



Children with Focal Epilepsy - Profile of Patients based on risk factors, EEG and MRI changes

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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.

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.

In this article profiles of patients based on risk factors, EEG and MRI changes have been examined. Majority of them with abnormal EEG did not have any significant antenatal history (80. %), least number of them had history of polyhydramnios (2.5%), some of them had history of oligohydramnios (5.%), some had history of hypothyroidism (12.5%). Majority of them with normal MRI changes did not have any significant antenatal history(90%),none of them had history of polyhydramnios(0%), none of them had history of oligohydramnios (0.%), some had history of hypothyroidism(10%) Majority of them with abnormal MRI brain findings did not have any significant antenatal history(78.9%),some of them had hypothyroidism (15.7%) least number of them had history of oligohydramnios(5.2%), none of them had history of polyhydramnios. %) Majority of them with normal MRI brain findings did not have any significant antenatal history (91%), none of them had hypothyroidism (0%) none of them had history of oligohydramnios (0%), only 1had history of polyhydramnios. (8%).

Keywords: Birth asphyxia, Neonatal Seizure, Family History of Seizure, Global Developmental Delay, Microcephaly, Neurological Examination Findings, Epilepsy, EEG Characteristics, PET Scanning, Focal Epilepsy, Magnetic Resonance Imaging, EEG Changes, EEG findings, Diagnosis, MRI Changes, Profile of Patients based on Risk Factors, Pediatric Epilepsy.

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.

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 ($\geq 6-10$ 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.

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 dyselectrolytemia 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.

PROFILE OF PATIENTS BASED ON RISK FACTORS, EEG AND MRI CHANGES.

1)BASED ON RISK FACTORS:

MATERNAL ILLNESS:

Majority of them with abnormal EEG did not have any significant antenatal history (80.%), least number of them had history of polyhydramnios (2.5%), some of them had history of oligohydramnios (5.%), some had history of hypothyroidism (12.5%). Majority of them with normal MRI changes did not have any significant antenatal history(90%), none of them had history of polyhydramnios(0%), none of them had history of oligohydramnios(0.%), some had history of hypothyroidism(10%) Majority of them with abnormal MRI brain findings did not have any significant antenatal history(78.9%),some of them had hypothyroidism (15.7%) least number of them had history of oligohydramnios(5.2%), none of them had history of polyhydramnios. %) Majority of them with normal MRI brain findings did not have any significant antenatal history (91%), none of them had hypothyroidism (0%) none of them had history of oligohydramnios (0%), only 1had history of polyhydramnios. (8%)

BIRTH ASPHYXIA:

Minimum number of them with abnormal EEG changes had history of birth asphyxia(17.5%), majority of them did not have history of birth asphyxia(82.5%). Minimum number of them with normal EEG changes had history of birth asphyxia(10%), majority of them did not have history of birth asphyxia(90%). Minimum number of them with abnormal MRI brain changes had history of birth asphyxia(21%), majority of them did not have history of birth asphyxia(79%). None of them with normal MRI brain changes had history of birth asphyxia(0%).

NEONATAL SEIZURE:

Minimum number of them with abnormal EEG changes had history of neonatal seizure(25.%), majority of them did not have history of neonatal seizure(75%). Minimum number of them with normal EEG changes had history of neonatal seizure(10%), majority of them did not have history of neonatal seizure(90%). Minimum number of them with abnormal MRI brain changes had history of neonatal seizure(28.9%), majority of them did not have history of neonatal seizure(71.1%). None of them with normal MRI brain changes had history of neonatal seizure(0%).

FAMILY HISTORY OF SEIZURE:

Minimum number of them with abnormal EEG changes had family history of seizure(25.%), majority of them did not have family history of seizure(75%). Minimum number of them with normal EEG changes had family history of seizure(20.%), majority of them did not have family history of seizure(80%). Minimum number of them with abnormal MRI brain changes had family history of seizure(21.0%), majority of them did not have family history of seizure(79%). Majority of them with normal MRI brain changes had family history of seizure(40%) rest of them did not have.(0%).

GLOBAL DEVELOPMENTAL DELAY:

Majority of them with abnormal EEG changes did not have global developmental delay(67.5%), minority of them had history of global developmental delay(32.5%). Majority of them with normal EEG changes did not have global

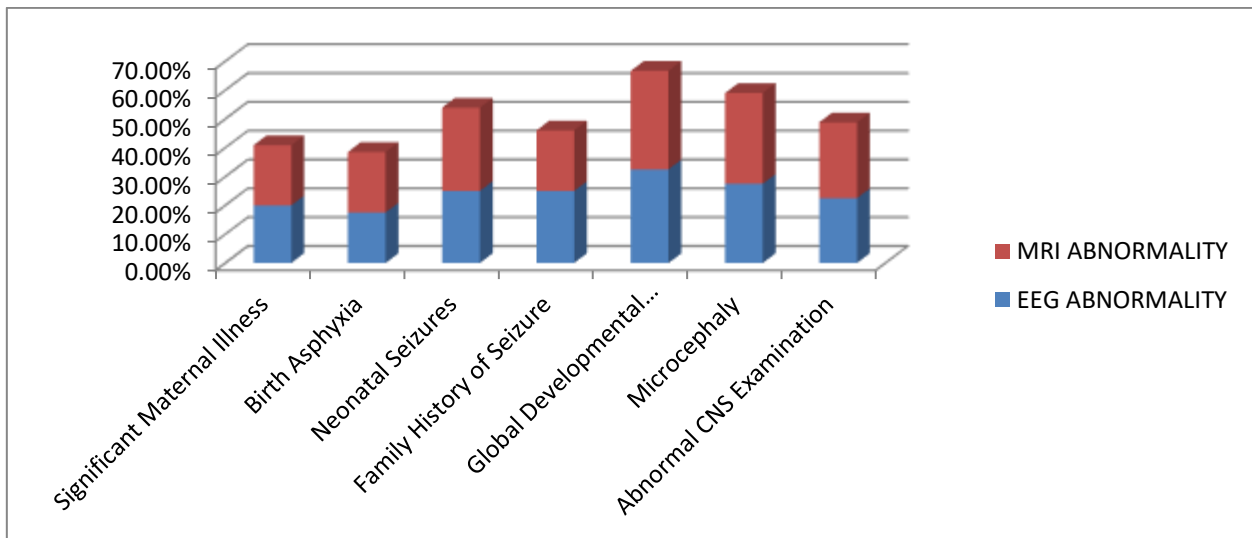
developmental delay(80%), minority of them had history of global developmental delay(20%). Majority of them with abnormal MRI brain changes did not have global developmental delay(65.8%), minority of them had history of global developmental delay(34.2%). Majority of them with normal MRI brain changes did not have global developmental delay(84%), minority of them had history of global developmental delay(16%).

MICROCEPHALY:

Majority of them with abnormal EEG changes did not have microcephaly(72.5%), minority of them had microcephaly(27.5%). Majority of them with normal EEG changes did not have microcephaly(80%), minority of them had microcephaly(20%). Majority of them with abnormal MRI brain changes did not have microcephaly(68.5%), minority of them had microcephaly(31.5%). Majority of them with normal MRI brain changes did not have microcephaly(92%), minority of them had microcephaly(8%).

NEUROLOGICAL EXAMINATION:

Minority of them with abnormal EEG changes have abnormal neurological examination findings(22.5%), majority of them with abnormal EEG changes did not have abnormal neurological examination findings(77.5%). Minority of them with normal EEG changes have abnormal neurological examination findings(20%), majority of them with normal EEG changes did not have abnormal neurological examination findings(80%). Minority of them with abnormal MRI brain changes have abnormal neurological examination findings(26.3%), majority of them with abnormal MRI changes did not have abnormal neurological examination findings(73.7%). Minimum number of them with normal MRI changes have abnormal neurological examination(8%), majority of them with normal MRI changes did not have abnormal neurological changes(92%).

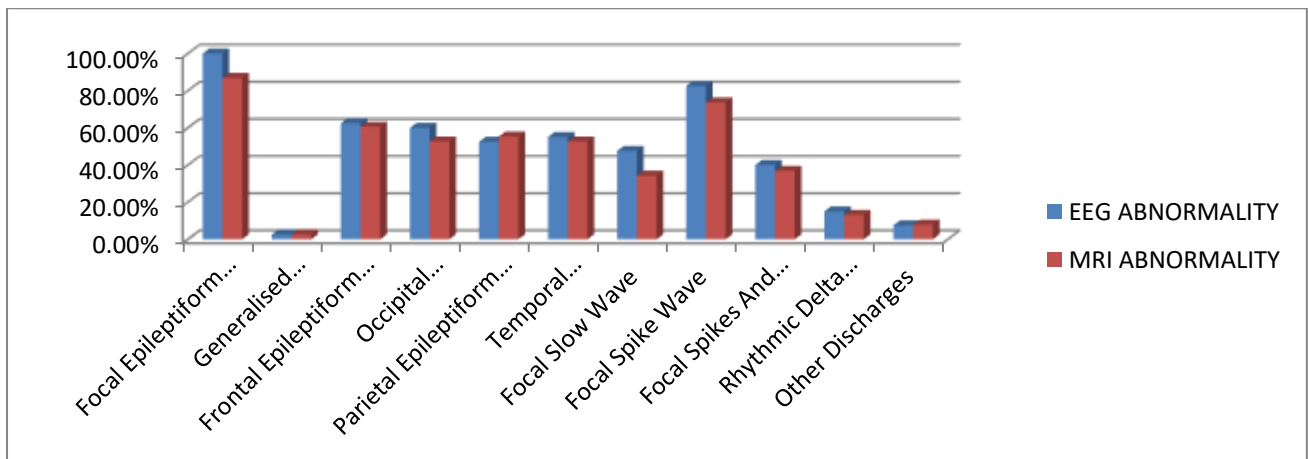
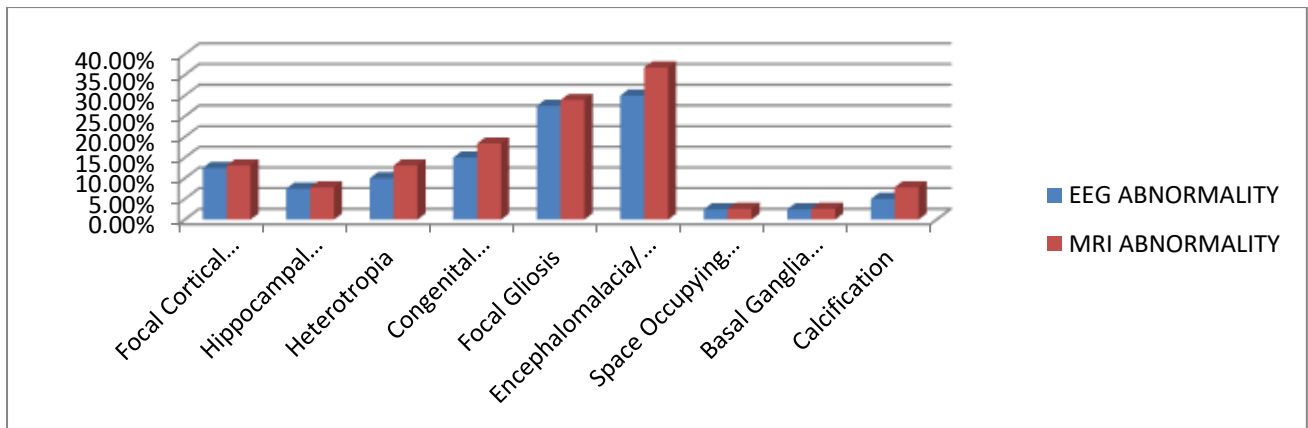


EEG CHANGES:

All of them with abnormal EEG changes, had focal epileptiform activity(100%), least number of them had generalized epileptiform activity(2.5%), some of them had frontal epileptiform activity(62.5%), some had occipital epileptiform activity(60%), some had parietal epileptiform activity(52.5%), some had temporal epileptiform activity(55%), some had focal slow wave(47.5%), some had focal spike wave(82.5%), some had focal spikes and polyspikes(40%), some had rhythmic delta activity(15 %). None of them with normal EEG changes, had focal epileptiform activity(0%), generalized epileptiform activity(0%), frontal epileptiform activity(0%), occipital epileptiform activity(0%), parietal epileptiform activity(0%), temporal epileptiform activity(0%), focal slow wave(0%), focal spike wave(0%), focal spikes and polyspikes(0%), rhythmic delta activity(0 %). Majority of them with abnormal MRI brain changes, had focal epileptiform activity(86.8%), least number of them had generalized epileptiform activity(2.6%), some of them had frontal epileptiform activity(60.5%), some had occipital epileptiform activity(52.6%), some had parietal epileptiform activity(55.2%), some had temporal epileptiform activity(52.6%), some had focal slow wave(34.2%), some had focal spike wave(73.6%), some had focal spikes and polyspikes(36.8%), some had rhythmic delta activity(13.1%), other discharges(7.8%). Majority of them with normal MRI brain changes, had focal epileptiform activity(58%), none of them had generalized epileptiform activity(0%),and parietal epileptiform activity(0%), some of them had frontal epileptiform activity(16%), some had occipital epileptiform activity(33%), , some had temporal epileptiform activity(16%), some had focal slow wave(50%), some had focal spike wave(41%), some had focal spikes and polyspikes(16%), some had rhythmic delta activity(8%),none had other discharges(0%).

MRI BRAIN CHANGES:

Majority of them with abnormal EEG findings had encephalomalacia/leukomalacia(30.0%), least number of them had space occupying lesion (2.5%) and basal ganglia involvement(2.5%), some of them had hippocampal sclerosis(7.5%), some had focal cortical dysplasia(12.5%), some of them had heterotropia(10%), some of them had congenital malformation(15%), some of them had focal gliosis(27.5%), some had calcification(5%). Majority of them with normal EEG findings had encephalomalacia/leukomalacia(20.0%), none of them had space occupying lesion (0%) and basal ganglia involvement(0%), none them had hippocampal sclerosis(0%), focal cortical dysplasia(0%), some of them had heterotropia(10%), some of them had congenital malformation(10%), none of them had focal gliosis(0%), some had calcification(10%). Majority of them with abnormal MRI brain findings had encephalomalacia/leukomalacia(36.8%), least number of them had space occupying lesion (2.6%) and basal ganglia involvement(2.6%), some of them had hippocampal sclerosis(7.8%), some had focal cortical dysplasia(13.1%), some of them had heterotropia(13.1%), some of them had congenital malformation(18.4%), some of them had focal gliosis(28.9%), some had calcification(7.8%). None them with normal MRI brain findings had encephalomalacia/leukomalacia(0%), space occupying lesion (0%), basal ganglia involvement(0%), hippocampal sclerosis(0%), focal cortical dysplasia(0%), heterotropia(0%), congenital malformation(0%), focal gliosis(0%), calcification(0%).



DIAGNOSIS:

Majority of them with abnormal EEG changes were diagnosed with symptomatic focal epilepsy(75%). minimum number of them diagnosed with early childhood occipital lobe epilepsy(2.5%), late childhood occipital lobe epilepsy (2.5%) and idiopathic focal epilepsy(2.5%), , some of them diagnosed with frontal lobe epilepsy(7.5%) some of them diagnosed with temporal lobe epilepsy(7.5%). Majority of them with normal EEG changes were diagnosed with symptomatic focal epilepsy(50%) and idiopathic focal epilepsy(50%), none of them diagnosed with frontal lobe epilepsy(0%) and temporal lobe epilepsy(0%). Majority of them with abnormal MRI brain changes were diagnosed with symptomatic focal epilepsy(92.1%). minimum number of them diagnosed with frontal lobe epilepsy(2.6%), some of them diagnosed with temporal lobe epilepsy(5.2%), none of them diagnosed with occipital lobe epilepsy(early and late) and idiopathic focal epilepsy. Majority of them with normal MRI brain changes were diagnosed with idiopathic focal epilepsy(50%). minimum number of them diagnosed with temporal lobe epilepsy(8%) and early childhood with occipital lobe epilepsy(8%) some of them diagnosed with frontal lobe epilepsy(16%) and late childhood with occipital lobe epilepsy 916%), none of them diagnosed with symptomatic focal epilepsy(0%).

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

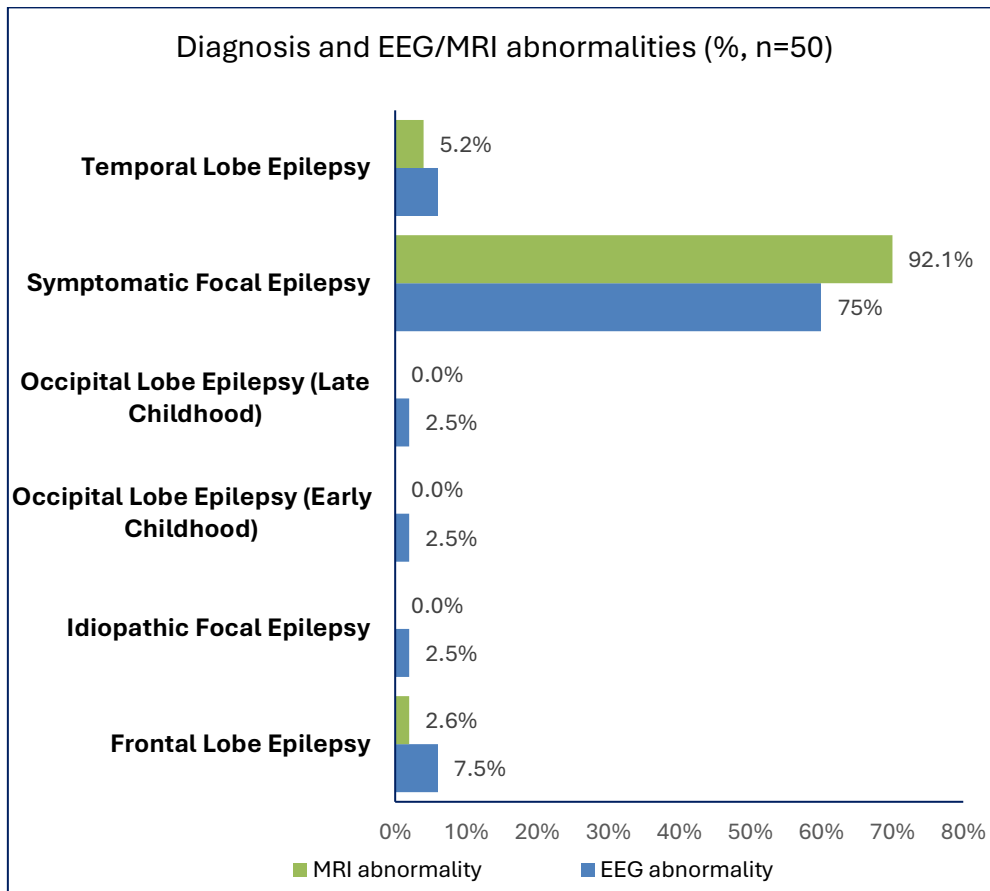
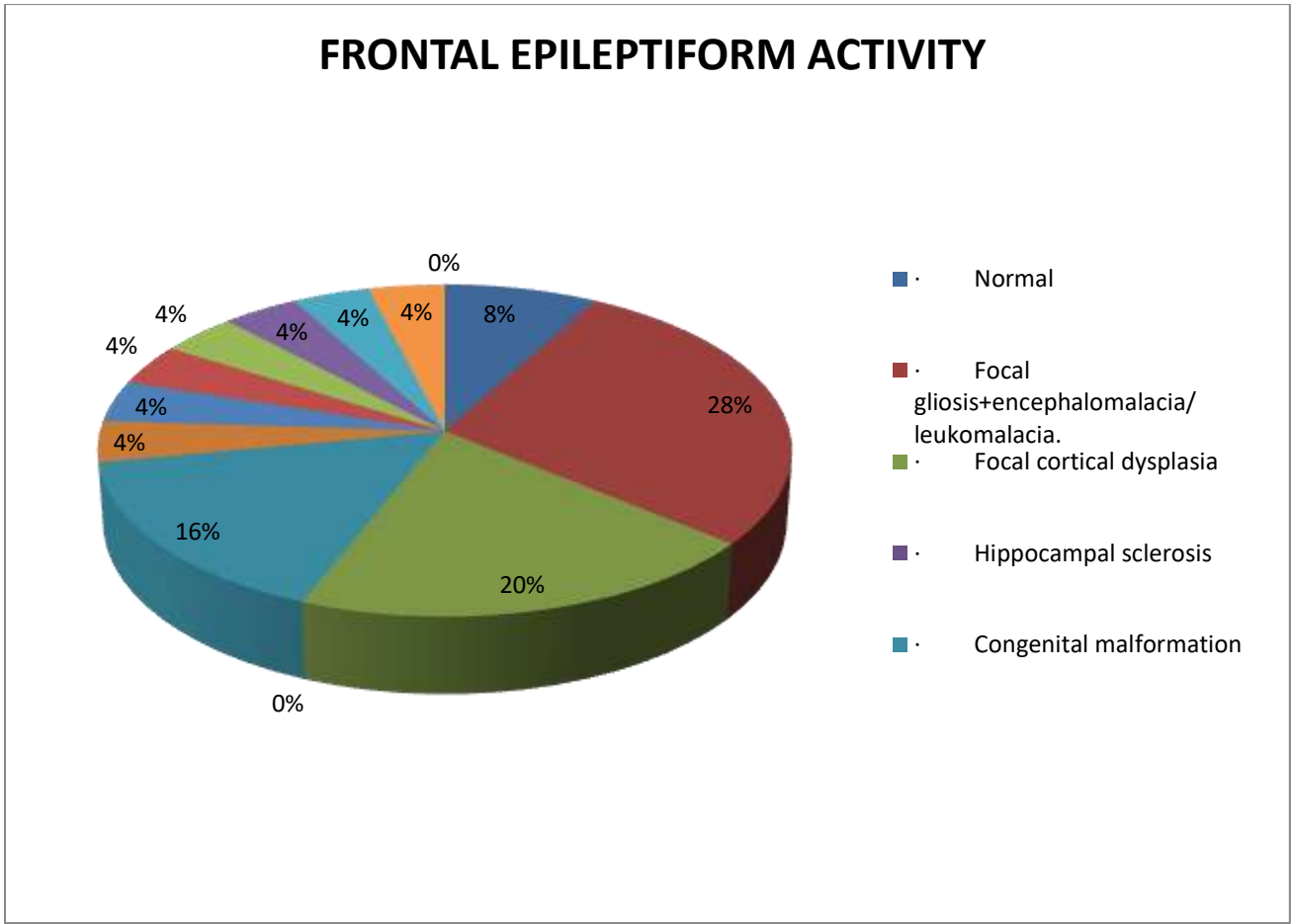


