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Original Research Article

Role of MRI in Evaluation of Focal Epilepsy

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Abstract:

Background: In this study, we aimed to characterise the CNS lesion and aid in planning as to whether medical or surgical modalities were necessary, aid in the diagnostic challenge in pharmacologically refractory seizure cases such as epilepsy syndromes and temporal lobe epilepsy, and to ascertain the varied role of neuroimaging in seizure disorder.

Methods: This was a hospital-based cross sectional observational study, conducted among 88 patients with clinical presentation of focal seizure disorder, at the Department of Neuromedicine, including the epilepsy clinic of Bangur Institute of Neurosciences and the Department of Radiology, I.P.G.M.E. and R. and S.S.K.M. Hospital, from February 2012 to July 2013, after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

Results: The presenting seizure pattern was classified into two groups: simple partial seizures and complex partial seizures (n=88). Co-relation of a history of febrile convulsions with mesial-temporal sclerosis (n=23). The various causes of refractory epilepsy were subdivided and classified. The patients were observed for premonitory symptoms or auras and noted down (n = 88). Comparison of coronal FLAIR with an axial or coronal T2 weighted sequence for finding hippocampal hyperintensity, also called hippocampal sclerosis (n = 23). Based on MRI features, the causes were determined. Mesial temporal sclerosis (26.1%), space-occupying lesions (19.3), and patients with normal MRI appearance (22.7%) were the three most frequent causes.

Conclusion: MRI is an effective tool for detecting pathologies causing focal or partial epilepsy, except in some cases of non-lesional focal epilepsy. Coronal FLAIR images are superior to axial or coronal T2-weighted images for the detection of hippocampal sclerosis. There will be future scope for functional MRI, MRI and PET fusion or MRI and interictal SPECT fusion in cases of non-lesional MRI-negative focal epilepsy.

Keywords: MRI, Evaluation, Focal Epilepsy.

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Introduction

A seizure is a paroxysmal occurrence caused by aberrant, excessive, hypersynchronous discharges from a group of central nervous system (CNS) neurons (from the Latin word sacire, "to take possession of").[1] The term epilepsy is defined as two or more unprovoked seizures occurring at intervals of 24 hours or more apert. [2] Epileptic seizures can be defined as intermittent and stereotyped disturbances of consciousness, behaviour, emotion, motor function or sensation that are clinically believed to result from cortical neural discharge.[3] The early clinical and EEG alterations in focal (synonym-partial) seizures typically show the first activation of a network of neurons that is only present in a portion of one brain hemisphere. Seizures that originate from an epileptogenic centre located anywhere in the brain are referred to as focal (anatomical, topographical, or localization related) epilepsies. Ictal symptoms are not caused by the aetiology but rather by localization, especially at the start. The assignment of some epilepsies to particular anatomical localizations or lobes, however, is challenging, as is frequently the case with seizures coming from clinically quiet epileptogenic locations.[4] Epilepsy: According to the International League

Against Epilepsies (ILAE) epilepsy is a condition characterised by recurrent (two or more) epileptic seizures, unprovoked by any immediate identified cause.[4]

The most widely used classification of epileptic seizures is the International League Against Epilepsy (ILAE) classification[5], which is principally based on the clinical seizure type and interictal electroencephalography (EEG) findings.

Using the definition of epilepsy as two or more unprovoked seizures, the incidence of epilepsy is 0.3-0.5% in different populations throughout the world, where the prevalence of epilepsy is in the range of 4-10 per 1000. Incidence and prevalence are higher in 3rd world countries than in the developed world, with higher rates found in rural as opposed to urban communities, with slightly higher prevalence in men than women, with 2/3 being partial epilepsy and approximately 1/3 being generalized.

These epilepsies might be symptomatic, idiopathic, or cryptogenic. The new diagnostic method distinguishes between "symptomatic (or likely symptomatic) focal epilepsies" and "idiopathic focal epilepsies." There is now solid proof that some focal epilepsies should be accepted, diagnosed, and treated based on aetiology rather than just localization. One of the more prevalent and distinctive epileptic syndromes, mesial temporal lobe epilepsy (MTLE), with hippocampal sclerosis, is a prominent illustration of this. The new ILAE diagnostic system further divides focal epilepsies into limbic and neocortical subtypes.

