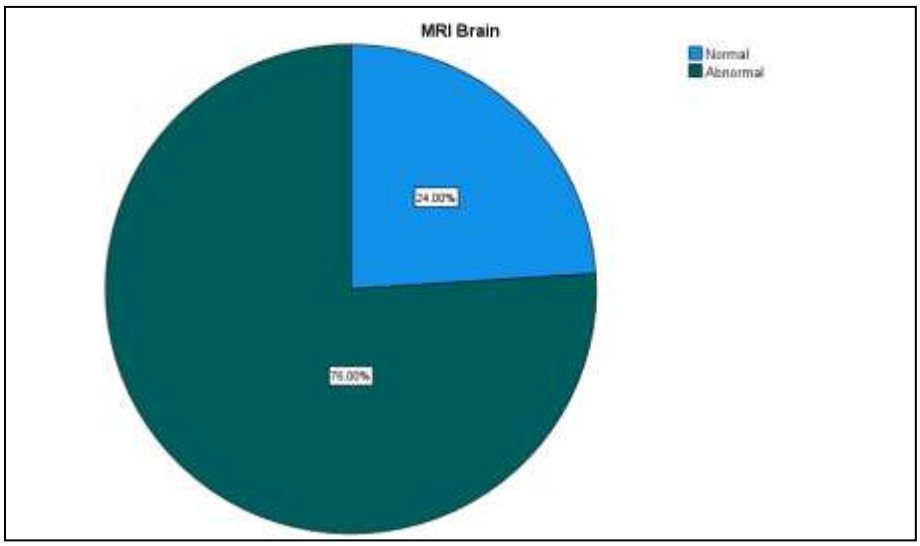


Table 5 shows diagnosis, of patients with epilepsy.

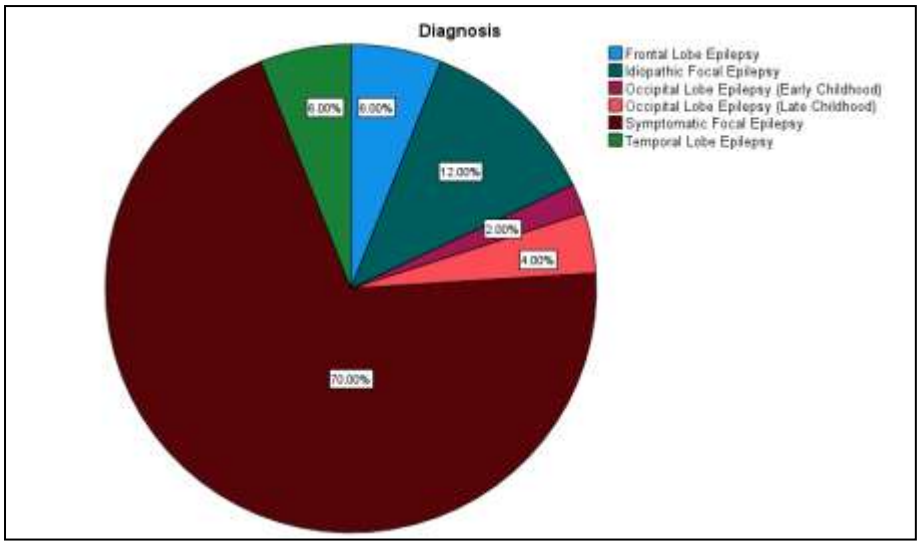
Table 5: Diagnosis, of patients with epilepsy (n=50)

		No.	*%*
Diagnosis	Frontal Lobe Epilepsy	3	6.0%
	Idiopathic Focal Epilepsy	6	12.0%
	Occipital Lobe Epilepsy (Early Childhood)	1	2.0%
	Occipital Lobe Epilepsy (Late Childhood)	2	4.0%
	Symptomatic Focal Epilepsy	35	70.0%
	Temporal Lobe Epilepsy	3	6.0%
Total		50	100.0

PROFILE OF PATIENTS BASED ON DIAGNOSIS:

Majority of them diagnosed with symptomatic focal epilepsy(70%), minimum number of them diagnosed with early childhood occipital lobe epilepsy(2%), some of them diagnosed with late childhood occipital lobe epilepsy (4%), some of them diagnosed with frontal lobe epilepsy(6%), some of them diagnosed with idiopathic focal epilepsy(12%), some of them diagnosed with temporal lobe epilepsy(6%). Figure 12 shows the profile of patients based on MRI Brain findings.