Table 1: Frontal lobe epilepsy with MRI changes. (n=50)

FRONTAL LOBE EPILEPTIFORM ACTIVITY		
	No.	%
MRI CHANGES		
Normal	2	8%
Focal gliosis+encephalomalacia/ leukomalacia.	7	28%
Focal cortical dysplasia	5	20%
Hippocampal sclerosis	0	0%
Congenital malformation	4	16%
Focal gliosis	1	4%
Calcification	1	4%
Heterotropia	1	4%
Basal ganglia change with calcification.	1	4%
Encephalomalacia /leukomalacia	1	4%
Focal cortical dysplasia+ heterotropia	1	4%
Space occupying lesion	1	4%
Focal gliosis + encephalomalacia+heterotropia	0	0%
	T=25	100%

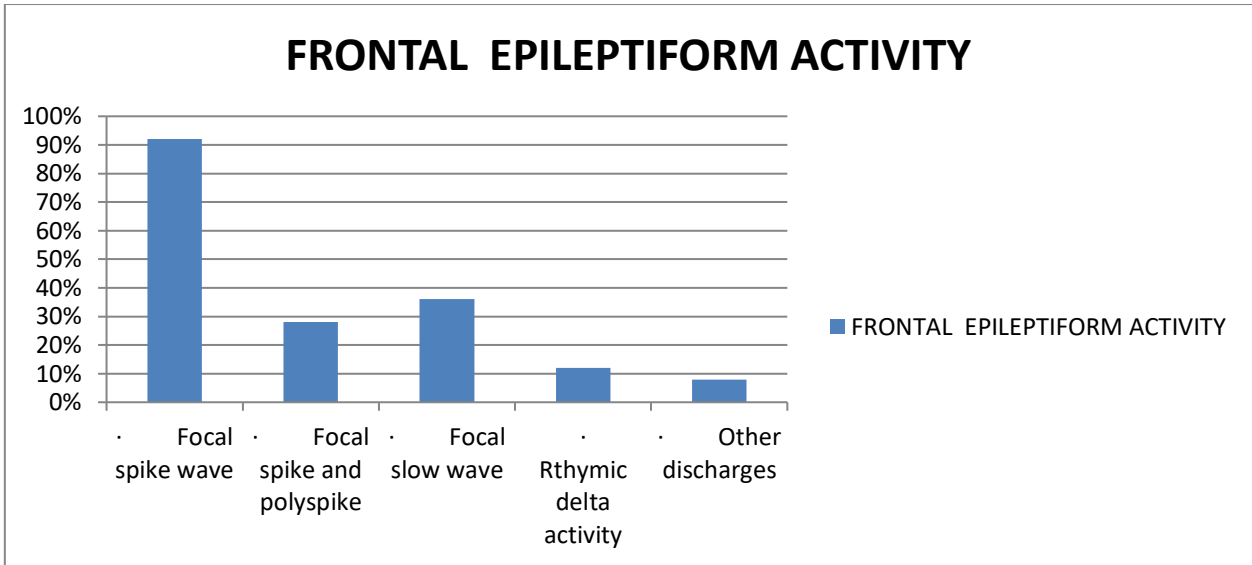


EXPLANATION:

- Most of the patients with EEG changes of frontal lobe epileptiform activity had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia. Some of them had changes with Focal cortical dysplasia(20%), Congenital malformation(16%), Focal gliosis(4%), Calcification(4%), Heterotropia(4%), Basal ganglia change with Calcification (4%), Encephalomalacia /leukomalacia(4%) , Focal cortical dysplasia+ heterotropia(4%), Space occupying lesion(4%). None of them had changes with hippocampal sclerosis(0%) and Focal gliosis + encephalomalacia+ heterotropia(0%). 8% of them had normal MRI findings.

FRONTAL LOBE EPILEPTIFORM ACTIVITY (n=25)		
	No.	%
EEG WAVEFORMS		
Focal spike wave	23	92%
Focal spike and polyspike	7	28%
Focal slow wave	9	36%
Rhythmic delta activity	3	12%
Other discharges	2	8%

- EXPLANATION:** Majority of them with frontal epileptiform activity have Focal spike wave(92%). Least number of them have other discharges(8%). Some of them focal spike and polyspike(28%), some have focal slow wave(36%), some have rhythmic delta activity(12%).

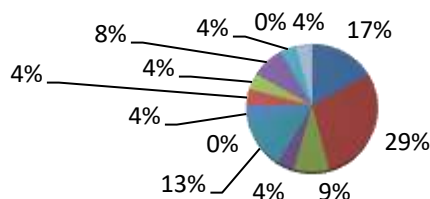


OCCIPITAL LOBE EPILEPTIFORM ACTIVITY

MRI CHANGES	No.	%
Normal	4	16.6%
Focal gliosis+encephalomalacia/ leukomalacia.	7	29.1%
Focal cortical dysplasia	2	8.3%
Hippocampal sclerosis	1	4.1%
Congenital malformation	3	12.5%
Focal gliosis	0	0%
Calcification	1	4.1%
heterotropia	1	4.1%
Basal ganglia change with calcification.	1	4.1%
Encephalomalacia /leukomalacia	2	8.3%
Focal cortical dysplasia+ heterotropia	1	4.1%
Space occupying lesion	0	0%
Focal gliosis + encephalomalacia+heterotropia	1	4.1%
T=24		100%

% IN OCCIPITAL EPILEPTIFORM ACTIVITY

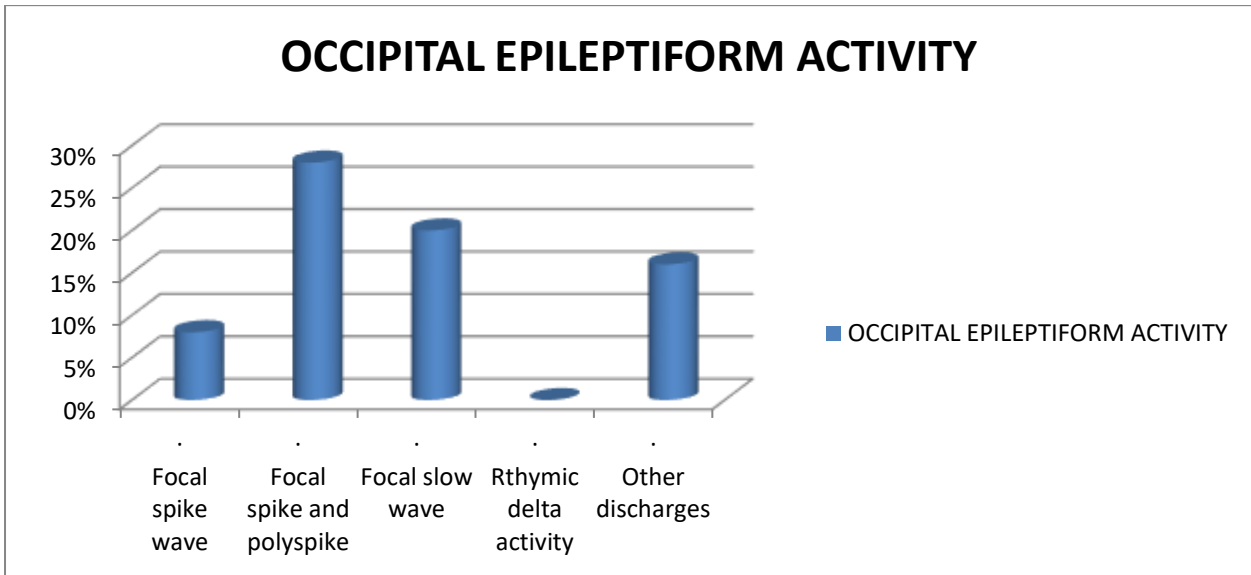
- Normal
- Focal gliosis+encephalomalacia/ leukomalacia.
- Focal cortical dysplasia
- Hippocampal sclerosis
- Congenital malformation
- Focal gliosis
- Calcification
- heterotropia
- Basal ganglia change with calcification.
- Encephalomalacia /leukomalacia
- Focal cortical dysplasia+ heterotropia
- Space occupying lesion



EXPLANATION:

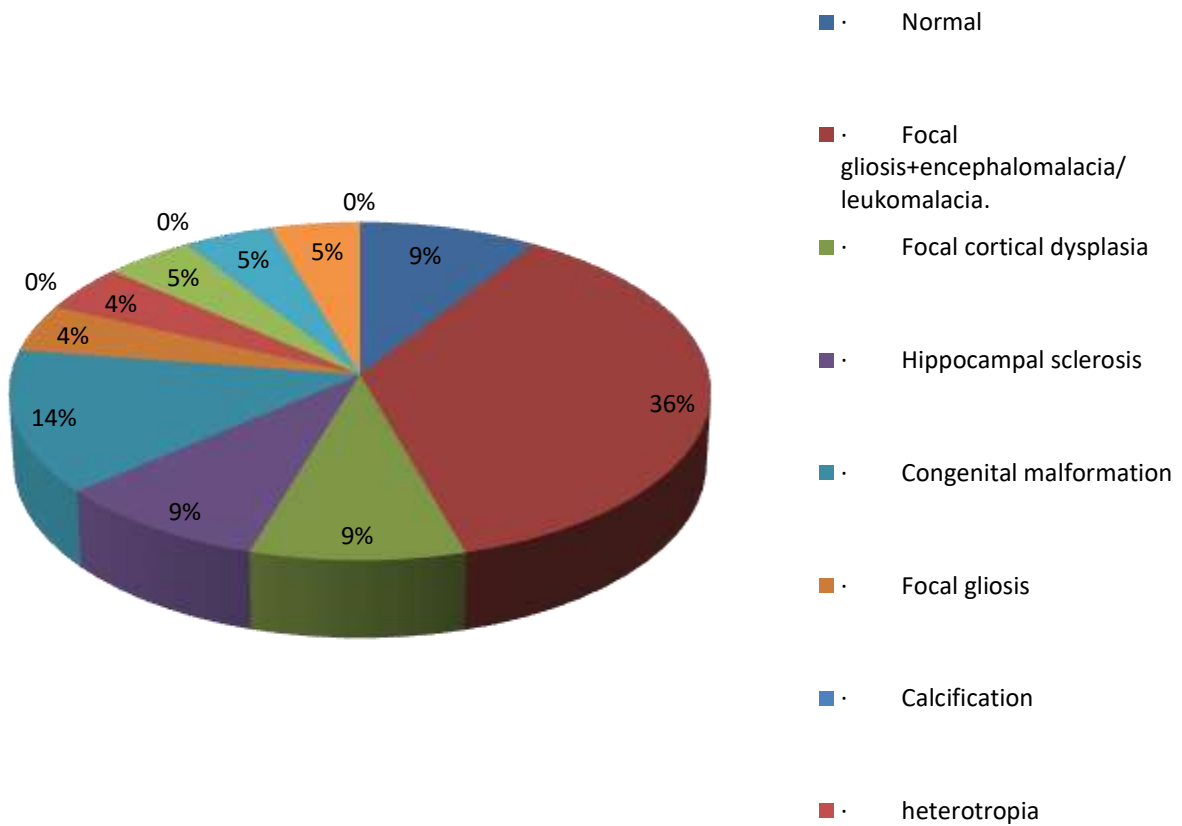
Most of the patients with EEG changes of occipital lobe epileptiform activity had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia (29.1%). Some of them had changes with Focal cortical dysplasia (8.3%), Congenital malformation (12.5%), hippocampal sclerosis (4.1%), Focal gliosis + encephalomalacia+ heterotropia (4.1%). Calcification (4.1%), Heterotropia (4.1%), Basal ganglia change with calcification (4.1%), Encephalomalacia /leukomalacia (8.3%), Focal cortical dysplasia+ heterotropia (4.1%), Space occupying lesion (0%). None of them had changes with Focal gliosis (0%), Space occupying lesion (0%).16.6% of them had normal MRI findings.

- EXPLANATION:** majority of them with occipital epileptiform activity have Focal spike and polyspikes wave (28%). None of them have rthmic delta activity (0%). Some of them focal spike (8%), some have focal slow wave (20%), some have other discharges (16%).



TEMPORAL LOBE EPILEPTIFORM ACTIVITY		
MRI CHANGES	No.	%
Normal	2	9%
Focal gliosis+encephalomalacia/ leukomalacia.	8	36.3%
Focal cortical dysplasia	2	9%
Hippocampal sclerosis	2	9%
Congenital malformation	3	13.6%
Focal gliosis	1	4.5%
Calcification	0	0%
heterotropia	1	4.5%
Basal ganglia change with calcification.	1	4.5%
Encephalomalacia /leukomalacia	0	0%
Focal cortical dysplasia+ heterotropia	1	4.5%
Space occupying lesion	1	4.5%
Focal gliosis + encephalomalacia+heterotropia	0	0%
	T=22	100%

TEMPORAL EPILEPTIFORM ACTIVITY

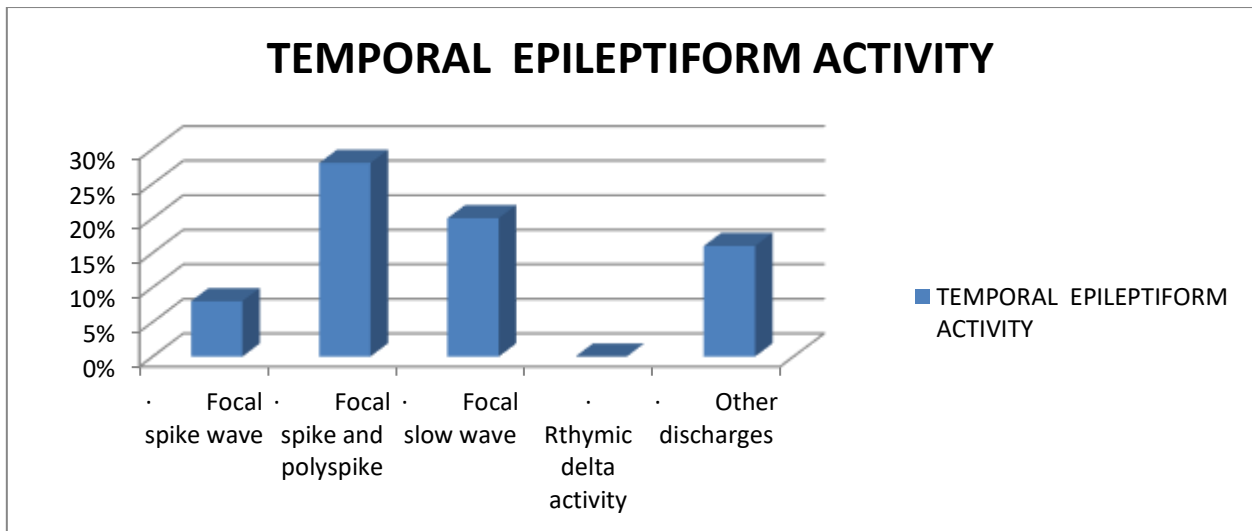


EXPLANATION:

Most of the patients with EEG changes of temporal lobe epileptiform activity had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia(36.3%). Some of them had changes with Focal cortical dysplasia(9%), Congenital malformation(13.6%) , Focal gliosis(4.5%), Space occupying lesion(4.5%), hippocampal sclerosis(9%), Heterotopia(4.5%), Basal ganglia change with calcification(4.5%), Focal cortical dysplasia+ heterotopia(4.5%) , Space occupying lesion(4.5%). None of them had changes with Focal gliosis + encephalomalacia+ heterotopia(0%), Calcification(0%), Encephalomalacia /leukomalacia(0%). 9% of them had normal MRI findings.

TEMPORAL LOBE EPILEPTIFORM ACTIVITY(n=25)		
	No.	%
EEG WAVEFORMS		
Focal spike wave	2	8%
Focal spike and polyspike	7	28%
Focal slow wave	5	20%
Rhythmic delta activity	0	0%
Other discharges	4	16%

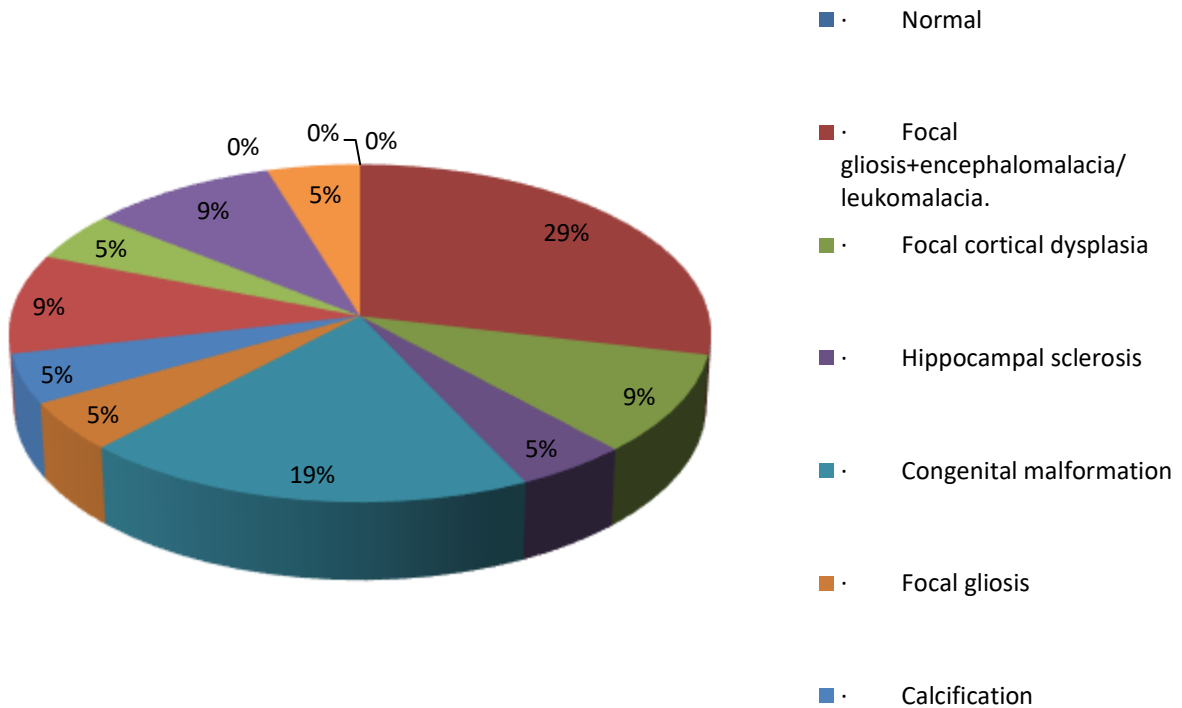
- EXPLANATION:** majority of temporal epileptiform activity have Focal spike and polyspike wave(28%). None of them have rhythmic delta activity(0%). Some of them focal spike (8%), some have focal slow wave(20%), some have other discharges(16%).