The effects of epilepsy on people and society are severe. Finding a specific structural brain abnormality in individuals with partial complex epilepsy gives the best chance for a surgical cure and a rise in quality of life. Surgery is not only the sole remaining choice for treating individuals with medically resistant focal epilepsy, but it also provides the highest opportunity for a long-term cure and is the most economical strategy overall. Because it allows for precise epileptogenic focus identification, which is essential for preoperative planning and localization, MRI is essential to surgical success.

MRI plays a key role in diagnosis and localization. [6] Over the past decade, the role of neuroimaging in the diagnosis and management of epilepsy has changed considerably. MRI is more sensitive in diagnosing most of the cerebral pathologies associated with chronic epilepsy, with the exception of calcification, which is better seen in CT. A complete blood count (CBC), blood chemistry, liver and thyroid function tests, EEG, and most importantly, a brain imaging scan, ideally an MRI, should all be routinely included in the first diagnostic workup of seizures. In an emergency or for treating extremely young children, CT scanning may be the only practical test.[7]

MRI is much more sensitive than CT for early detection of the causative lesion. Mesial temporal sclerosis, low grade neoplasia, vascular lesions, particularly cavernomas and developmental abnormalities particularly abnormalities of cortical neural migration are all likely to be missed by CT but can be easily detected on MRI. For the early identification of causal lesions, MRI is more sensitive than CT. The majority of patients who experience new-onset seizures should get a brain imaging examination to see if a structural defect may be to blame for the seizure disease. The sensitivity for detecting abnormal cortical architecture, including hippocampal atrophy, as well as abnormalities of cortical migration and infectious aetiologies has increased with the use of newer MRI techniques like FLAIR (Fluid Attenuated Inversion Recovery), MRS (Magnetic Resonance Spectroscopy), and DWI (Diffusion Weighted Images). MRI is indicated in all patients with epilepsy who appear refractory to pharmacological treatment, irrespective of previous imaging studies. MRI sequences will usually include a T1-weighted thin slice scan, often with hippocampal volumetry, a T2-weighted coronal fluid attenuated inversion recovery (FLAIR), and gradient echo sequences (GRE). In patients with suspected CNS infections, CT will be performed on an emergency basis when MRI is not available. It is usually appropriate to obtain an MRI within a few days of the initial evaluation. Neoplasia overall contributes 4% of cases of seizure disorder; the rest of the cases are non-neoplastic in the 1–20 yr. age group.[8]

Aims and Objectives

1. To determine if a CNS lesion in a patient having a focal seizure matches the seizure semiology.
2. To describe the lesion and assist in determining if medicinal or surgical treatments are necessary.
3. To assist in the diagnostic difficulty of seizure cases that are unresponsive to medication, such as epileptic syndromes and temporal lobe epilepsy.
4. To identify the many functions of neuroimaging in epilepsy.

Materials & Methods

This was a hospital-based cross sectional observational study, conducted among 88 patients with clinical presentation of focal seizure disorder, at the Department of Neuromedicine, including the epilepsy clinic of Bangur Institute of Neurosciences and the Department of Radiology, I.P.G.M.E. and R. and S.S.K.M. Hospital, from February 2012 to July 2013, after obtaining clearance from the

institutional ethics committee and written informed consent from the study participants.

Inclusion Criteria

Patient with a clinical diagnosis of partial (focal) Epilepsy.

Exclusion Criteria

Patient with partial epilepsy due to an acute symptomatic cause

Statistical Methods

All the relevant data regarding clinical features, history obtained and MRI findings were recorded in a standard, uniform format on a record sheet for each patient.