Figure 12: Clinical diagnosis of patients (%, n=50)



Conclusion

Epilepsy is a disease of the cerebral cortex and can cause severe long-term neurological illnesses with notorious physical and social limitations. It has been shown that MRI is effective for examining brain structure and imaging potential pathologic conditions that could be related to the etiology of seizures in pediatric epilepsy.11 Although MRI is a noninvasive form of imaging and very sensitive, it is not readily available or expensive.12 Because of its greater resolution and lack of radiation exposure to patients, MRI has been favored over computed tomography.1 We analyzed 50 pediatric patients with focal epileptic seizures aged between 0 −12 years. In our study, we highlighted MRI and EEG findings in patients with seizures. MRI was chosen due to its critical and essential role in the medical management of patients with seizures. MRI holds significant etiological diagnostic abilities. The gender characteristics of the present study were similar to published literature that revealed male predominance.

Out of the total patients, majority of them have ages 3-6 years(30%), Majority of them were male(76%). Majority of them were on 1 anti-epileptic drug(54%), Majority of them had abnormal EEG changes(78%), Majority of them had abnormal MRI brain findings(76%), Majority of them had abnormal EEG with abnormal MRI changes(66%).Majority of them had focal epileptiform activity(80%), In overall cases majority of them had frontal epileptiform activity(50%), Among them, most of them had encephalomalacia/leukomalacia(28%). Majority of them were on Leveriteracetam /brivacetam(56%).

Majority of them with with abnormal EEG changes were on 1 anti-epileptic drug(57.5%),. Majority of them with abnormal EEG changes were on leveriteracetam/brivacetam(50%),. Majority of them with abnormal MRI brain changes were on leveriteracetam/brivacetam(52.6%), All of them with abnormal EEG changes, had focal epileptiform activity(100%), majority of them had frontal epileptiform activity(62.5%), majority of them had focal spike wave(82.5%), Majority of them with abnormal MRI brain changes, had focal epileptiform activity(86.8%), majority of them had frontal epileptiform activity(60.5%), majority had focal spike wave (73.6%), Majority of them with abnormal EEG findings had encephalomalacia/leukomalacia(30.%). Majority of them with abnormal MRI brain findings had encephalomalacia/leukomalacia (36.8%).

Majority of them with abnormal EEG changes were diagnosed with symptomatic focal epilepsy(75%). Majority of them with abnormal MRI brain changes were diagnosed with symptomatic focal epilepsy(92.1%).

From this study we found that childrens from 3-6 years were more affected with focal epilepsy, majority of them were males, some of them had significant maternal illness. some of them had history of birth asphyxia, some had family history of seizure, some had delayed developmental milestones. Majority of them were on 1 anti-epileptic drug, among them leviteracetam was most common. On examination, very few had microcephaly with abnormal neurological findings. On EEG abnormality majority of them showed focal epileptiform activity mostly in frontal lobe. On MRI changes, majority of them had changes of encephalomalacia/ leukomalacia.

Most of the patients with EEG changes of frontal lobe epileptiform activity had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia. Majority of them with frontal lobe epileptiform activity have changes of congenital malformation, focal cortical dysplasia, focal gliosis, encephalomalacia/leukomalacia, space occupying lesion, basal ganglia changes in frontal lobe. Also in certain cases with temporal lobe epileptiform activity, heterotropia, and

hippocampal sclerosis were seen in temporal lobe only. From this we would be able to evaluate most probable site of lesion in MRI brain with EEG changes. But in some of the cases there limited correlation of EEG with MRI changes, eg: there are cases with temporal lobe epileptiform activity having MRI changes of calcification in parietal lobe. Out of overall EEG abnormalities, 62.5% of them were localized to site of lesion in brain, remaining 37.5% of them were localised to some other site of lesion.

In contrast to a study examining 59 patients done by Mehmet alp dirik et al with intracranial space-occupying lesion, scalp EEG was unlocalized in 29% of the cases, localized to the tumor area in 44% of the cases, and localized elsewhere in 27% of the cases, which was also seen in our study In a study done by Najafi MA et al among 222 epileptic patients, majority of them 212 (95.5%) had documented epileptiform activity based on an EEG examination, and 10 (4.5%) had a normal initial EEG record, but in our study abnormal EEG changes were seen in most of the patient(78%) and 20% of them had normal EEG changes. Hence, we would say that most of the patients with focal epilepsy can be diagnosed with EEG.

Even though our sample size was small, we assessed EEG and MRI findings in pediatric epilepsy. Our results demonstrated that there is no statistically significant relationship between EEG and MRI results. This implies that normal MRI scan results cannot be predicted from normal EEG readings. These findings highlight that an epileptic patient should not be placed in a low-risk category based on normal EEG readings, and these patients should have an MRI for additional assessment. Another study, however, concluded that CNS infection is the main etiology in pediatric patients with seizures. This difference is attributed to the fact that referral centers in India usually manifest a high number of cases of infection.

It is recommended to perform an MRI examination even if the EEG examination was normal. According to the literature, MRI is one of the integral modalities in evaluating seizure etiology.

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 factors can be included in study such as no.
- 3 Tesla MRI should use.

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