PARIETAL LOBE EPILEPTIFORM ACTIVITY

	No.	%
MRI CHANGES		
Normal	0	0%
Focal gliosis+encephalomalacia/ leukomalacia.	6	28.5%
Focal cortical dysplasia	2	9.5%
Hippocampal sclerosis	1	4.7%
Congenital malformation	4	19%
Focal gliosis	1	4.7%
Calcification	1	4.7%
heterotropia	2	9.5%
Basal ganglia change with calcification.	1	4.7%
Encephalomalacia /leukomalacia	2	9.5%
Focal cortical dysplasia+ heterotropia	0	0%
Space occupying lesion	1	4.7%
Focal gliosis + encephalomalacia+heterotropia	0	0%
	T=21	100%

PARIETAL LOBE EPILEPTIFORM ACTIVITY



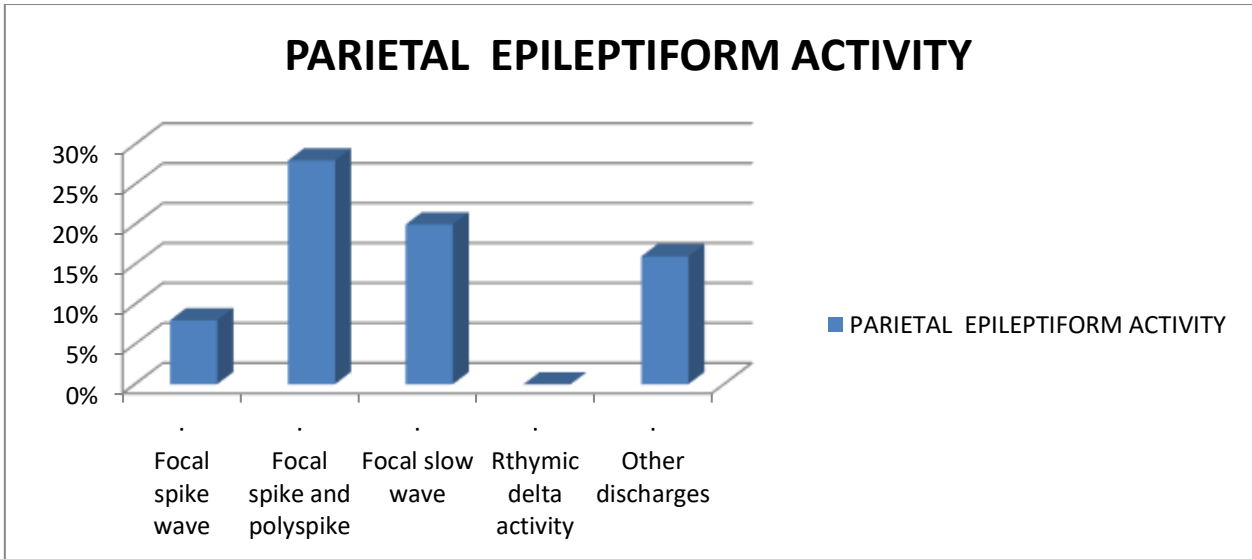
EXPLANATION:

Most of the patients with EEG changes of parietal lobe epileptiform activity had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia(28.5%). Some of them had changes with Focal cortical dysplasia(9.5%), Congenital malformation(19%) , Focal gliosis(4.7%), Calcification(1%), Encephalomalacia /leukomalacia(9.5%),Space occupying lesion(4.7%), hippocampal sclerosis(4.7%), Heterotropia(9.5%), Basal ganglia change with calcification(4.7%), Focal cortical dysplasia+ heterotropia(0%) , Space occupying lesion(4.7%). None of them had changes with Focal gliosis + encephalomalacia+ heterotropia(0%), Focal cortical dysplasia+ heterotropia(0%), normal MRI findings(0%).

PARIETAL LOBE EPILEPTIFORM ACTIVITY(n=25)

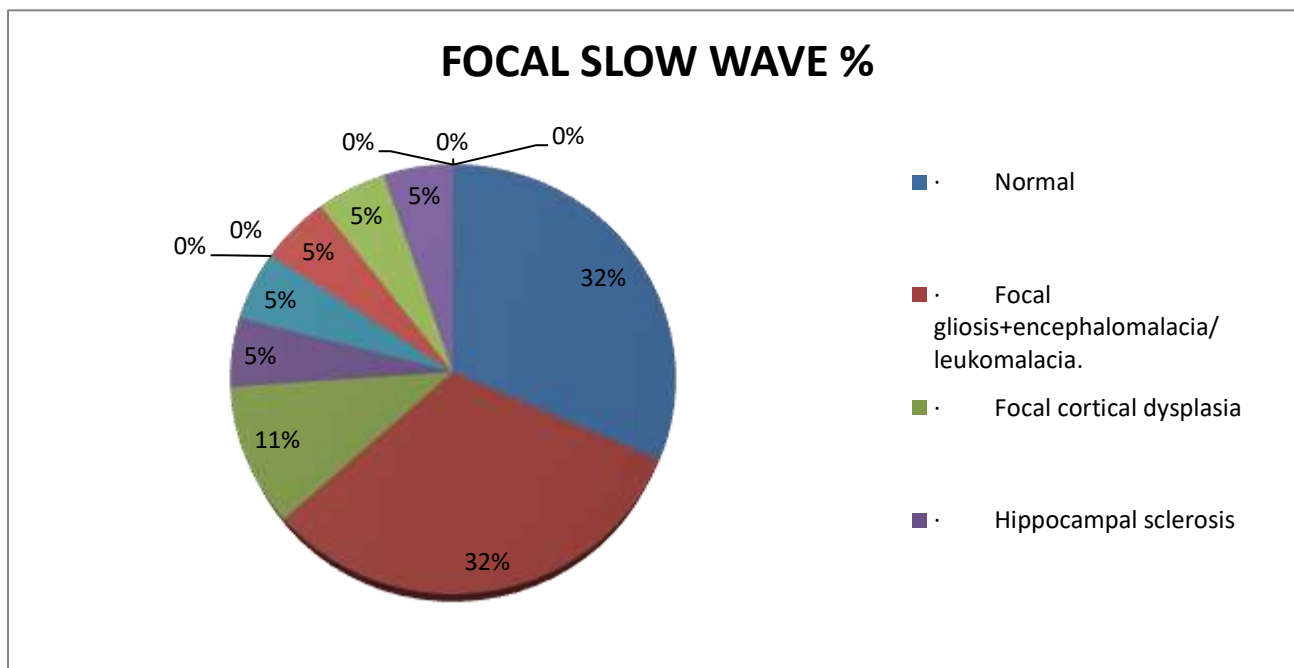
	No.	%
EEG WAVEFORMS		
Focal spike wave	2	8%
Focal spike and polyspike	7	28%
Focal slow wave	5	20%
Rhythmic delta activity	0	0%
Other discharges	4	16%

- **EXPLANATION:** majority of parietal epileptiform activity have Focal spike and polyspike wave(28%). None of them have rhythmic delta activity(0%). Some of them focal spike (8%), some have focal slow wave(20%), some have other discharges(16%).



PROFILE OF PATIENT BASED ON EEG WAVEFORMS WITH MRI CHANGES:

FOCAL SLOW WAVE		
	No.	%
MRI CHANGES:		
Normal	6	31.5%
Focal gliosis+encephalomalacia/ leukomalacia.	6	31.5%
Focal cortical dysplasia	2	10.52%
Hippocampal sclerosis	1	5.2%
Congenital malformation	1	5.2%
Focal gliosis	0	0%
Calcification		0 0%
heterotropia	1	5.2%
Basal ganglia change with calcification.	1	5.2%
Encephalomalacia /leukomalacia	1	5.2%
Focal cortical dysplasia+ heterotropia	0	0%
Space occupying lesion	0	0%
Focal gliosis + encephalomalacia+heterotropia	0	0%
		T=19100%

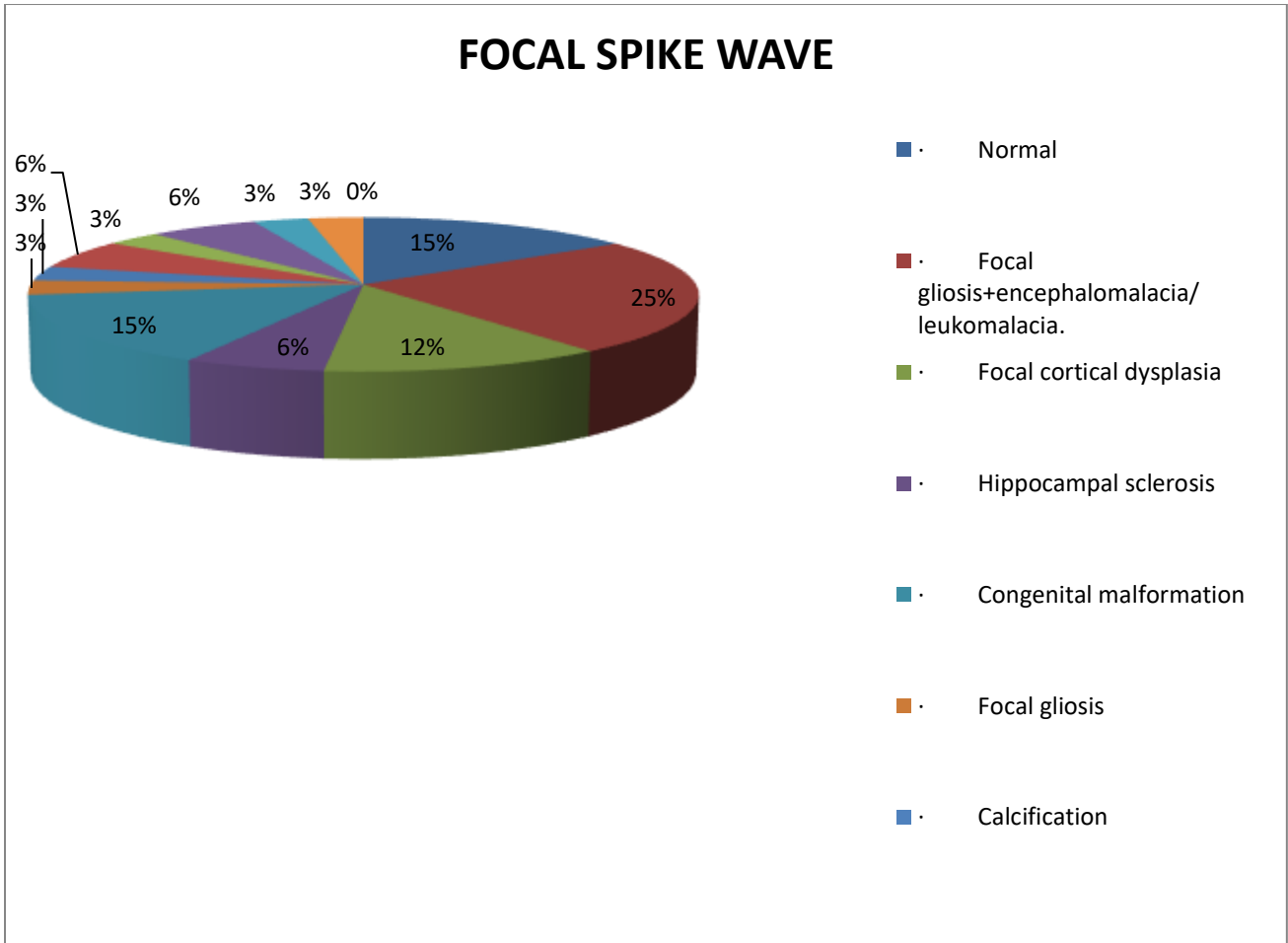


EXPLANATION:

Most of the patients with EEG changes of focal slow wave had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia(31.5%) . Majority also showed normal MRI findings(31.5%). Some of them had changes with Focal cortical dysplasia(10.52%), Congenital malformation(5.2%) , Focal gliosis(0%), Encephalomalacia /leukomalacia(5.2%), hippocampal sclerosis(5.2%), Heterotropia(5.2%), Basal ganglia change with calcification(5.2%), Focal cortical dysplasia+ heterotropia(0%) .None of them had changes with Focal gliosis + encephalomalacia+heterotropia(0%), Focal cortical dysplasia+ heterotropia(0%), Calcification(0%), Space occupying lesion(0%).

FOCAL SPIKE WAVE

	No.	%
MRI CHANGES		
Normal	5	15.1%
Focal gliosis+encephalomalacia/ leukomalacia	8	24.2%
Focal cortical dysplasia	4	12.1%
Hippocampal sclerosis	2	6%
Congenital malformation	5	15.1%
Focal gliosis	1	3%
Calcification	1	3%
heterotropia	2	6%
Basal ganglia change with calcification.	1	3%
Encephalomalacia /leukomalacia	2	6%
Focal cortical dysplasia+ heterotropia	1	3%
Space occupying lesion	1	3%
Focal gliosis + encephalomalacia+heterotropia	0	0%
		T=33100%



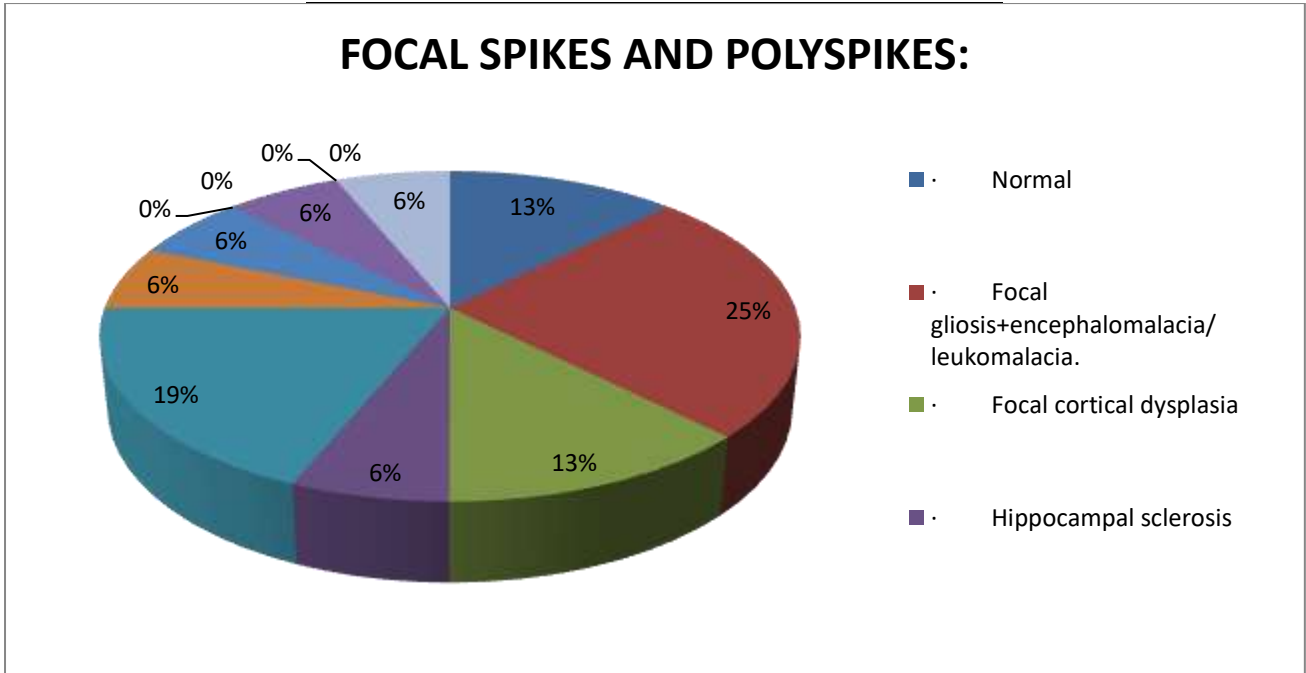
EXPLANATION:

Most of the patients with EEG changes of focal spike wave had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia(24.2%) . Some of them had changes with Focal cortical dysplasia(12.1%), Congenital malformation(15.1%) , Focal gliosis(3%), Encephalomalacia /leukomalacia(6%), hippocampal sclerosis(6%), Calcification(3%), Space occupying lesion(3%), Heterotropia(6%), Basal ganglia change with calcification(3%), Focal cortical dysplasia+ heterotropia(3%) .None of them had changes with Focal gliosis + encephalomalacia+ heterotropia(0%). 15.1% of them had normal MRI changes.

FOCAL SPIKES AND POLYSPIKES:

	No.	%
MRI CHANGES		
Normal	2	12.5%
Focal gliosis+encephalomalacia/ leukomalacia	4	25%
Focal cortical dysplasia	2	12.5%
Hippocampal sclerosis	1	6.25%
Congenital malformation	3	18.7%
Focal gliosis	1	6.25%
Calcification	1	6.25%
Heterotropia	0	0%
Basal ganglia change with calcification.	0	0%
Encephalomalacia /leukomalacia	1	6.25%
Focal cortical dysplasia+ heterotropia	0	0%
Space occupying lesion	0	0%
Focal gliosis + encephalomalacia+heterotropia	1	6.25%

T=16100%

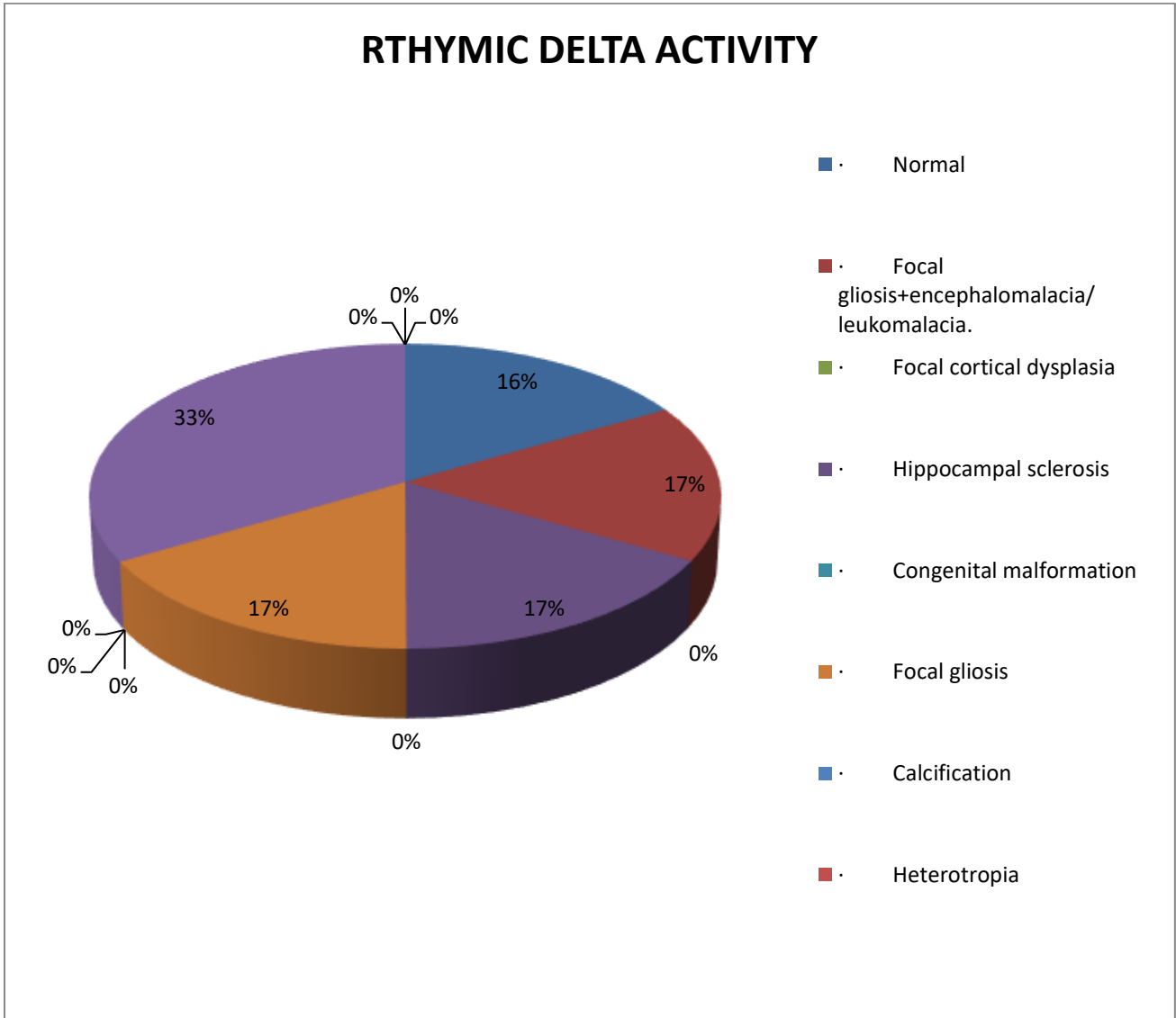


EXPLANATION:

Most of the patients with EEG changes of focal spike and polyspikes had abnormal MRI changes with focal gliosis with encephalomalacia/leukomalacia(25%) . Some of them had changes with Focal cortical dysplasia(12.5%), Congenital malformation(18.7%) , Focal gliosis + encephalomalacia+ heterotropia(6.25%) Focal gliosis(6.25%), Encephalomalacia /leukomalacia(6.25%), hippocampal sclerosis(6.25%), Calcification(6.25%), .None of them had changes with Space occupying lesion(0%), Heterotropia(0%), Basal ganglia change with calcification(0%), Focal cortical dysplasia+ heterotropia(0%). 12.5% of them had normal MRI changes.

RHYMIC DELTA ACTIVITY

	No.	%
MRI CHANGES		
Normal	1	16.6%
Focal gliosis+encephalomalacia/ leukomalacia	1	16.6%
Focal cortical dysplasia	0	0%
Hippocampal sclerosis	1	16.6%
Congenital malformation	0	0%
Focal gliosis	1	16.6%
Calcification	0	0%
Heterotropia	0	0%
Basal ganglia change with calcification.	0	0%
Encephalomalacia /leukomalacia	2	33.3%
Focal cortical dysplasia+ heterotropia	0	0%
Space occupying lesion	0	0%
Focal gliosis + encephalomalacia+heterotropia	0	0%
		T=6100%

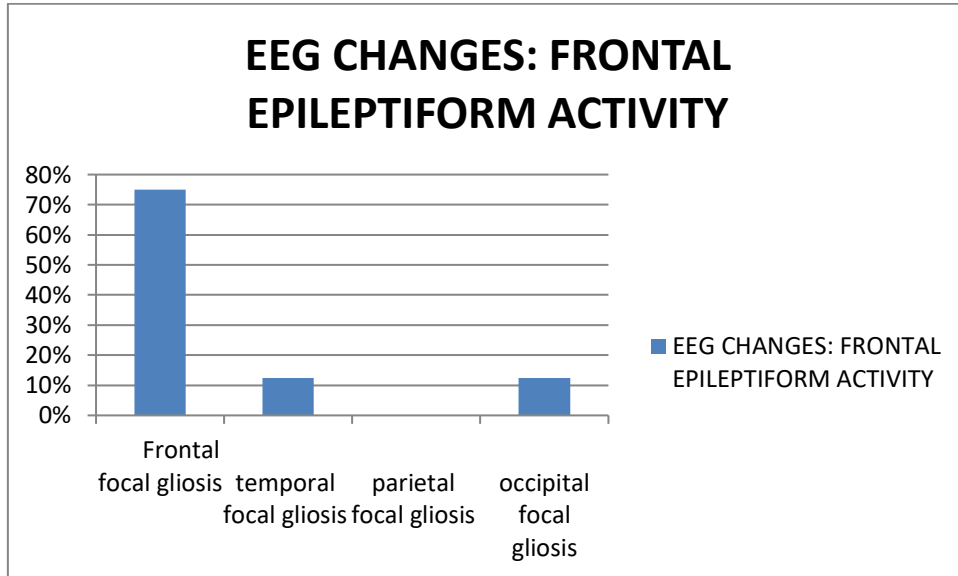


EXPLANATION:

Most of the patients with EEG changes of rhythmic delta activity had abnormal MRI changes encephalomalacia/leukomalacia(33.3%). Some of them had changes with Focal gliosis(16.6%), Focal gliosis+encephalomalacia/leukomalacia(16.6%),hippocampal sclerosis(16.6%), Calcification(0%), None of them had changes with Space occupying lesion(0%), Focal cortical dysplasia(0%), Congenital malformation(0%) , Focal gliosis + encephalomalacia+heterotropia(0%) Heterotropia(0%), Basal ganglia change with calcification(0%), Focal cortical dysplasia+heterotropia(0%). 16.6% of them had normal MRI changes.