Results

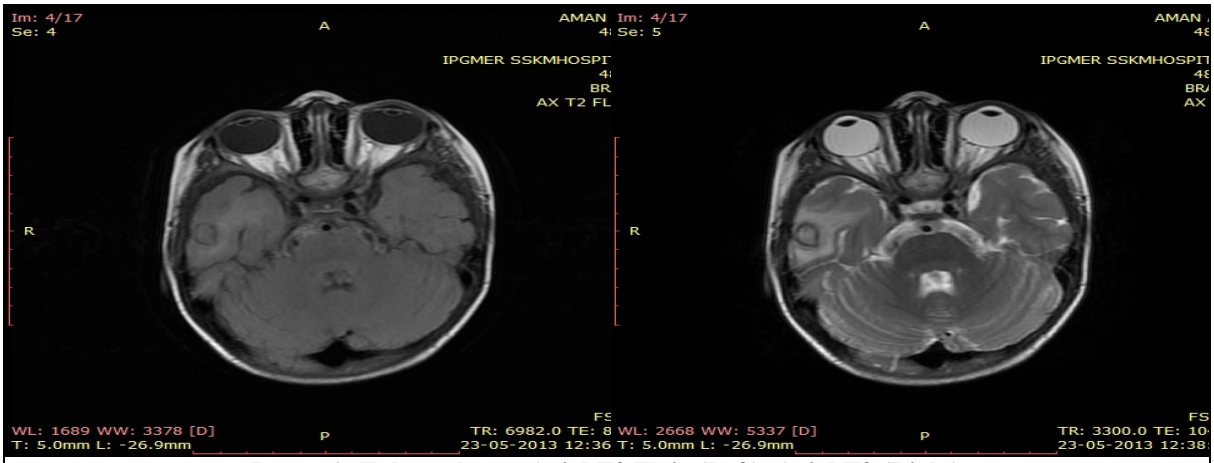

Image 1: Tuberculoma- Axial T2 Flair (Left), Axial T2 (Right)

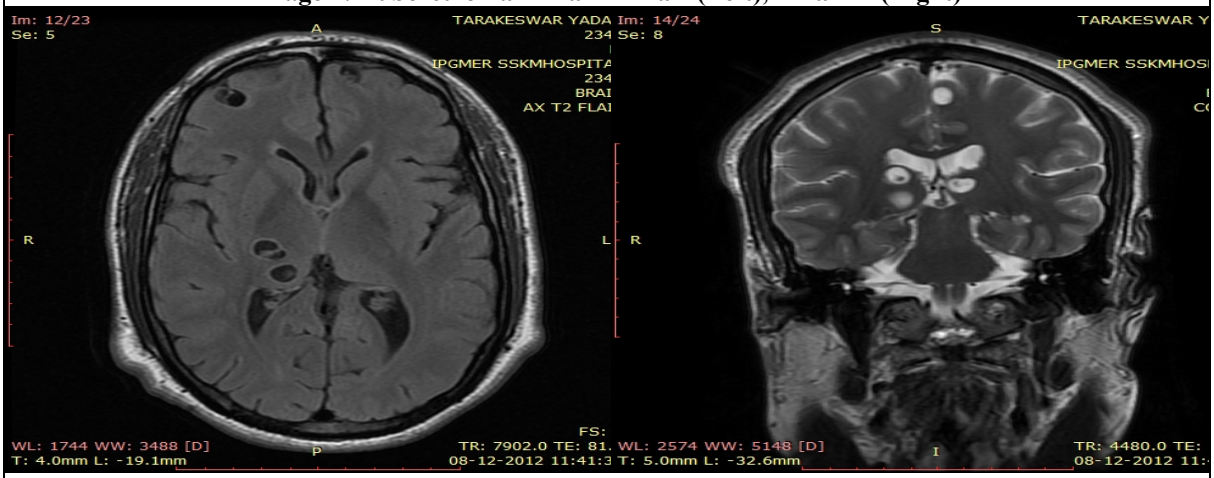

Image 2: NCC- Axial T2 Flair (Left), Coronal T2 (Right)