FRONTAL EPILEPTIFORM ACTIVITY WITH FOCAL GLIOSIS(N=8)

	No.	%
MRI CHANGES		
Frontal focal gliosis	6	75%
temporal focal gliosis	1	12.5%
parietal focal gliosis	0	nil
occipital focal gliosis	1	12.5%



EXPLANATION:

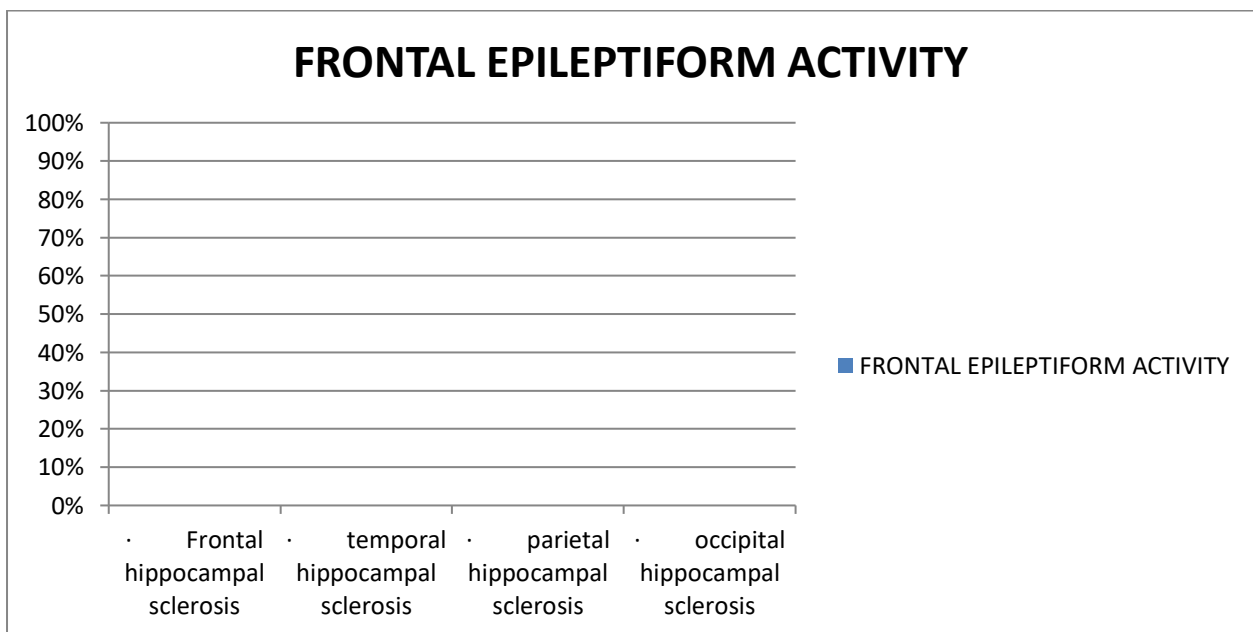
Majority of them with frontal lobe epileptiform activity have focal gliosis in frontal lobe(75%). None of them have focal gliosis in parietal lobe. Some of them have focal gliosis in temporal(12.5%) and occipital lobe(12.5%).

FRONTAL EPILEPTIFORM ACTIVITY WITH HIPPOCAMPAL SCLEROSIS(N=0)

	No.	%
MRI CHANGES		
Frontal hippocampal sclerosis	0	Nil
temporal hippocampal sclerosis	0	Nil
parietal hippocampal sclerosis	0	Nil
occipital hippocampal sclerosis	0	Nil

EXPLANATION:

None of them with frontal lobe epileptiform have hippocampal sclerosis.

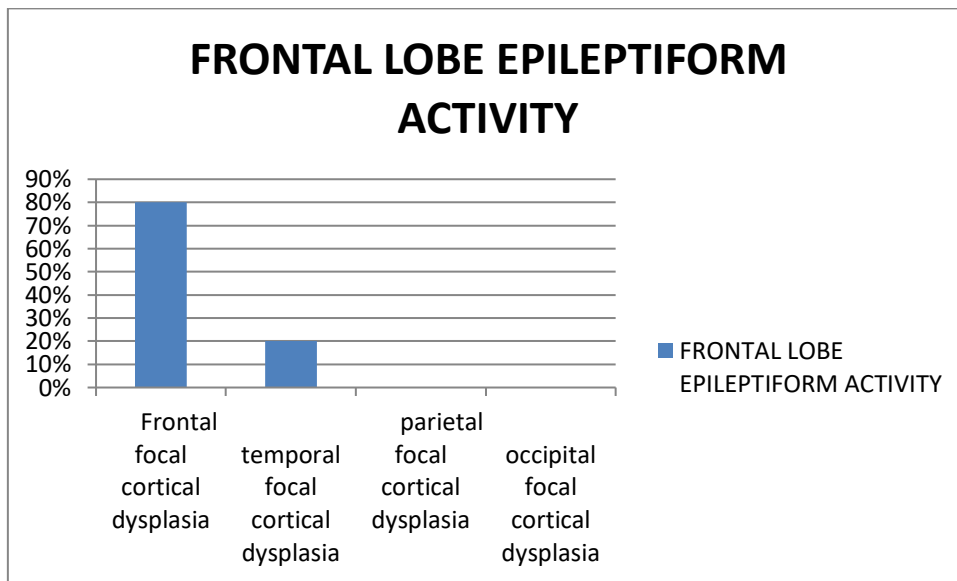


FRONTAL EPILEPTIFORM ACTIVITY WITH FOCAL CORTICAL DYSPLASIA (N=5)

	No.	%
MRI CHANGES		
Frontal focal cortical dysplasia	4	80%
temporal focal cortical dysplasia	1	20%
parietal focal cortical dysplasia	0	Nil
occipital focal cortical dysplasia	0	Nil

EXPLANATION:

Majority of them with frontal lobe epileptiform activity have focal cortical dysplasia in frontal lobe(80%). None of them have focal cortical dysplasia in parietal and occipital lobe. Some of them have focal cortical dysplasia in temporal lobe(20%).

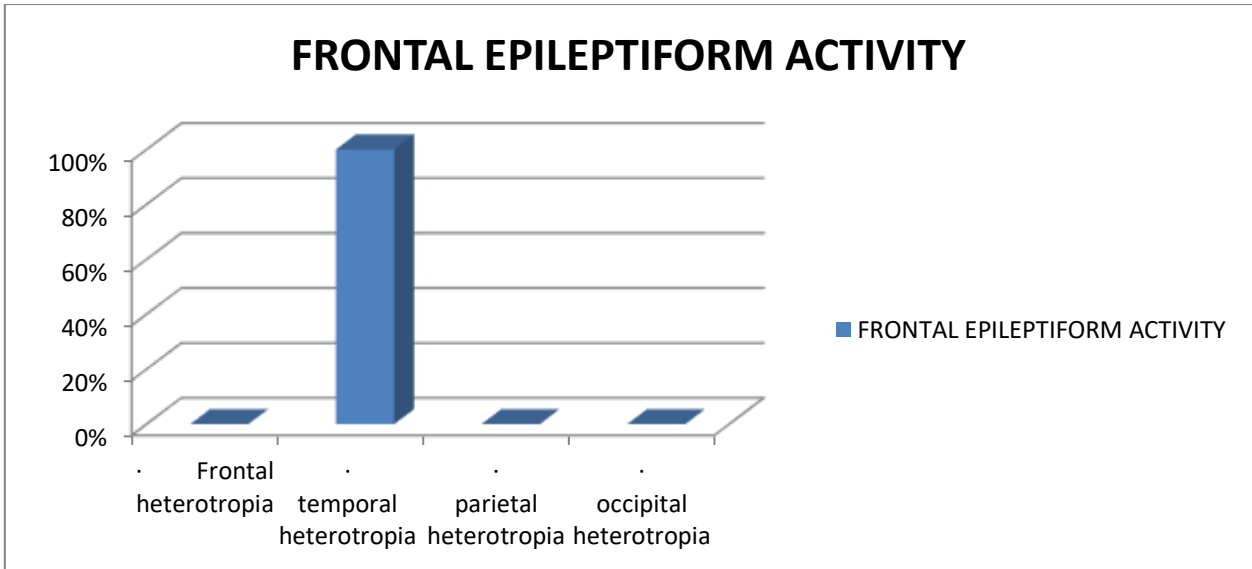


FRONTAL EPILEPTIFORM ACTIVITY WITH HETEROTROPIA (N=2)

	No.	%
MRI CHANGES		
Frontal heterotropia	0	Nil
temporal heterotropia	2	100%
parietal heterotropia	0	Nil
occipital heterotropia	0	Nil

EXPLANATION:

All of them with frontal lobe epileptiform activity have heterotropia in temporal lobe.(100%). None of them have heterotropia in frontal, parietal and occipital lobe.

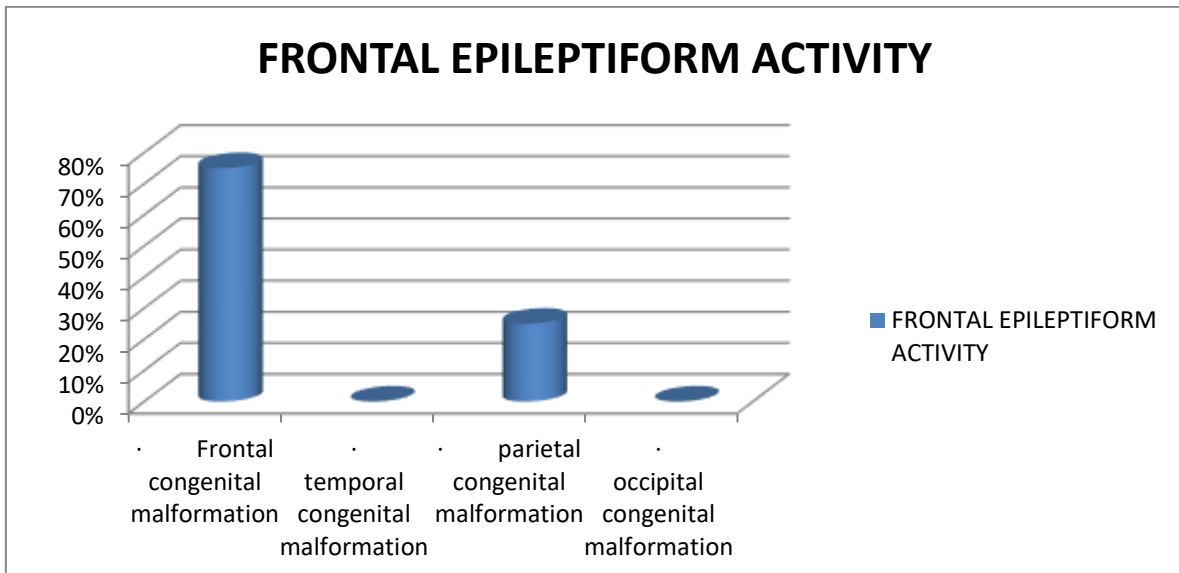


FRONTAL EPILEPTIFORM ACTIVITY WITH CONGENITAL MALFORMATION (N=4)

	<i>No.</i>	<i>%</i>
MRI CHANGES		
Frontal congenital malformation	3	75%
temporal congenital malformation	0	Nil
parietal congenital malformation	1	25%
occipital congenital malformation	0	Nil

EXPLANATION:

Majority of them with frontal lobe epileptiform activity have congenital malformation in frontal lobe(75%). None of them have congenital malformation in temporal and occipital lobe. Some of them have congenital malformation in parietal lobe(25%).

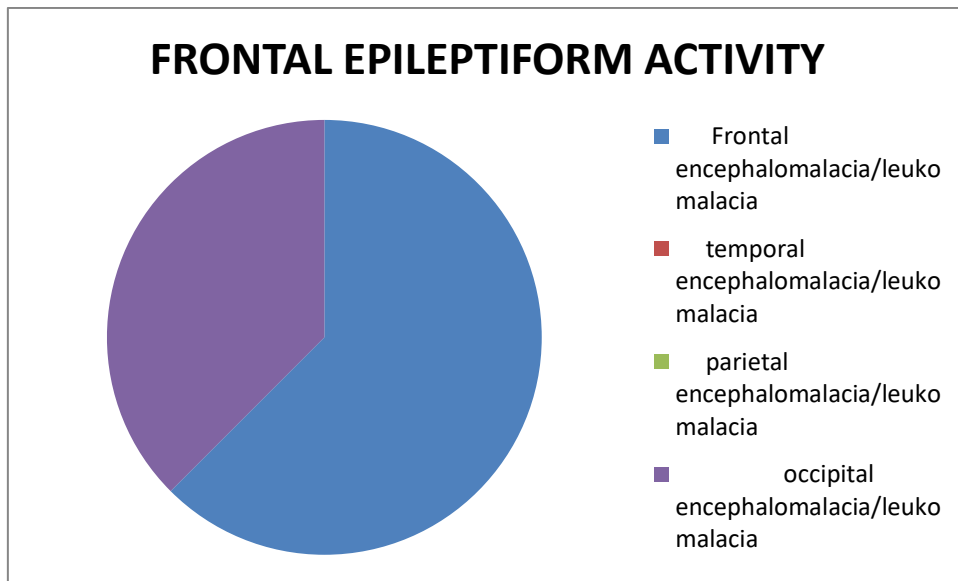


FRONTAL EPILEPTIFORM ACTIVITY WITH ENCEPHALOMALACIA/LEUKOMALACIA (N=8)

	No.	%
MRI CHANGES		
Frontal encephalomalacia/leukomalacia	5	62.5%
temporal encephalomalacia/leukomalacia	0	nil
parietal encephalomalacia/leukomalacia	0	nil
occipital encephalomalacia/leukomalacia	3	37.5%

EXPLANATION:

Majority of them with frontal lobe epileptiform activity have encephalomalacia/leukomalacia in frontal lobe(62.5%). None of them have encephalomalacia/leukomalacia in temporal and parietal lobe(0%). Some of them have encephalomalacia/leukomalacia in occipital lobe(37.5%).

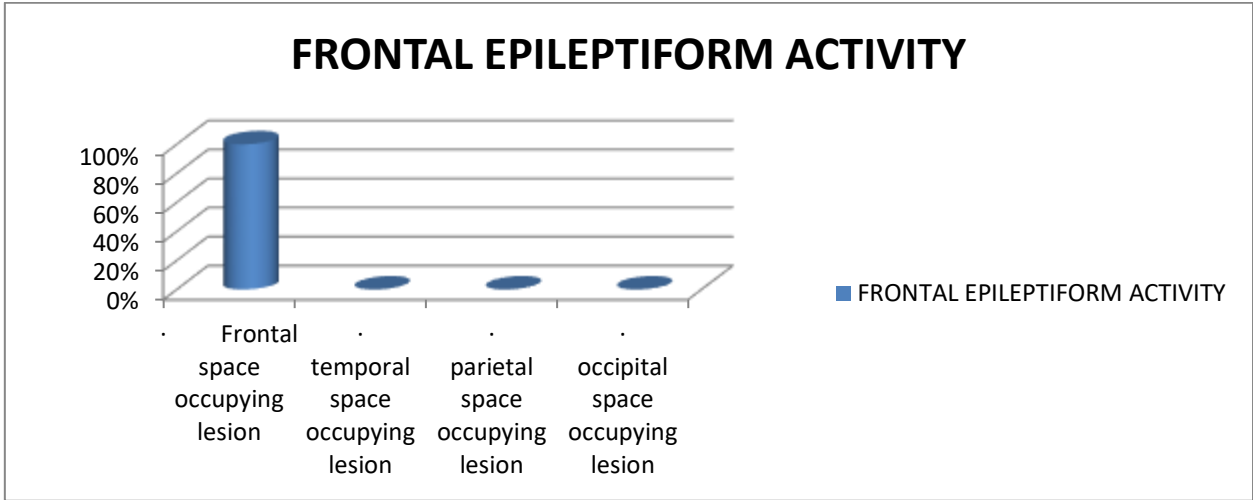


FRONTAL EPILEPTIFORM ACTIVITY WITH SPACE OCCUPYING LESION (N=1)

	No.	%
MRI CHANGES		
Frontal space occupying lesion	1	100%
temporal space occupying lesion	0	0%
parietal space occupying lesion	0	0%
occipital space occupying lesion	0	0%

EXPLANATION:

All of them with frontal lobe epileptiform activity have space occupying lesion in frontal lobe(100%). None of them have in occipital, temporal, parietal lobe(0%).

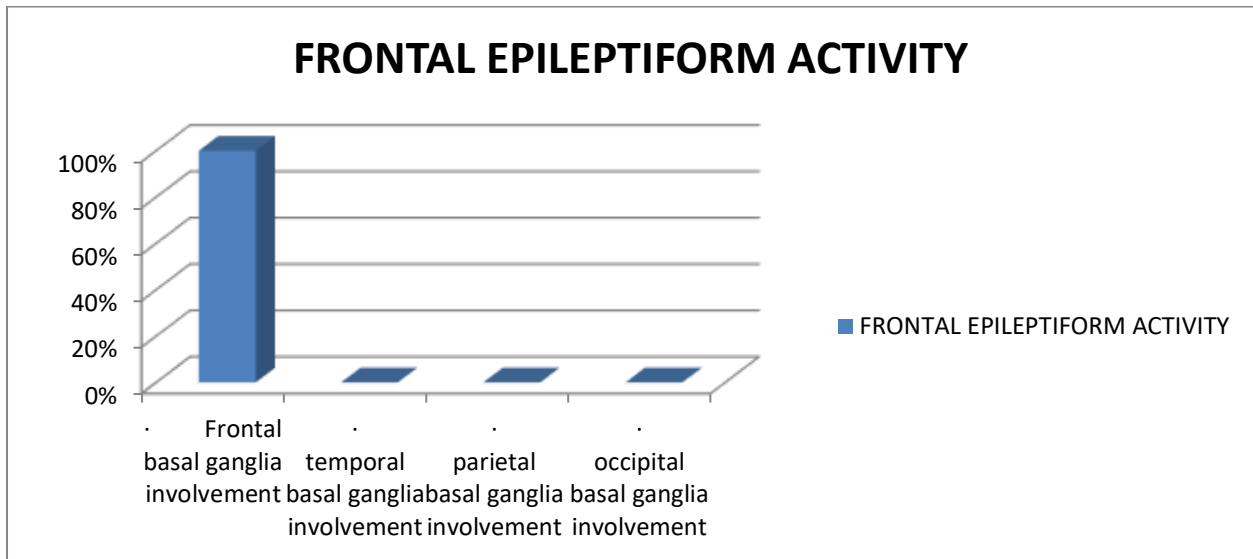


FRONTAL EPILEPTIFORM ACTIVITY WITH BASAL GANGLIA INVOLVEMENT (N=1)

	No.	%
MRI CHANGES		
Frontal basal ganglia involvement	1	100%
temporal basal ganglia involvement	0	0%
parietal basal ganglia involvement	0	0%
occipital basal ganglia involvement	0	0%

EXPLANATION:

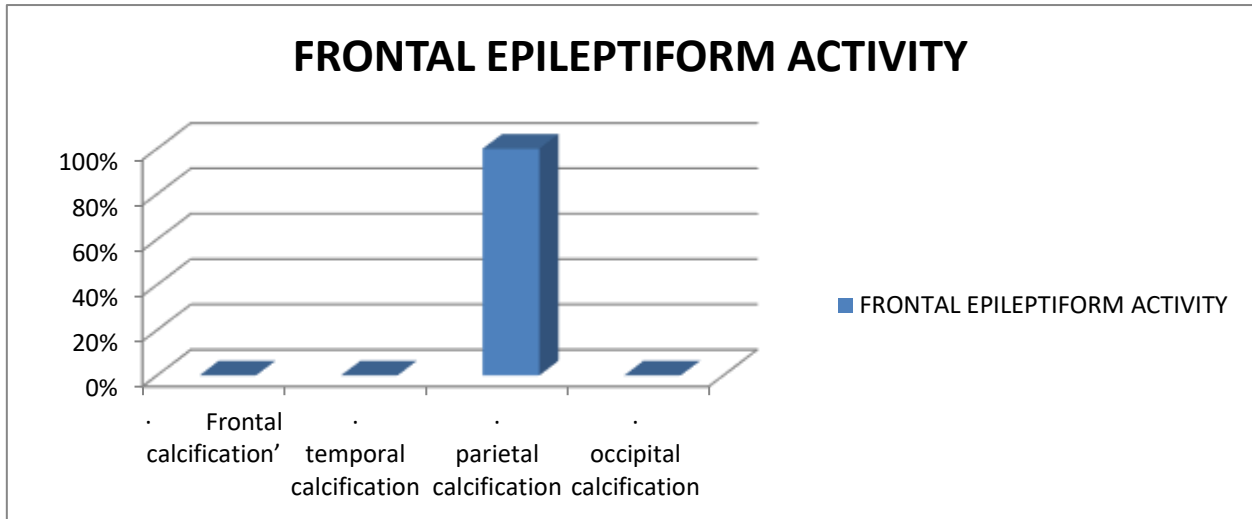
All of them with frontal lobe epileptiform activity have basal ganglia involvement in frontal lobe(100%). None of them have in occipital, temporal, parietal lobe(0%).



FRONTAL EPILEPTIFORM ACTIVITY WITH CALCIFICATION (N=2)

	No.	%
MRI CHANGES		
Frontal calcification'	0	Nil
temporal calcification	0	Nil
parietal calcification	1	100%
occipital calcification	0	Nil

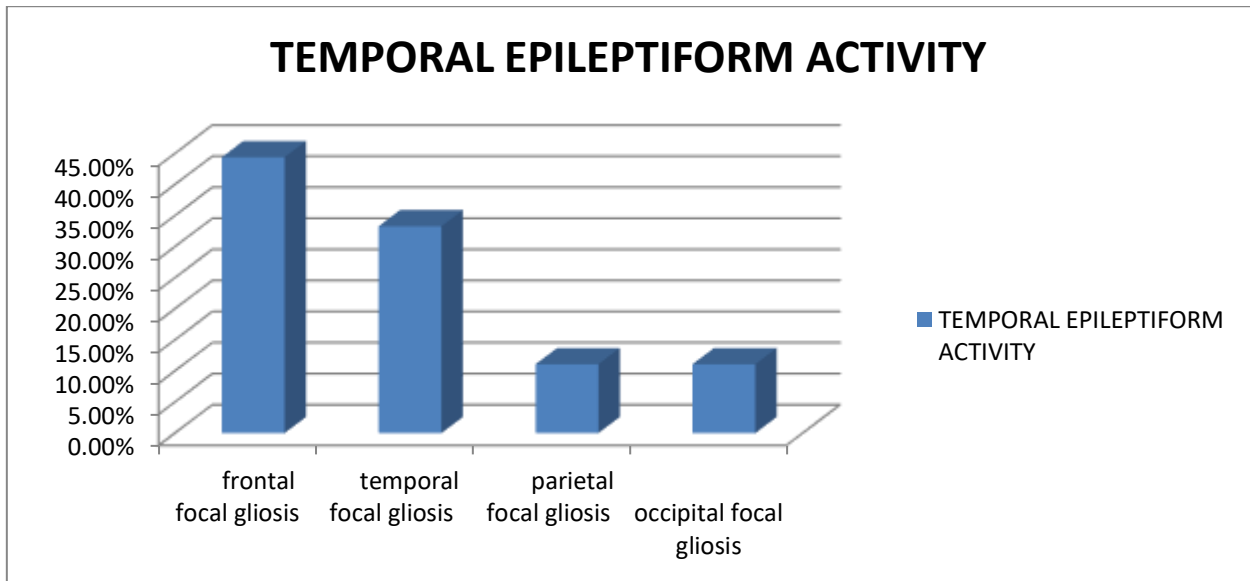
EXPLANATION: All of them with frontal lobe epeleptiform activity have calcification in parietal lobe(100%). None of them have in occipital, temporal, frontal lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH FOCAL GLIOSIS(N=9)

	No.	%
MRI CHANGES		
frontal focal gliosis	4	44.4%
temporal focal gliosis	3	33.3%
parietal focal gliosis	1	11.1%
occipital focal gliosis	1	11.1%

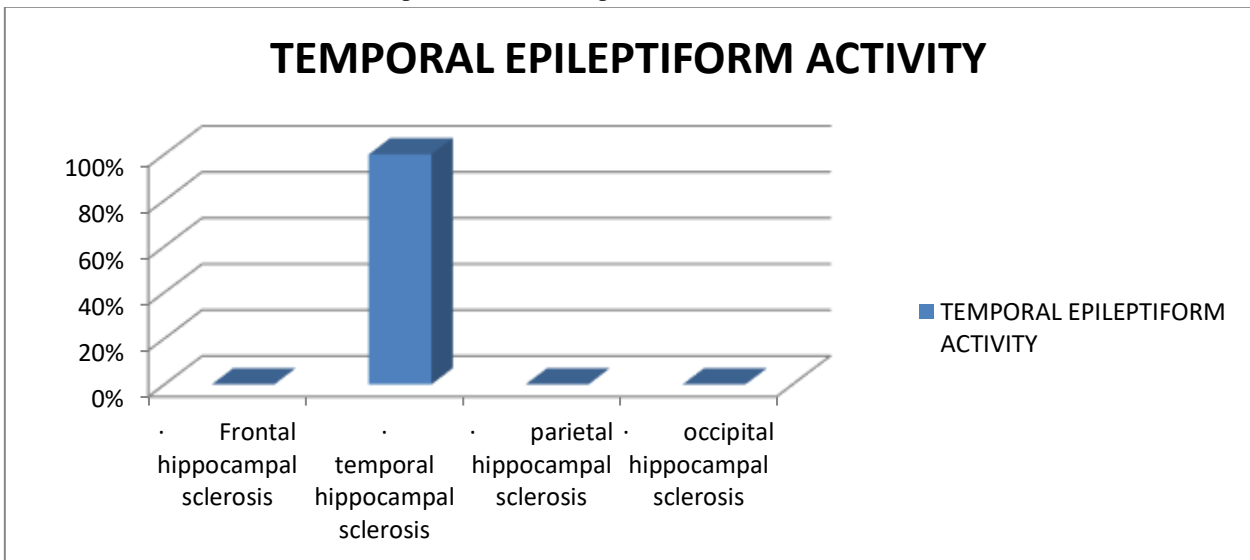
EXPLANATION: majority of them with temporal lobe epileptiform activity have focal gliosis in frontal lobe(44.4%). Least number of them have in parietal and occipital lobe(11.1%). Some of them have in temporal lobe(33.3%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH HIPPOCAMPAL SCLEROSIS(N=2)

	No.	%
MRI CHANGES		
Frontal hippocampal sclerosis	0	Nil
temporal hippocampal sclerosis	2	100%
parietal hippocampal sclerosis	0	Nil
occipital hippocampal sclerosis	0	Nil

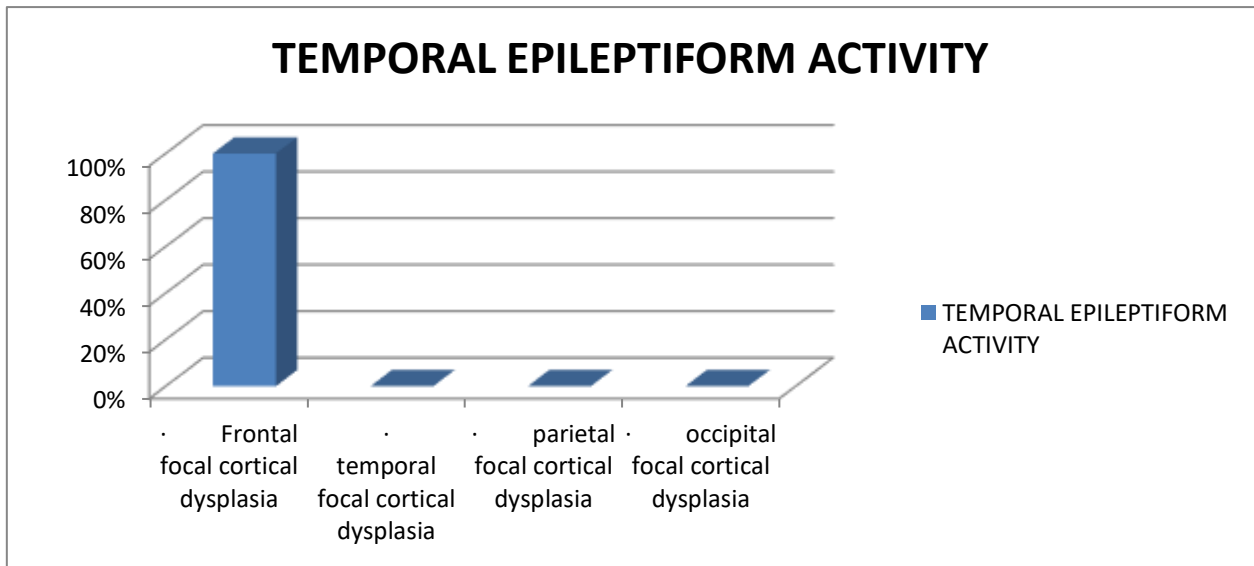
EXPLANATION: All of them with temporal lobe epileptiform activity have hippocampal sclerosis in temporal lobe(100%). None of them have in occipital, frontal , and parietal lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH FOCAL CORTICAL DYSPLASIA(N=3)

	No.	%
MRI CHANGES		
Frontal focal cortical dysplasia	3	100%
temporal focal cortical dysplasia	0	nil
parietal focal cortical dysplasia	0	nil
occipital focal cortical dysplasia	0	nil

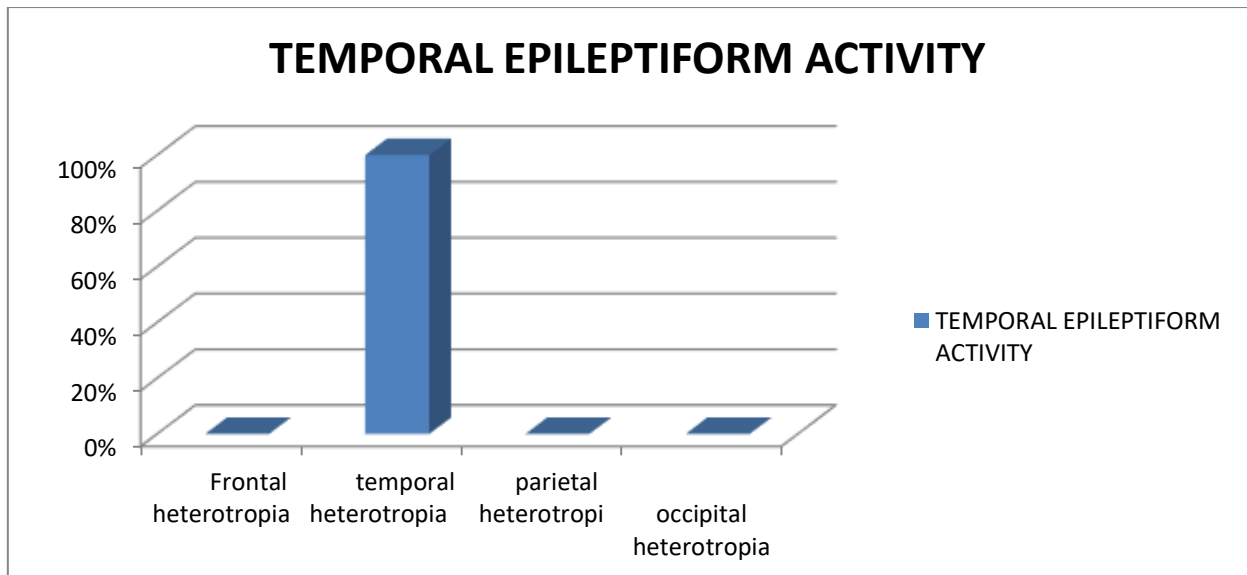
EXPLANATION: All of them with temporal lobe epileptiform activity have focal cortical dysplasia in frontal lobe. None of them have in occipital, temporal, and parietal lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH HETEROTROPIA (N=2)

MRI CHANGES	No.	%
Frontal heterotopia	0	Nil
temporal heterotopia	2	100%
parietal heterotopia	0	Nil
occipital heterotopia	0	Nil

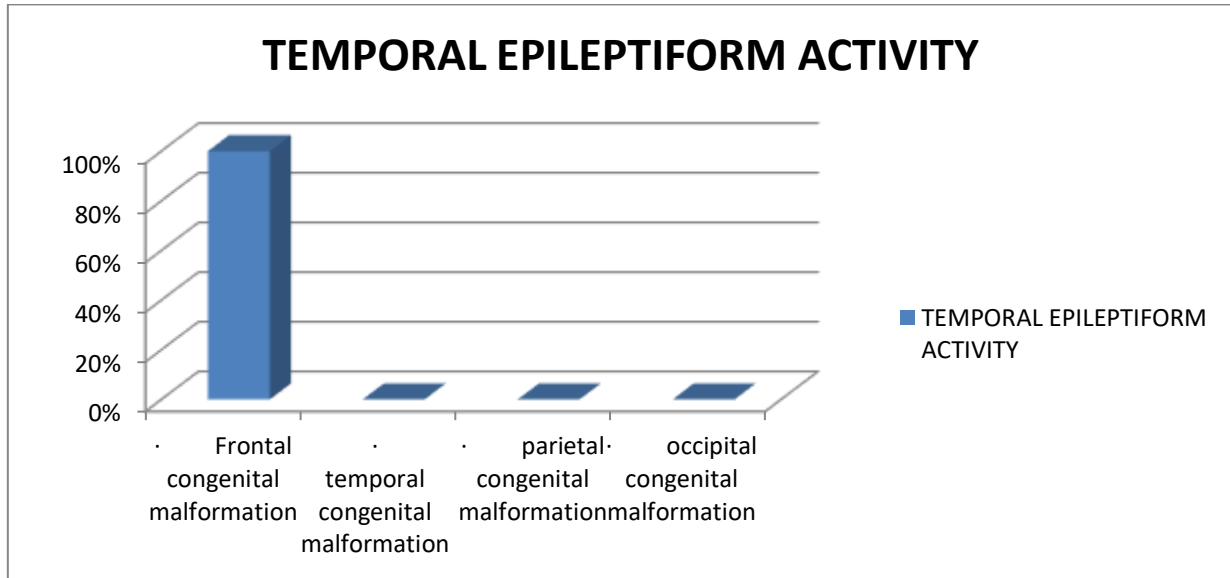
EXPLANATION: All of them with temporal lobe epileptiform activity have heterotopia in temporal lobe(100%). None of them have in occipital, frontal, and parietal lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH CONGENITAL MALFORMATION (N=3)

	No.	%
MRI CHANGES		
Frontal congenital malformation	3	100%
temporal congenital malformation	0	Nil
parietal congenital malformation	0	Nil
occipital congenital malformation	0	Nil

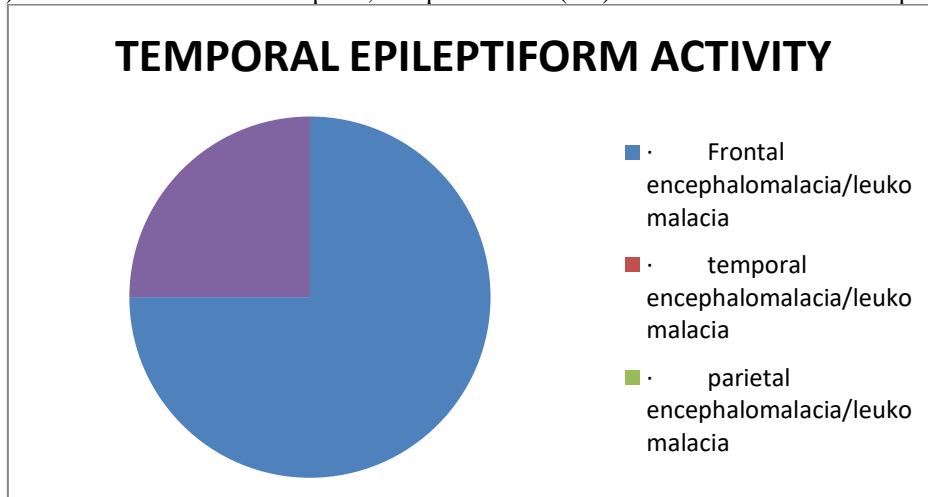
EXPLANATION: All of them with temporal lobe epileptiform activity have congenital malformation in frontal lobe(100%). None of them have in occipital, temporal, and parietal lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH ENCEPHALOMALACIA/LEUKOMALACIA (N=8)

	No.	%
MRI CHANGES		
Frontal encephalomalacia/leukomalacia	6	75%
temporal encephalomalacia/leukomalacia	0	Nil
parietal encephalomalacia/leukomalacia	0	Nil
occipital encephalomalacia/leukomalacia	2	25%

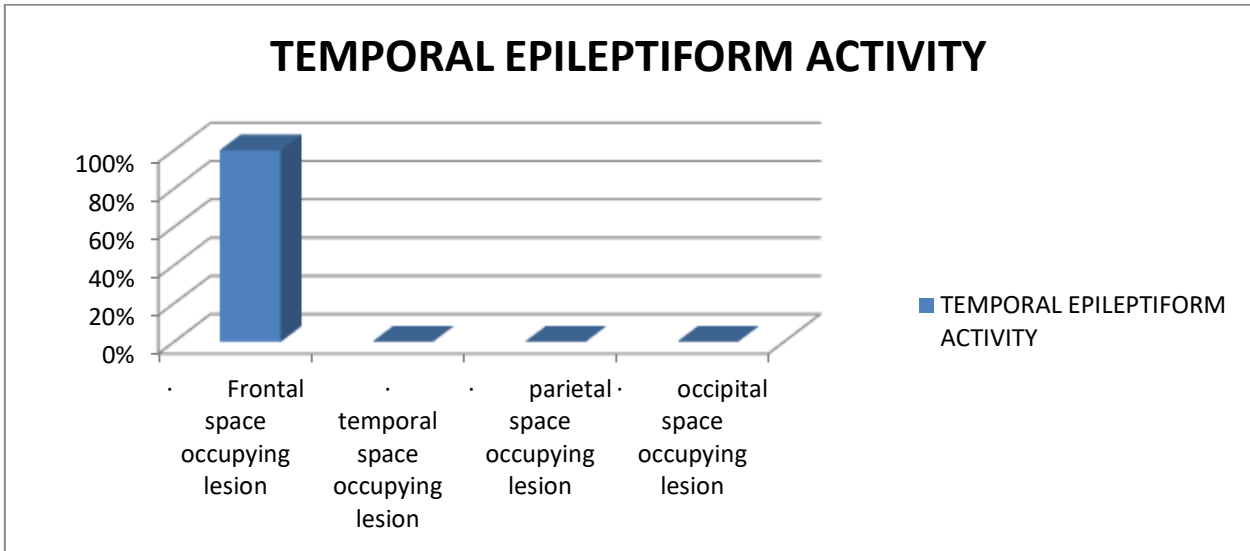
EXPLANATION: Majority of them with temporal lobe epileptiform activity have encephalomalacia/leukomalacia in frontal lobe(75%). None of them have in temporal, and parietal lobe(0%).some of them have in occipital lobe(25%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH SPACE OCCUPYING LESION (N=1)

MRI CHANGES	No.	%
Frontal space occupying lesion	1	100%
temporal space occupying lesion	0	0%
parietal space occupying lesion	0	0%
occipital space occupying lesion	0	0%

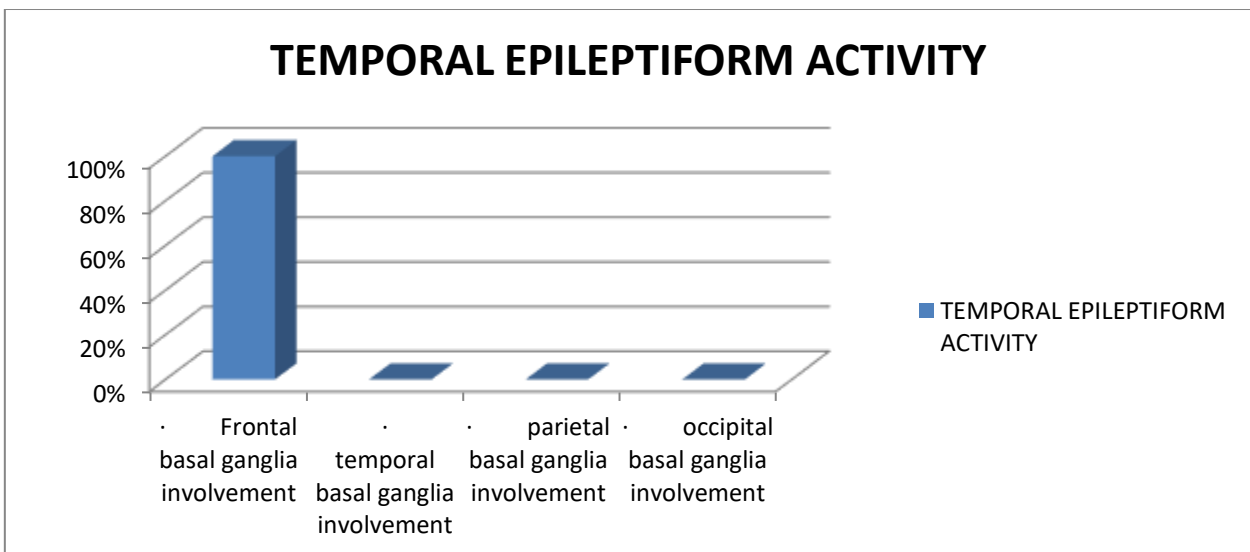
EXPLANATION: All of them with temporal lobe epileptiform activity have space occupying lesion in frontal lobe(100%). None of them have in temporal, parietal lobeand occipital lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH BASAL GANGLIA INVOLVEMENT (N=1)