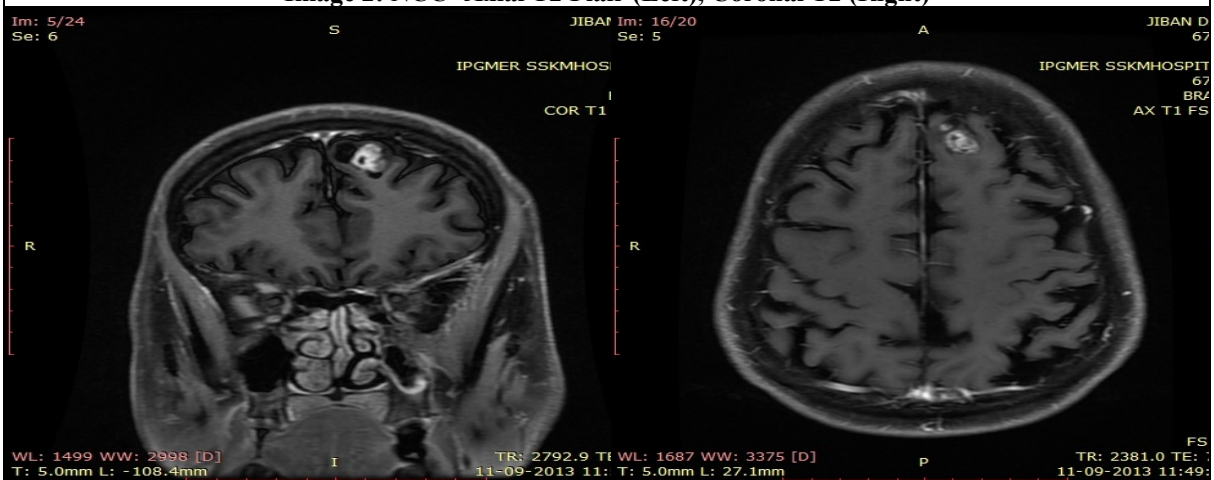

Image 3: Oligodendroglioma—Coronal T1 FS+ C (Left), Axial T1 FS+C (Right)

A total of 88 patients with clinical features of focal epilepsy were included in the study. Age group: The age group of patients included in our study ranges from 7 to 62 years. The mean age was years. The patients were grouped into male and female groups (n=88).

Table 1: History of Febrile Convulsion

Pattern of Seizure	**No. of Patients**	**Percentage**
Simple Partial	26	29.5
Complex Partial	62	70.5
Seizure Pattern		
History of Febrile Convulsion	**No. of Patients**	**Percentage**
Present	24	27.2
Absent	64	72.8

The presenting seizure pattern was classified into two groups: simple partial seizures and complex partial seizures (n=88). The patients were categorised (n=88) based on their history of febrile convulsions.

Table 2: Treatment Response Pattern

Mesial Temporal sclerosis		
History of Febrile Convulsion	**No. of Patients**	**Percentage**
Present	16	69.5
Absent	7	30.5
History of Febrile Convulsion		
Treatment	No. of Patients	Percentage
Well responding	64	72.7
Refractory	24	27.3

Co-relation of a history of febrile convulsions with mesial temporal sclerosis (n=23). In this study, we divided patients into two groups: (1) Treatment well responding and (2) refractory epilepsy (n=23)

Table 3: Association with Aura

Causes	**No. of Patients**	**Percentage**
Mesial Temporal Sclerosis	9	37.5
Glioma	4	16.6
Oligodendroglioma	2	8.3
Schizencephaly	2	8.3
Heterotopia	5	20.8
Post traumatic Gliosis	2	8.3
Causes of Refractory Epilepsy (n=24)		
Aura	**No. of Patients**	**Percentage**
Present	56	63.6
Absent	32	36.4

The various causes of refractory epilepsy were subdivided and classified. The patients were observed for premonitory symptoms or auras and noted down (n=88).

Table 4: Causes of Focal Epilepsy

	Coronal FLAIR		**Axial/Coronal T2**	
Hippocampal Sclerosis	**No. of Patients**	**Percentage**	**No of Patients**	**Percentage**
23	21	91.3	13	56.5
Comparison between Coronal Flair and Axial/Cor T2				
Cause	**No. of Patients**		**Percentage**	
Mesial Temporal Sclerosis	23		26.1	
Space Occupying Lesion	17		19.3	
Normal	20		22.7	
Demyelination/Gliosis/Traumatic	8		9	
Infection	12		13.6	
Congenital	8		9	

Comparison of coronal FLAIR with axial or coronal T2 weighted sequence regarding the detection of hippocampal hyperintensity known as hippocampal sclerosis (n=23).

Based on MRI features, the causes were determined and classified (n=88). The most common cause was mesial temporal sclerosis (26.1%), followed by space occupying lesions (19.3) and patients with normal MRI appearance (22.7%).