MRI CHANGES	No.	%
Frontal basal ganglia involvement	1	100%
temporal basal ganglia involvement	0	0%
parietal basal ganglia involvement	0	0%
occipital basal ganglia involvement	0	0%

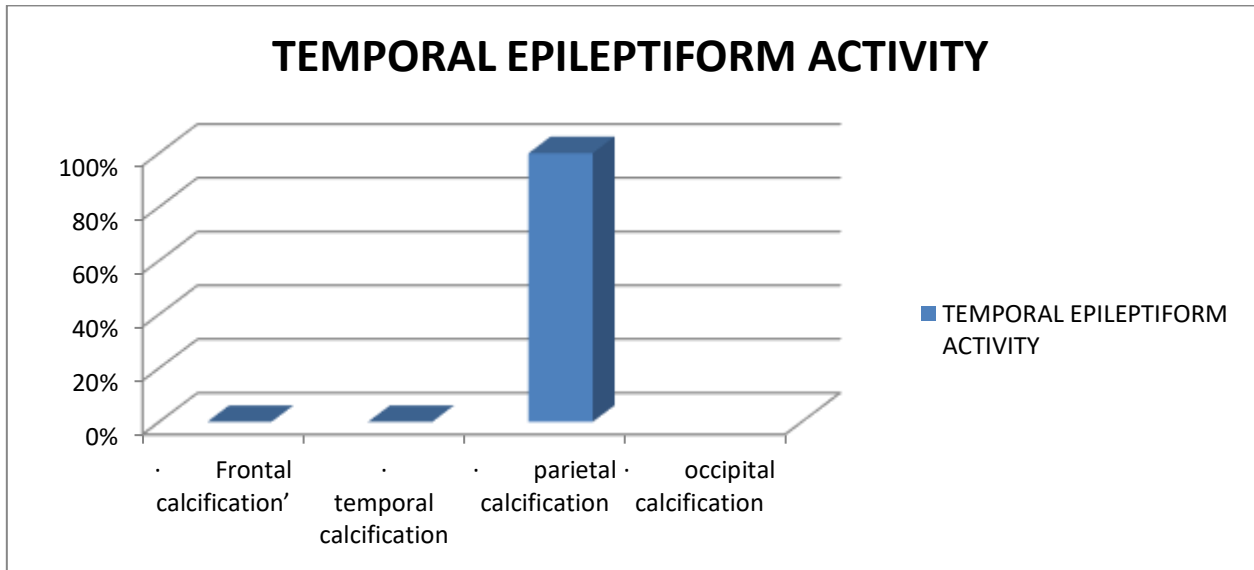
EXPLANATION: All of them with temporal lobe epileptiform activity have basal ganglia involvement in frontal lobe(100%). None of them have in temporal, parietal and occipital lobe(0%).



TEMPORAL EPILEPTIFORM ACTIVITY WITH CALCIFICATION (N=1)

	No.	%
MRI CHANGES		
Frontal calcification'	0	Nil
temporal calcification	0	nil
parietal calcification	1	100%
occipital calcification	0	nil

EXPLANATION: All of them with temporal lobe epileptiform activity have calcification in parietal lobe(100%). None of them have in temporal, frontal and occipital lobe(0%).

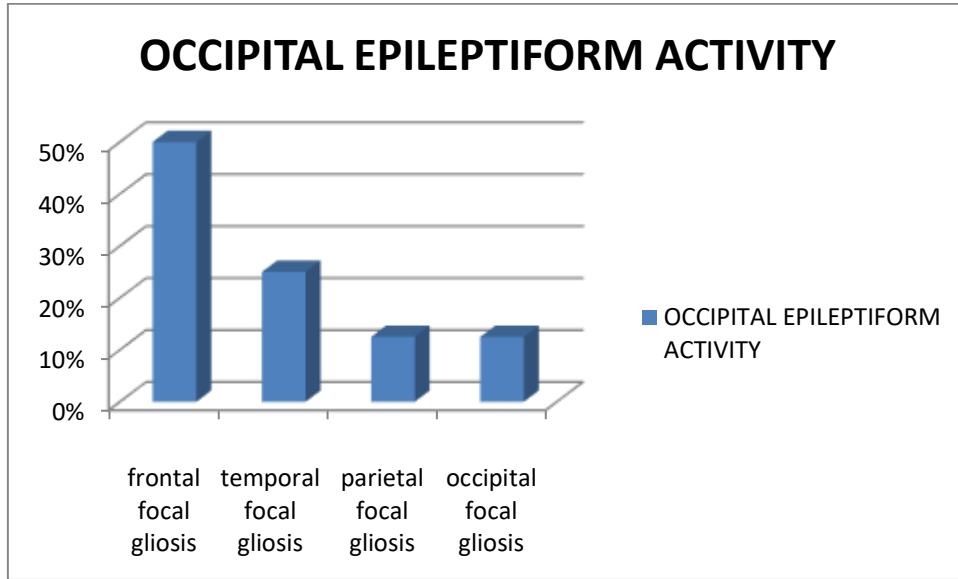


OCCIPITAL EPILEPTIFORM ACTIVITY WITH FOCAL GLIOSIS(N=8)

	No.	%
MRI CHANGES		
frontal focal gliosis	4	50%
temporal focal gliosis	2	25%
parietal focal gliosis	1	12.5%
occipital focal gliosis	1	12.5%

EXPLANATION:

Majority of them with occipital lobe epileptiform activity have focal gliosis in frontal lobe(50%). Least number of them have in parietal and occipital lobe(12.5%). Some of them have in temporal lobe(25%).

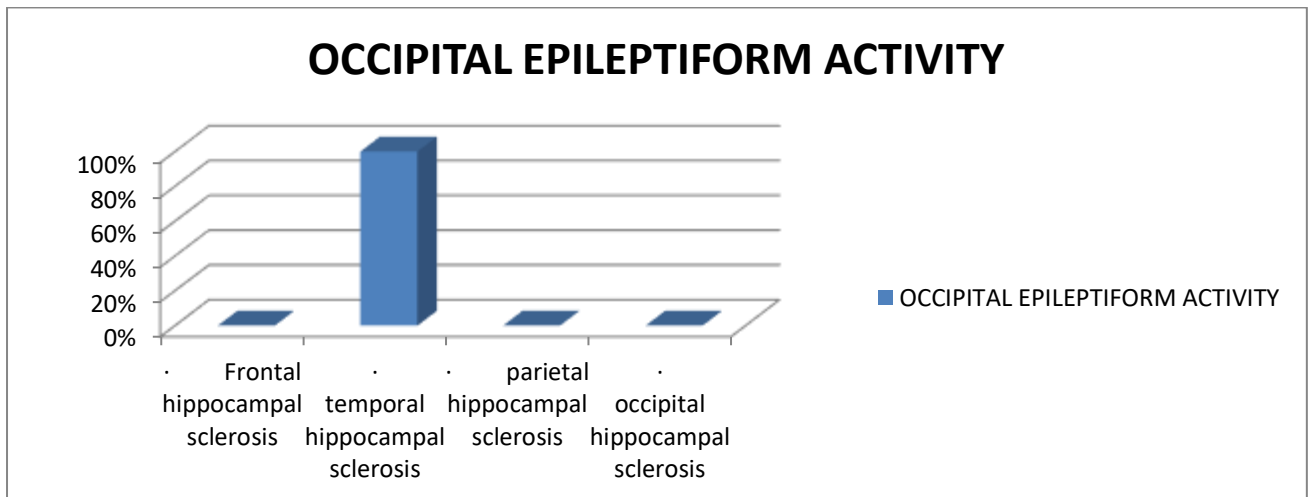


OCCIPITAL EPILEPTIFORM ACTIVITY WITH HIPPOCAMPAL SCLEROSIS(N=1)

	No.	%
MRI CHANGES		
Frontal hippocampal sclerosis	0	Nil
temporal hippocampal sclerosis	1	100%
parietal hippocampal sclerosis	0	Nil
occipital hippocampal sclerosis	0	Nil

EXPLANATION:

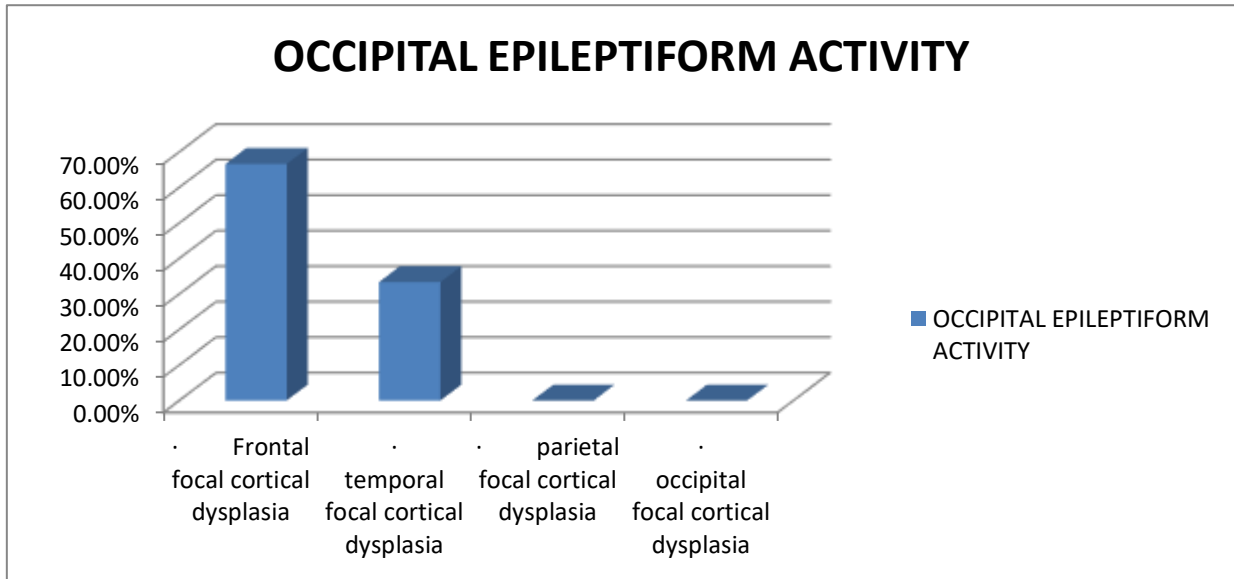
All of them with occipital lobe epileptiform activity have hippocampal sclerosis in temporal lobe(100%). None of them have in parietal, occipital and frontal lobe(0%).



OCCIPITAL EPILEPTIFORM ACTIVITY WITH FOCAL CORTICAL DYSPLASIA(N=3)

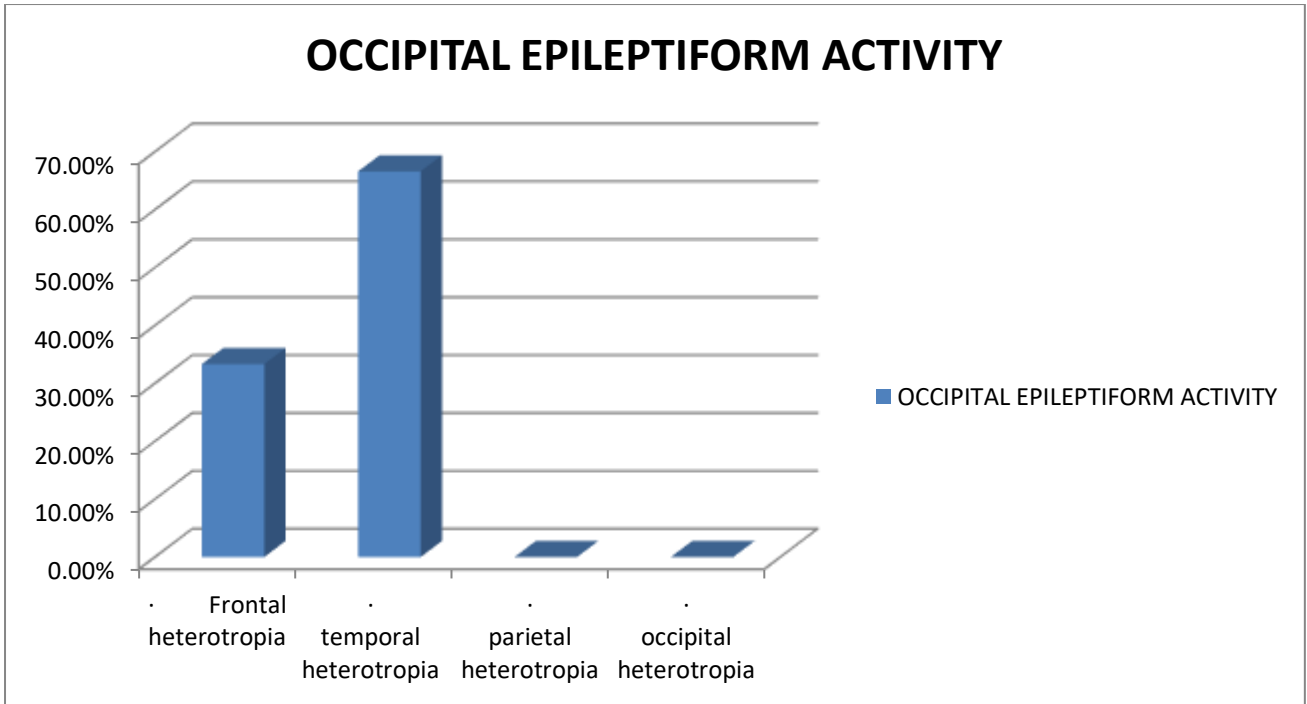
	No.	%
MRI CHANGES		
Frontal focal cortical dysplasia	2	66.6%
temporal focal cortical dysplasia	1	33.3%
parietal focal cortical dysplasia	0	Nil
occipital focal cortical dysplasia	0	Nil

EXPLANATION: Majority of them with occipital lobe epileptiform activity have focal cortical dysplasia in frontal lobe(66.6%). None of them have in parietal and occipital (0%). Some of them have in temporal lobe(33.3%).



OCCIPITAL EPILEPTIFORM ACTIVITY WITH HETEROTROPIA (N=3)

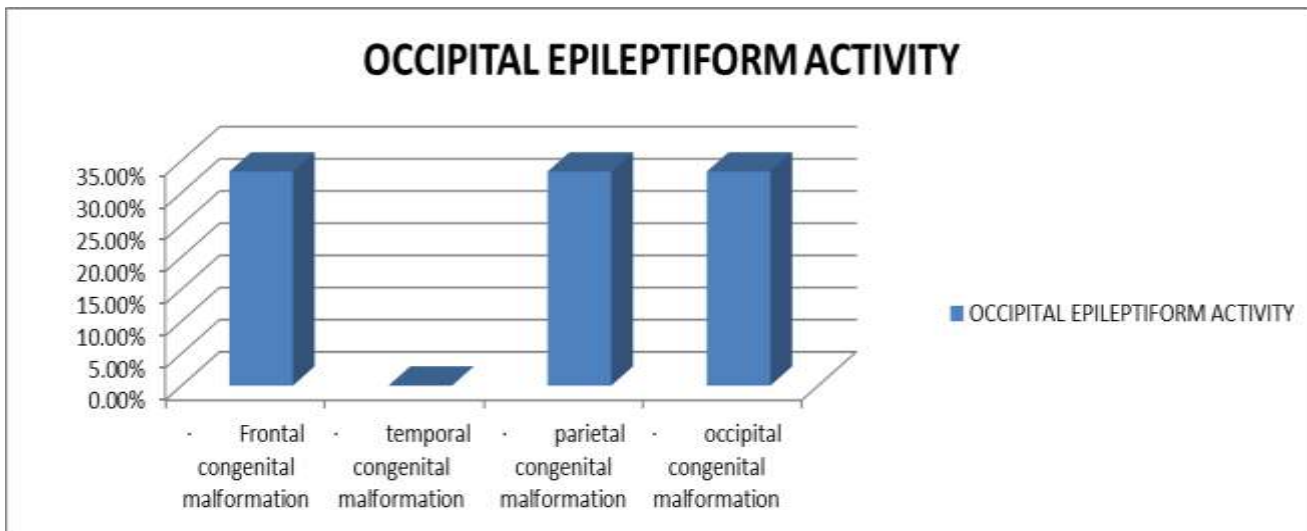
	No.	%
MRI CHANGES		
Frontal heterotropia	1	33.3%
temporal heterotropia	2	66.6%
parietal heterotropia	0	Nil
occipital heterotropia	0	Nil



OCCIPITAL EPILEPTIFORM ACTIVITY WITH CONGENITAL MALFORMATION (N=3)

MRI CHANGES	No.	%
Frontal congenital malformation	1	33.3%
temporal congenital malformation	0	Nil
parietal congenital malformation	1	33.3%
occipital congenital malformation	1	33.3%

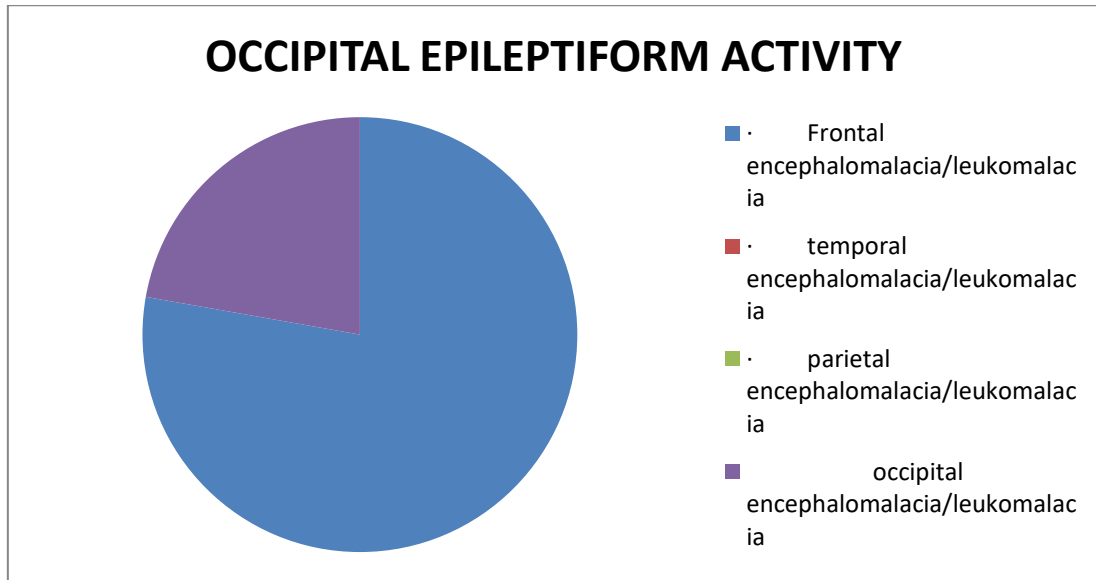
EXPLANATION: Majority of them with occipital lobe epileptiform activity have congenital malformation in frontal(33.3%) , parietal(33.3%) and occipital lobe(33.3%). None of them have in temporal lobe (0%).



OCCIPITAL EPILEPTIFORM ACTIVITY WITH ENCEPHALOMALACIA/LEUKOMALACIA (N=9)

MRI CHANGES	No.	%
Frontal encephalomalacia/leukomalacia	7	77.7%
temporal encephalomalacia/leukomalacia	0	Nil
parietal encephalomalacia/leukomalacia	0	Nil
occipital encephalomalacia/leukomalacia	2	22.2%

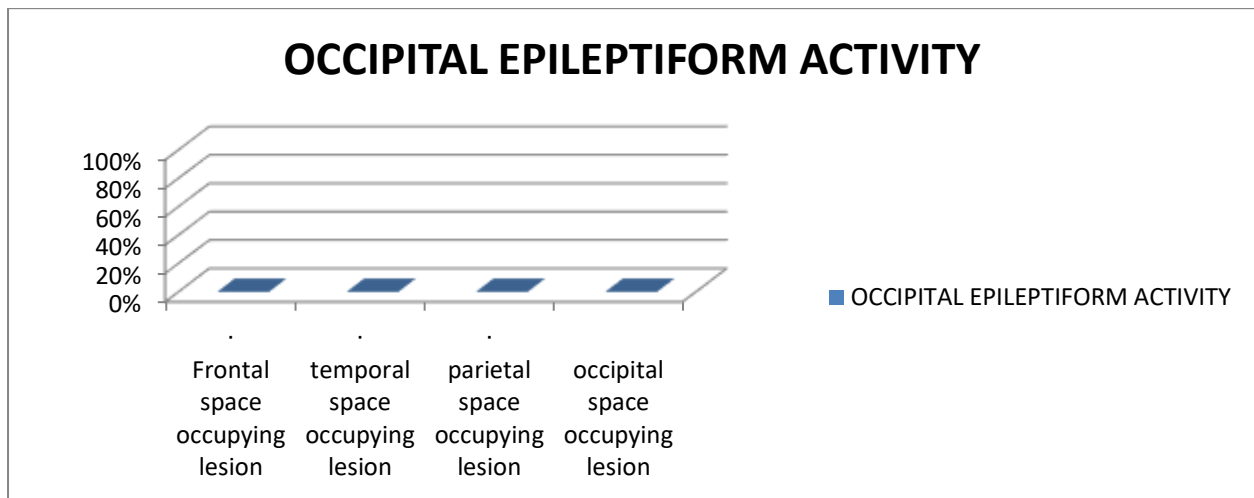
EXPLANATION: Majority of them with occipital lobe epileptiform activity have congenital malformation in frontal lobe (77.7%). None of them have in temporal lobe and parietal lobe (0%). Some of them have in occipital lobe (22.2%).