Discussion

Epilepsy is a major health problem throughout the world, leading to a significant amount of physical, psychological, and social morbidity. Although a variety of factors influence the incidence and prevalence of seizures, 5-10% of the population will have at least one seizure, with the highest incidence occurring in childhood and early adulthood. Using the definition of epilepsy as two more unprovoked seizures, the incidence of epilepsy is, 0.3-0.5% in different populations throughout the world, and the prevalence of epilepsy has been estimated at 5-10 persons per 1000.[1] The cumulative lifetime incidence of epilepsy is 3% in children. However, the annual prevalence of epilepsy is lower (0.5-0.8%) because many children outgrow epilepsy.[2] Although the outlook for most children with symptomatic seizures is generally good, seizures may signal a potentially serious underlying systemic or central nervous system disorder that requires thorough investigation and management. For children with epilepsy the prognosis is generally good, but 20% of patients have persistent seizures refractory to drugs and those causes pose a diagnostic and management challenge.[5]

Localization-related epilepsy, or focal epilepsy, has typical clinical and EEG characteristics. The most frequent forms of complex partial seizures in the adult population are those coming from the temporal lobe. The majority of patients who are referred for surgery have partial seizures with temporal lobe complications.[9]

Patients with clinical features and symptoms of Focal epilepsy diagnosed in the epilepsy clinic and Neurology department of the Bangur Institute of Neuroscience and Psychiatry were evaluated in the department of Radiology by MRI (Magnetic Resonance Imaging). 88 patients were evaluated in this study to detect the aetiology of their seizure disorder. The age group of patients in our study was 7-62 years. The mean age was 22.54+8.2 years. Focal epilepsy is commonly a childhood onset disorder and is mostly diagnosed in adolescence.

A study by Cohn–Gadol Aaron A., Bradley Christopher C., Williamson C. regarding normal magnetic resonance imaging and medial temporal lobe epilepsy, the clinical syndrome of paradoxical temporal lobe epilepsy has shown a mean age of 32 years + 11 years (mean standard deviation). An article published by Blaise F. D. Bourgeois regarding Temporal Lobe epilepsy in infants and children has shown the mean age at presentation is 10 years. [10]

In another study by E. Tasch, F. Cendes, L. M. Li, and Fi Anderman regarding Neuroimaging evidence of progressive neuronal loss and dysfunction in Temporal lobe epilepsy, the mean age was 28 years + 11 years (mean+- standard deviation). The chief clinical association with temporal epilepsy is aura. In our study, 63.6% of patients presented with a history of aura. Auras are present in 80% of patients with temporal lobe epilepsy. They are usually features of simple partial seizures that may precede complex partial seizures. Acharya et al. have shown that olfactory auras are associated more commonly with temporal lobe tumours, while auditory auras are common in neocortical temporal lobe epilepsy.[11]

The male –female ratio in our study is 7:4; which suggest male preponderance of this disorder.

A study named Temporal Lobe Epilepsy 1948–1986 by Christopher Ounsted, Janet Lindsay, and Perowile Richards has shown a sex ratio of 63 males to 37 females, which means two-thirds of the patients were male. But a study by Fatma Mujgan Aynaci et al. has shown no significant difference between sex groups among children.[12]

In our study, we observed that there were different etiopathologies of focal epilepsy, which were subdivided into the following groups: mesial temporal sclerosis, congenital causes, space-occupying lesions, demyelination, infarction, or post traumatic gliosis, inflammatory/infections and no significant abnormality detected.

S. Lehericy, F. Semah, D. Hasboun, D. Dornont, O. Grant, C. Marsault, and M. Baulac performed an MRI study on 222 patients with temporal lobe epilepsy: with varying severity in the years 1991–1993. Temporal lobe abnormalities were found in 180 (81%) patients, followed by hippocampal sclerosis in 122 (55%), developmental abnormalities in 16 patients (7.2%), and cavernous angiomas in 10 (4.5%). MRI was normal or showed unrelated changes in 42 patients (19%). Visual assessments have correctly localised Hippocampal sclerosis in 79 out of 84 patients (94%). Patients with normal MRIs had an older age of seizure presentation and were often more drug responsive. MRI normal patients have less severe forms of disease.

The patient with mesial temporal sclerosis diagnosed in our study has shown the following features.

1. Hippocampal hyper intensity in T2 and coronal FLAIR.
2. Ipsilateral volume loss of the hippocampus in T2 and coronal FLAIR.
3. Ipsilateral dilatation of the temporal horn.