OCCIPITAL EPILEPTIFORM ACTIVITY WITH SPACE OCCUPYING LESION (N=0)

MRI CHANGES	No.	%
Frontal space occupying lesion	0	Nil
temporal space occupying lesion	0	Nil
parietal space occupying lesion	0	Nil
occipital space occupying lesion	0	Nil

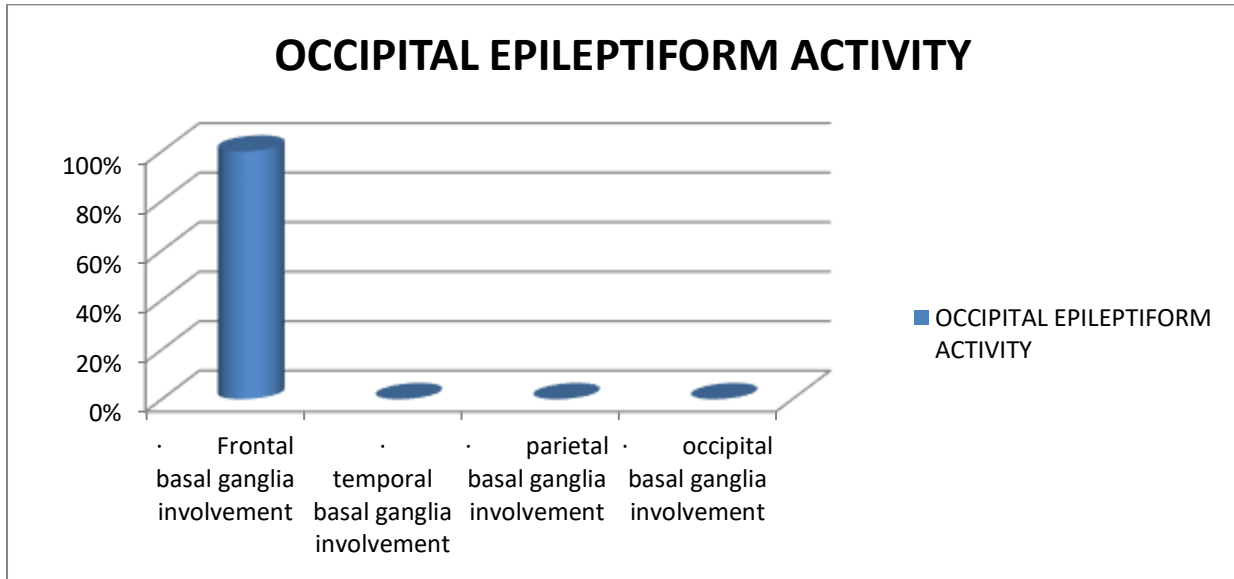
EXPLANATION: None of them with occipital lobe epileptiform activity have space occupying lesion (0%)



OCCIPITAL EPILEPTIFORM ACTIVITY WITH BASAL GANGLIA INVOLVEMENT (N=1)

MRI CHANGES	No.	%
Frontal basal ganglia involvement	1	100%
temporal basal ganglia involvement	0	0%
parietal basal ganglia involvement	0	0%
occipital basal ganglia involvement	0	0%

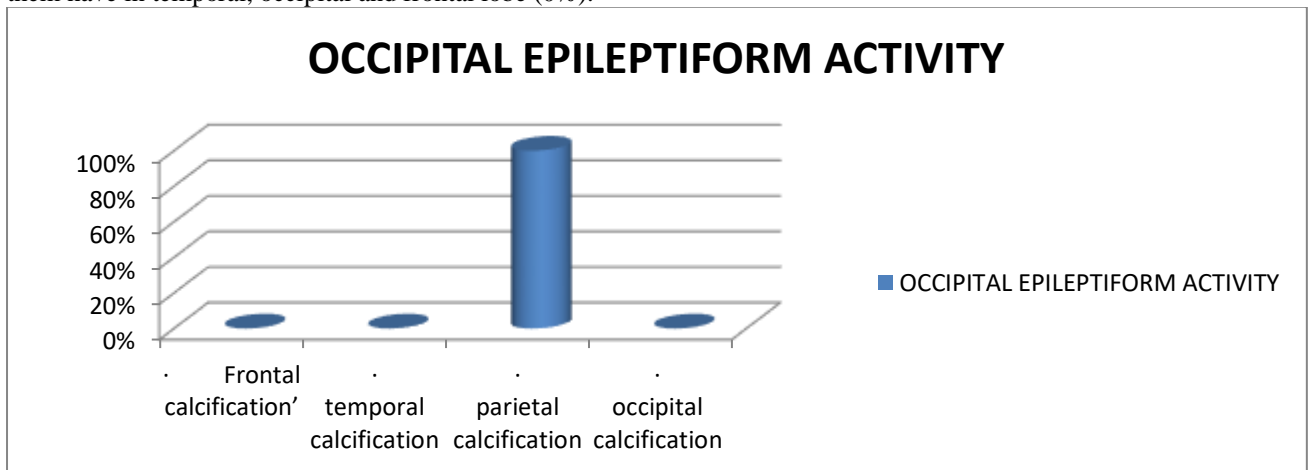
EXPLANATION: All of them with occipital lobe epileptiform activity have basal ganglia involvement in frontal lobe(100%). None of them have in temporal, occipital and parietal lobe (0%).



OCCIPITAL EPILEPTIFORM ACTIVITY WITH CALCIFICATION (N=2)

MRI CHANGES	No.	%
Frontal calcification'	0	Nil
temporal calcification	0	Nil
parietal calcification	2	100%
occipital calcification	0	Nil

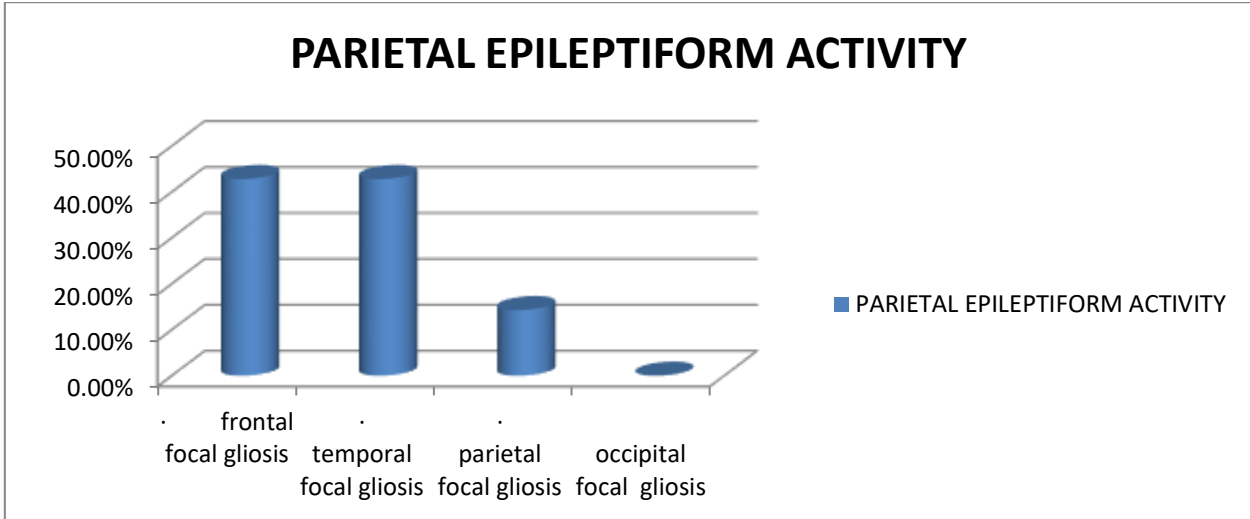
EXPLANATION: All of them with occipital lobe epileptiform activity have calcification in parietal lobe(100%). None of them have in temporal, occipital and frontal lobe (0%).



PARIETAL EPILEPTIFORM ACTIVITY WITH FOCAL GLIOSIS(N=7)

	No.	%
MRI CHANGES		
frontal focal gliosis	3	42.8%
temporal focal gliosis	3	42.8%
parietal focal gliosis	1	14.28%
occipital focal gliosis	0	Nil

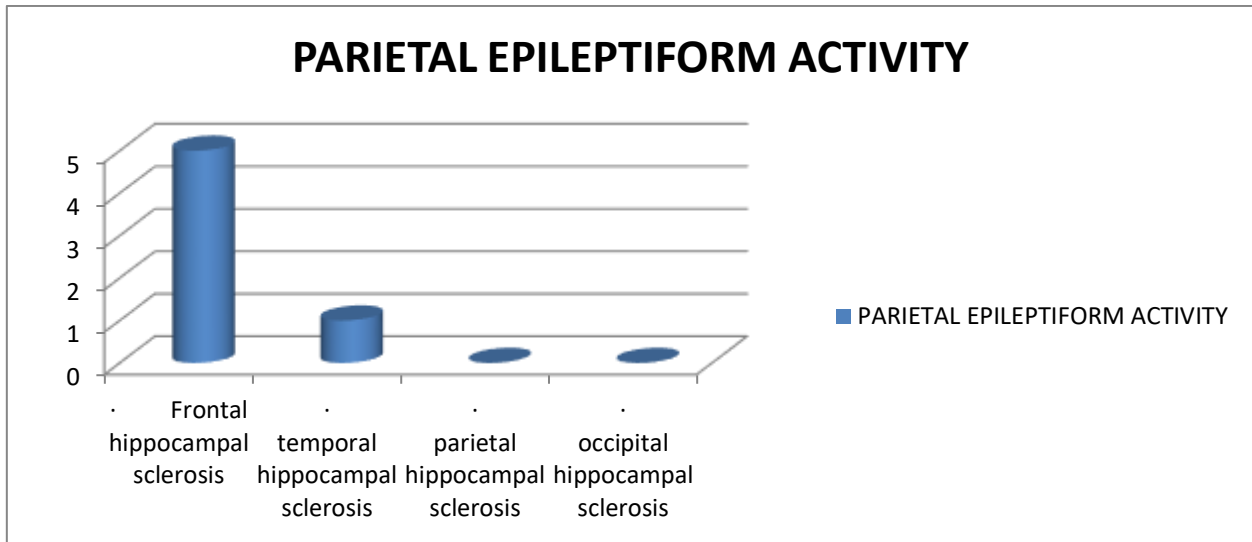
EXPLANATION: Majority of them with parietal lobe epileptiform activity have focal gliosis in frontal(42.8%) and temporal lobe(42.8%). Least number of them have in temporal lobe(42.8%). None of them have in occipital lobe(0%).



PARIETAL EPILEPTIFORM ACTIVITY WITH HIPPOCAMPAL SCLEROSIS(N=1)

	No.	%
MRI CHANGES		
Frontal hippocampal sclerosis	0	Nil
temporal hippocampal sclerosis	1	100%
parietal hippocampal sclerosis	0	Nil
occipital hippocampal sclerosis	0	Nil

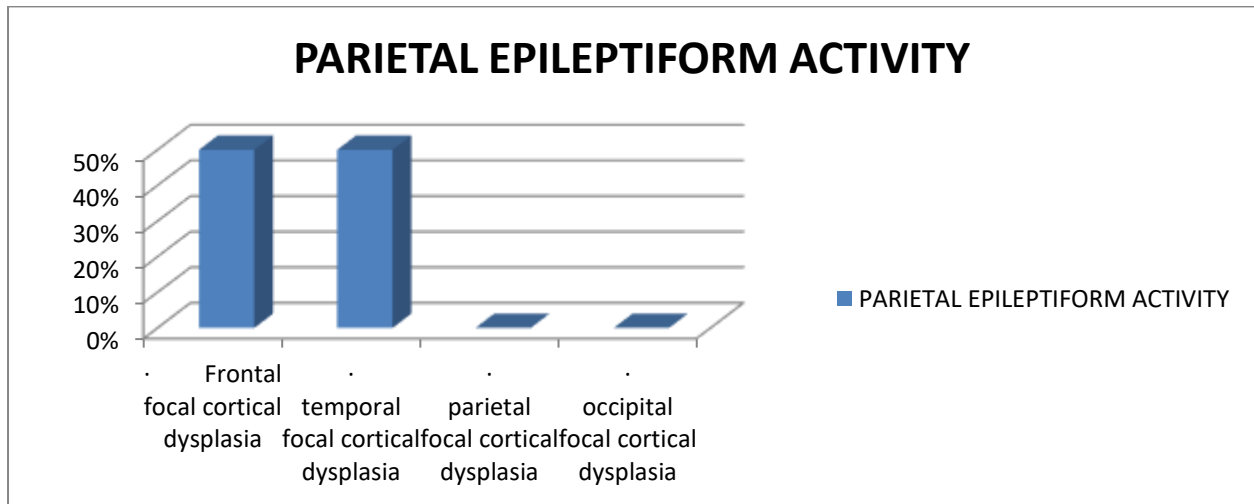
EXPLANATION: All of them with parietal lobe epleptiform activity have hippocampal sclerosis in temporal lobe(100%)
None of them have in frontal, parietal and occipital lobe(0%)



PARIETAL EPILEPTIFORM ACTIVITY WITH FOCAL CORTICAL DYSPLASIA(N=2)

	No.	%
MRI CHANGES		
Frontal focal cortical dysplasia	1	50%
temporal focal cortical dysplasia	1	50%
parietal focal cortical dysplasia	0	nil
occipital focal cortical dysplasia	0	nil

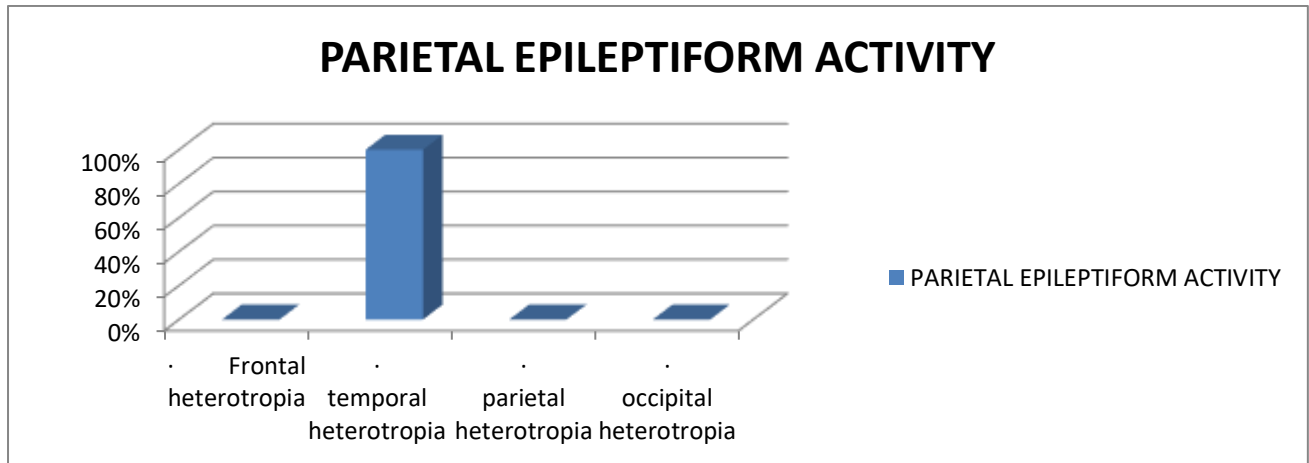
EXPLANATION: Majority of them with parietal lobe epleptiform activity have focal cortical dysplasia in frontal and temporal lobe(50%) None of them have, parietal and occipital lobe(0%)



PARIETAL EPILEPTIFORM ACTIVITY WITH HETEROTROPIA (N=2)

	No.	%
MRI CHANGES		
Frontal heterotropia	0	Nil
temporal heterotropia	2	100%
parietal heterotropia	0	Nil
occipital heterotropia	0	Nil

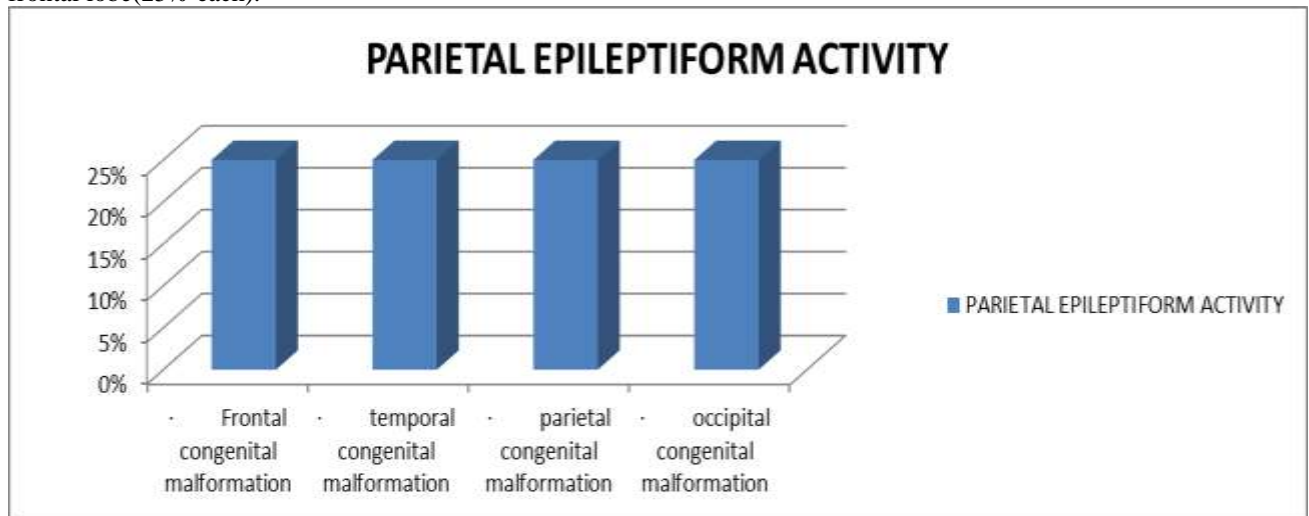
EXPLANATION: All of them with parietal lobe epleptiform activity have heterotropia in temporal lobe(100%).None of them have, frontal, parietal and occipital lobe(0%).



PARIETAL EPILEPTIFORM ACTIVITY WITH CONGENITAL MALFORMATION (N=4)

	No.	%
MRI CHANGES		
Frontal congenital malformation	1	25%
temporal congenital malformation	1	25%
parietal congenital malformation	1	25%
occipital congenital malformation	1	25%

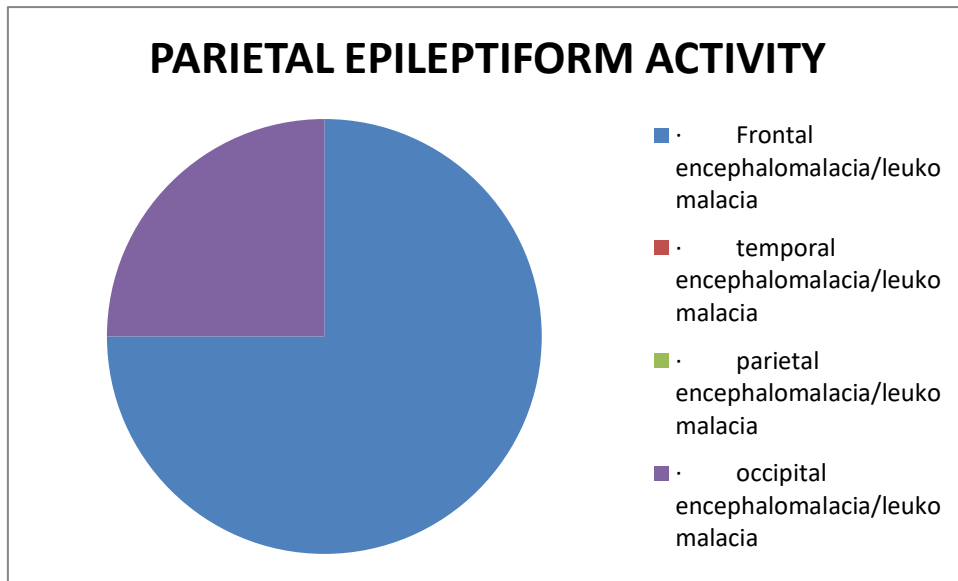
EXPLANATION: Parietal lobe epleptiform activity have congenital malformation changes in frontal, temporal, occipital, frontal lobe(25% each).



PARIETAL EPILEPTIFORM ACTIVITY WITH ENCEPHALOMALACIA/LEUKOMALACIA (N=8)

	No.	%
MRI CHANGES		
Frontal encephalomalacia/leukomalacia	6	75%
temporal encephalomalacia/leukomalacia	0	Nil
parietal encephalomalacia/leukomalacia	0	Nil
occipital encephalomalacia/leukomalacia	2	25%

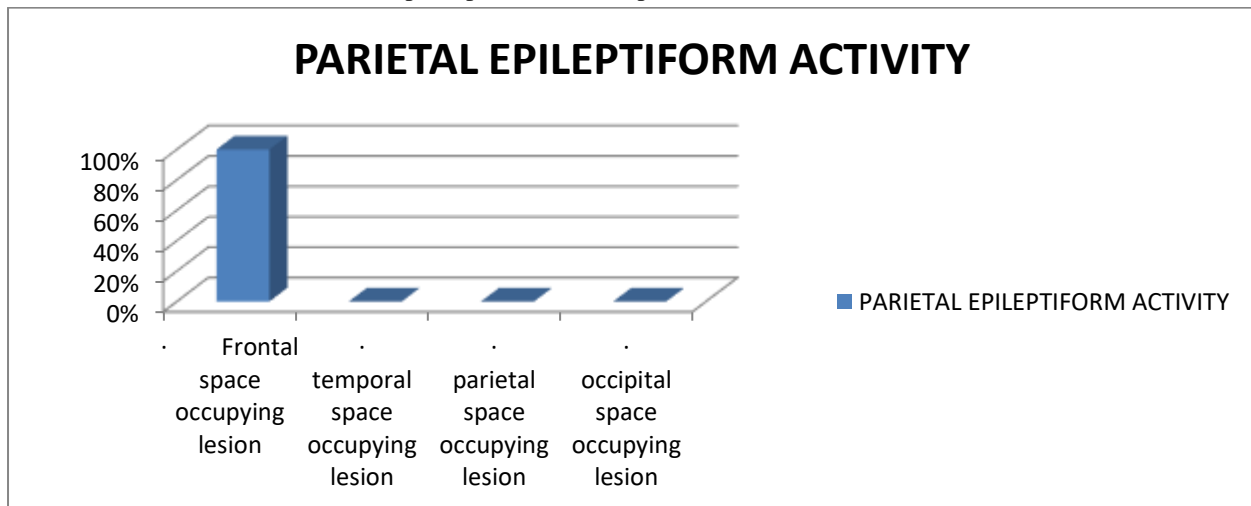
EXPLANATION: All of them with parietal lobe epleptiform activity have encephalomalacia/leukomalacia in frontal lobe(75%).None of them have, parietal and temporal lobe(0%).some of them have in occipital lobe(25%).



PARIETAL EPILEPTIFORM ACTIVITY WITH SPACE OCCUPYING LESION (N=1)

	No.	%
MRI CHANGES		
Frontal space occupying lesion	1	100%
temporal space occupying lesion	0	0%
parietal space occupying lesion	0	0%
occipital space occupying lesion	0	0%

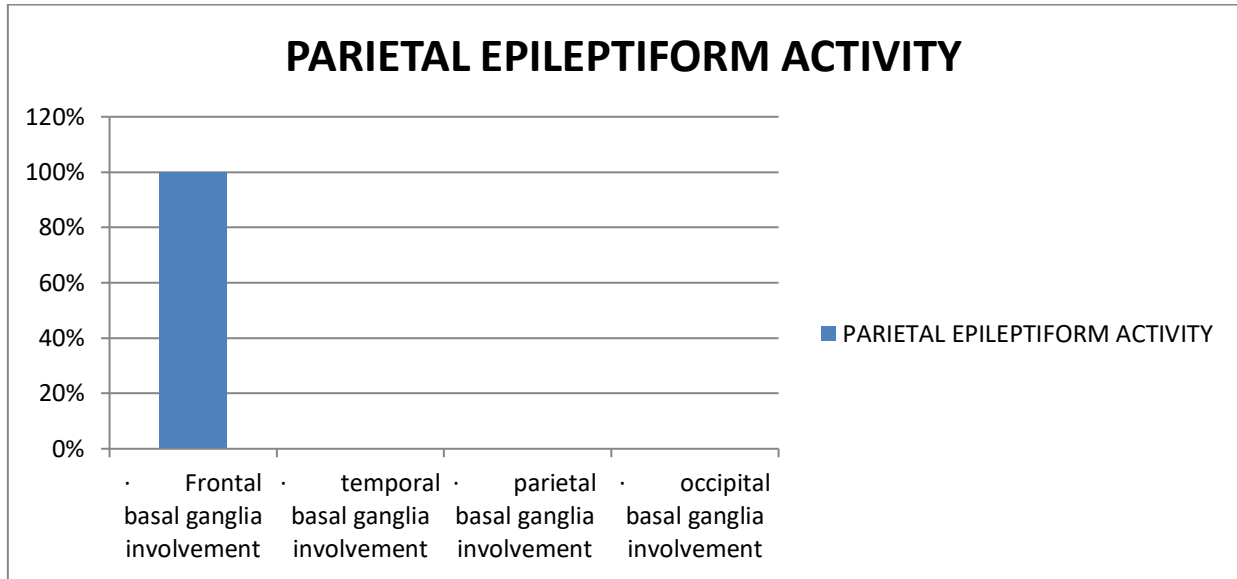
EXPLANATION: All of them with parietal lobe epleptiform activity have space occupying lesion in frontal lobe(100%). None of them have, occipital, parietal and temporal lobe(0%).



PARIETAL EPILEPTIFORM ACTIVITY WITH BASAL GANGLIA INVOLVEMENT (N=1)

	No.	%
MRI CHANGES		
Frontal basal ganglia involvement	1	100%
temporal basal ganglia involvement	0	0%
parietal basal ganglia involvement	0	0%
occipital basal ganglia involvement	0	0%

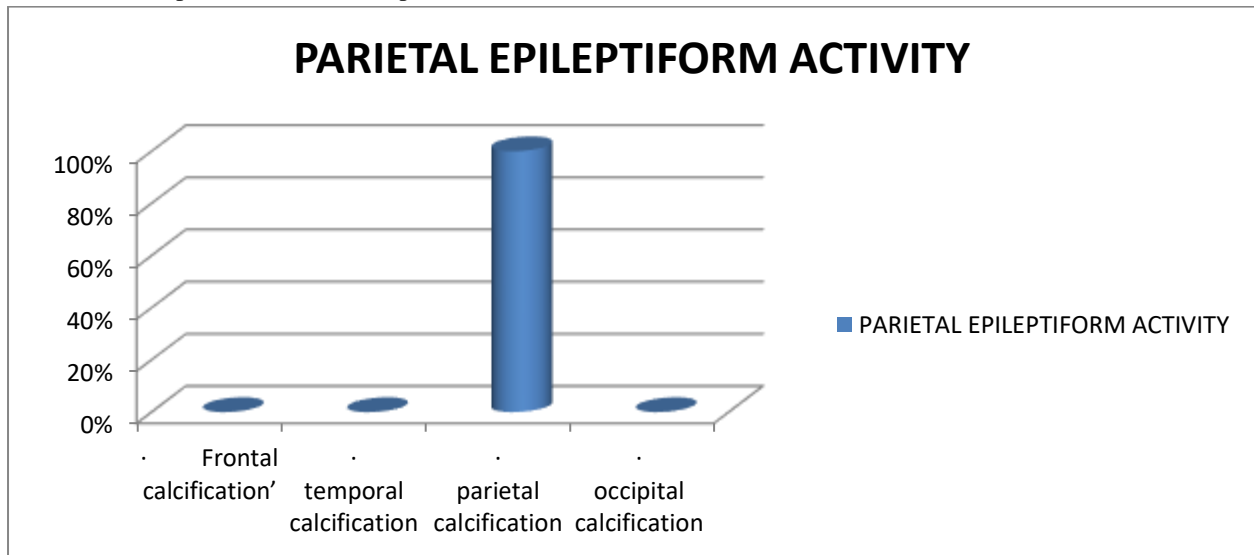
EXPLANATION: All of them with parietal lobe epleptiform activity have basal ganglia involvement in frontal lobe(100%) None of them have, occipital, parietal and temporal lobe(0%).



PARIETAL EPILEPTIFORM ACTIVITY WITH CALCIFICATION (N=2)

MRI CHANGES	No.	%
Frontal calcification	0	Nil
temporal calcification	0	Nil
parietal calcification	2	100%
occipital calcification	0	Nil

EXPLANATION: All of them with parietal lobe epleptiform activity have calcification in parietal lobe(100%).None of them have, occipital, frontal and temporal lobe (0%).



MRI CHANGES	FRONTAL EPILEPTIFOTM ACTIVITY	OCCIPITAL EPILEPTIFORM ACTIVITY	TEMPORAL EPILEPTIFORM ACTIVITY	PARIETAL EPILEPTIFORM ACTIVITY
Normal	8%	16.6%	9%	0%
Focal gliosis+encephalomalacia/ leukomalacia.	28%	29.1%	36.3%	28.5%
Focal cortical dysplasia	20%	8.3%	9%	9.5%
Hippocampal sclerosis	0%	4.1%	9%	4.7%
Congenital malformation	16%	12.5%	13.6%	19%

Focal gliosis	4%	0%	4.5%	4.7%
Calcification	4%	4.1%	0%	4.7%
Heterotropia	4%	4.1%	4.5%	9.5%
Basal ganglia change with calcification.	4%	4.1%	4.5%	4.7%
Encephalomalacia /leukomalacia	4%	8.3%	0%	9.5%
Focal cortical dysplasia+heterotropia	4%	4.1%	0%	0%
Space occupying lesion	4%	0%	4.5%	4.7%
Focal gliosis + encephalomalacia+heterotropia	0%	4.1%	0%	0%

MRI CHANGES	FOCAL SLOW WAVE	FOCAL SPIKE WAVE	FOCAL SPIKE AND POLYSPIKE	RHYTHMIC DELTA ACTIVITY
Normal	31.5%	15.1%	12.5%	16.6%
Focal gliosis+encephalomalacia/leukomalacia.	10.5%	24.2%	25%	16.6%
Focal cortical dysplasia	10.5%	12.1%	12.5%	0%
Hippocampal sclerosis	5.2%	6%	6.25%	16.6%
Congenital malformation	5.2%	15.1%	18.7%	0%
Focal gliosis	0%	3%	6.25%	16.6%
Calcification	0%	3%	6.25%	0%
Heterotropia	5.2%	6%	0%	0%
Basal ganglia change with calcification.	5.2%	3%	0%	0%
Encephalomalacia /leukomalacia	5.2%	6%	6.25%	33.3%
Focal cortical dysplasia+ heterotropia	0%	3%	0%	0%
Space occupying lesion	0%	0%	6.25%	0%
Focal gliosis + encephalomalacia+heterotropia				

EEG WAVEFORM	FRONTAL EPILEPTIFORM ACTIVITY	OCCIPITAL EPILEPTIFORM ACTIVITY	TEMPORAL EPILEPTIFORM ACTIVITY	PARIETAL EPILEPTIFORM ACTIVITY
Focal spike wave	92%	8%	8%	8%
Focal spike and polyspike.	28%	28%	28%	28%
Focal slow wave	36%	20%	20%	20%
Rhythmic delta activity	12%	0%	0%	0%
Other discharges	8%	16%	16%	16%

MRI CHANGES	FRONTAL EPILEPTIFORM ACTIVITY	OCCIPITAL EPILEPTIFORM ACTIVITY	TEMPORAL EPILEPTIFORM ACTIVITY	PARIETAL EPILEPTIFORM ACTIVITY
• Focal cortical dysplasia	80%	66.6%	100%	50%
1)frontal	20%	33.3%	0%	50%
2)temporal	0%	0%	0%	0%
3)parietal	0%	0%	0%	0%
4)occipital				
• Hippocampal sclerosis	0%	0%	0%	0%
1)frontal	0%	100%	100%	100%
2)temporal	0%	0%	0%	0%
3)parietal				
4)occipital				

• Congenital malformation	75%	33.3%	100%	25%
1)frontal	0%	0%	0%	25%
2)temporal	25%	33.3%	0%	25%
3)parietal	0%	33.3%	0%	25%
4)occipital				
• Focal gliosis				
1)frontal	75%	50%	44.4%	42.8%
2)temporal	12.5%	25%	33.3%	42.8%
3)parietal	0%	12.5%	11.1%	14.28%
4)occipital	12.5%	12.5%	11.15	0%
• Calcification				
1)frontal	0%	0%	0%	0%
2)temporal	0%	0%	0%	0%
3)parietal	100%	100%	100%	100%
4)occipital	0%	0%	0%	0%
• Heterotropia				
1)frontal	0%	33.3%	0%	0%
2)temporal	100%	66.6%	100%	100%
3)parietal	0%	0%	0%	0%
4)occipital	0%	0%	0%	0%
• Basal ganglia change	100%	100%	100%	100%
1)frontal	0%	0%	0%	0%
2)temporal	0%	0%	0%	0%
3)parietal	0%	0%	0%	0%
4)occipital				
• Encephalomalacia /leukomalacia	62.5	77.7%	75%	75%
1)frontal	0%	0%	0%	0%
2)temporal	37.5%	22.2%	25%	25%
3)parietal				
4)occipital		0%	100%	100%
• Space occupying lesion	100%	0%	0%	0%
1)frontal	0%	0%	0%	0%
2)temporal	0%			
3)parietal				
4)occipital				

RESULTS

Table 2: 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 3:
Correlation of MRI and EEG findings (n=50)

		EEG				Total	
		Normal		Abnormal			
		No.	%	No.	%	No.	%
MRI	Normal	5	10%	7	14.0%	12	24.0%
	Abnormal	5	10.0%	33	66.0%	38	76.0%
MRI	Total	10	20.0%	40	80%	50	100.0%

Fischer’s test: $\chi^2=7.214$, 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%).

Figure 2: MRI and EEG findings in patients (% , n=50)

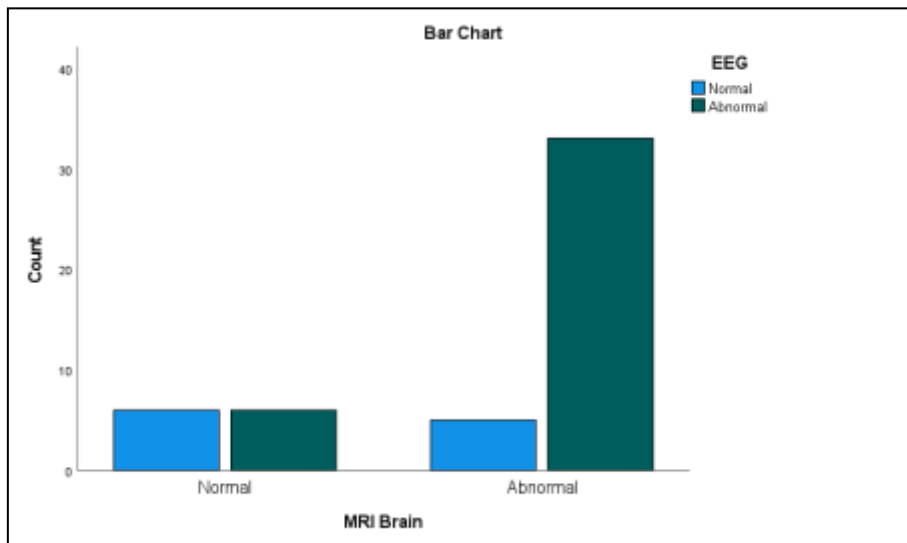


Table 4: EEG and MRI abnormalities in focal seizure (n=50)

		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%

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 rhythmic 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%).

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

Table 5: 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	%
Risk factors	Hypothyroidism	1	10%	5	5	12.5%	6	6	15.7%	0	0%
	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	80.0%	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%

		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	%
	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%
	Heterotopia	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%
	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%

	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	%
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.

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. Although MRI is a noninvasive form of imaging and very sensitive, it is not readily available or expensive. Because of its greater resolution and lack of radiation exposure to patients, MRI has been favored over computed tomography. 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, heterotopia, 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.

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 machine. 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.

REFERENCES

1. Berg AT, et al. Revised terminology and concepts for organization of seizures and epilepsies: Report of the ILAE Commission on Classification and Terminology, 2005–2009. *Epilepsia*. 2010;51(4):676–685.
2. Wirrell EC. Predicting pharmacoresistance in pediatric epilepsy. *Epilepsia*. 2013;54(Suppl 2):19–22.
3. Gaillard WD, Chiron C, Cross JH, et al. Guidelines for imaging infants and children with recent-onset epilepsy. *Epilepsia*. 2009;50(9):2147–2153.
4. Guerrini R. Epilepsy in children. *Lancet*. 2006;367(9509):499–524.
5. Radhakrishnan K, et al. Neurocysticercosis as an important cause of epilepsy in India: A community-based study. *Lancet*. 2000;355(9224):1180–1185.
6. Holmes GL, Lenck-Santini PP. *Role of interictal epileptiform abnormalities in cognitive impairment*. *Epilepsy Behav*. 2006;8(3):504–515.
7. Panayiotopoulos CP. *Benign childhood partial epilepsies: benign childhood seizure susceptibility syndromes*. *J Neurol Neurosurg Psychiatry*. 1993;56(1):2–5.
8. Loddenkemper T, Holland KD, Stanford LD, et al. *Developmental outcome in children with early-onset epilepsy: Importance of etiologies and seizure burden*. *Epilepsy Res*. 2017;136:1–8.
9. Wyllie E, et al. *Surgical treatment of epilepsy in children*. *Epilepsia*. 1998;39(Suppl 4):S17–S25.
10. Siniatchkin M, et al. *EEG-fMRI in children with focal epilepsy*. *Epilepsia*. 2010;51(6):1272–1278.
11. Kneen R, Solomon T, Appleton R. *The role of neuroimaging in the evaluation of children with epilepsy*. *J Neurol Neurosurg Psychiatry*. 2002;73(1):13–20.
12. Berg AT, Shinnar S. *The risk of seizure recurrence following a first unprovoked seizure: a quantitative review*. *Neurology*. 1991;41(7):965–972.
13. Iman S. (2025). Determine the impressed of Companies' financial performance of customer orientation, competitor orientation and inter-functional coordination with the role of mediator of innovation and corporate social responsibility (Case Study: Eshtehard Industrial Town). In *Global Journal of Research in Business Management* (Vol. 5, Number 2, pp. 111–121). <https://doi.org/10.5281/zenodo.15170159>
14. Cross JH, Jayakar P, Nordli D, et al. *Prognosis of epilepsy in children after surgery for focal cortical dysplasia: a retrospective multicenter study*. *Lancet Neurol*. 2013;12(6):525–533.
15. Shinnar S, Pellock JM. *Update on the epidemiology and prognosis of pediatric epilepsy*. *J Child Neurol*. 2002;17(Suppl 1):S4–S17.
16. Hassan T, Mohamed S, Basha A, et al. *MRI findings in children with epilepsy: A hospital-based study in Upper Egypt*. *Egypt J Radiol Nucl Med*. 2015;46(3):707–714.
17. Keller SS, Roberts N. *Voxel-based morphometry of temporal lobe epilepsy: an introduction and review of the literature*. *Epilepsia*. 2008;49(5):741–757.
18. Sankar R, Knowles WD. *Pharmacoresistant epilepsy in children*. *J Child Neurol*. 1999;14(Suppl 1):S35–S42.
19. Sisodiya SM. *Structural imaging and genetics in epilepsy*. *Epilepsia*. 2005;46(Suppl 10):23–24.

20. Koutroumanidis M, Panayiotopoulos CP. *Benign childhood focal seizures and related epileptic syndromes*. In: Panayiotopoulos CP. *A Clinical Guide to Epileptic Syndromes and their Treatment*. Springer; 2007.
21. Camfield PR, Camfield CS. *Idiopathic epilepsy in children: A population-based study of clinical features, outcome and school behavior*. *Can J Neurol Sci*. 1996;23(4):279–282.
22. Glauser TA, Ben-Menachem E, Bourgeois BFD, et al. *Updated ILAE evidence review of antiepileptic drug efficacy and effectiveness as initial monotherapy for epileptic seizures and syndromes*. *Epilepsia*. 2013;54(3):551–563.
23. Zijlmans M, Zweiphenning W, van Klink N. *Changing concepts in presurgical assessment for epilepsy surgery*. *Nat Rev Neurol*. 2019;15(10):594–606.
24. Loddenkemper T, Pressler RM. Management of epilepsy in childhood. *Nat Rev Neurol*. 2012;8(7):402–412.
25. Dube C, Yu H, Nalbantoglu J, et al. Neurodevelopmental outcome in children with intractable epilepsy and focal cortical dysplasia. *Epilepsy Behav*. 2006;9(4):619–627.
26. Arzimanoglou A, Resnick T, Hirsch E, et al. Focal epilepsies in children and adolescents: a guide to diagnosis and treatment. *Epileptic Disord*. 2020;22(6):833–856.
27. Maytal J, Krauss JM, Novak G, et al. The value of brain MRI in children with epilepsy and normal neurologic examination. *Neurology*. 1995;45(5):960–964.
28. O'Callaghan FJK, Lux AL, Darke K, et al. The effect of epilepsy on cognitive function in children with tuberous sclerosis complex. *Dev Med Child Neurol*. 2004;46(11):698–703.
29. Suhonen-Polvi H, Valanne L, Ketonen L, et al. MRI findings in children with newly diagnosed epilepsy. *Neuroradiology*. 1998;40(5):275–279.
30. Wang ZI, Alexopoulos AV, Jones SE, et al. The pathology of magnetic-resonance-imaging-negative epilepsy. *Mod Pathol*. 2013;26(8):1051–1058.
31. Pohlmann-Eden B, Beghi E, Camfield C, et al. The first seizure and its management in adults and children. *BMJ*. 2006;332(7537):339–342.
32. van der Kolk A, Braun KPJ, van Iterson L, et al. Early and late cognitive outcome after pediatric epilepsy surgery: A prospective longitudinal study. *Epilepsia*. 2013;54(3):e72–e75.

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