A study by Fatma Mujgan et al. regarding clinical, electrophysiological, and neuropsychological findings of twenty-two children with mesial temporal sclerosis has shown (1) hippocampal atrophy; (2) increased signal intensity in T2

weighted MRI. (3) dilatation of the ipsilateral temporal horn as the gold standard diagnostic criteria.

A study by L.C. Meniers et al. regarding temporal lobe epilepsy and various M.R. appearances in histologically proven mesial temporal lobe sclerosis has shown six MRI features regarding mesial temporal sclerosis:

1. High signal intensity in the hippocampus
2. Reduced hippocampal size.
3. Ipsilateral atrophy of hippocampal collateral white matter
4. Enlarged temporal horn.
5. Reduced grey-white matter demarcation in the temporal lobe
6. Reduced temporal lobe volume.

In our study, we observed that coronal FLAIR images significantly detected hippocampal hyperintensity and temporal volume loss compared to T2 weighted images. T2 weighted FLAIR and fast FLAIR protocols suppress high signals from the CSF and make cortical and paraventricular lesions more conspicuous.

Space occupying lesions (both benign and malignant) are associated with focal epilepsy. In our study, 19.3% of patients showed an association with a space-occupying lesion (n=17). A study by Matsura M. et al. has demonstrated 11 patients with temporal lobe epilepsy with space occupying lesions; out of them, tumors were present in 7 patients. A study concluded that extensive neuroimaging is needed in focal epilepsy patients.

In our study, 9% of patients showed congenital abnormalities, mainly schizencephaly and heterotopias. Nodular heterotopia and/or overlying polymicrogyria can result in medically uncontrolled seizures.[13] Malformations of cortical development, or cortical cerebral dysgenesis, form a heterogeneous group of disorders, commonly associated with epilepsy and sometimes with learning disabilities. These neuronal disorders are commonly diagnosed incidentally when an MRI of the brain is done.

In our study, 17.3% of patients who presented with focal epilepsy showed features of demyelination, post-traumatic gliosis, or infarctions. Swartz BE et al. performed a study regarding hippocampal cell loss in post-traumatic epilepsy. The study concluded that neocortical pathology was universally present after trauma. Neuronal loss in the hilar region was the most consistent finding in the hippocampal formation, similar to that found in the fluid percussion model of post traumatic head injury. These findings support the idea that head trauma can induce epilepsy in humans alone.

A study by Christine Labrun et al. regarding epilepsy and multiple sclerosis has shown that epilepsy is 3-6 times more common in multiple sclerosis patients than in general populations. The anatomic basis for the seizure is areas of inflammation and demyelination. Partial seizures with focal and atypical presentations were the most common.

Our study has shown that 13.6% of patients with focal epilepsy suffered from various inflammatory or infectious etiologies like cysticercosis, tuberculoma and herpes virus infection. Julie Fotheringham et al. conducted a study in 2007 regarding the associations of the human herpes virus with mesial temporal sclerosis and concluded that there was a potential etiology and pathologic mechanism of mesial temporal sclerosis. Usegi et al. described HHV–6B as a possible cause of focal epilepsy. They found that three out of six pathological specimens of the temporal lobe were seropositive for HHV. Jacobson et al. (2007) have shown seropositivity in 11 patients out of 16 specimens from drug refractory partial seizures. Jones CM et al. in 1994 showed that status epilepticus may be associated with HHV-6 infection.

A study in East Africa by Steven J. Schiff et al. in 2007 showed a high prevalence of focal epilepsy in malaria prone regions. This is presumably related to brain damage due to the high incidence of febrile illness in children in that area.

Cendes Farnando et al. in 2001 described the association of neurocysticecosis with late onset mesial temporal sclerosis. The study concluded that acute neurocysticercosis associated with repeated seizures may develop mesial temporal sclerosis later. Garg R. K. et al. performed a study regarding neuroimmunological abnormalities in Indian patients with uncontrolled partial seizures and they concluded that the causes of uncontrolled partial seizures in India revolve around neurocysticecosis and the sequel of neurocysticecosis in the form of calcifications, which is often a focus of epileptogenesity.

In our study, 22.7% of patients with focal epilepsy did not show any abnormalities on an MRI scan. Most patients with non-lesional focal epilepsy have the finding of hippocampal sclerosis on high resolution MRI, but some patients show no abnormality on MRI.

Carne RP et al. (2004) conducted a study regarding MRI-negative PET positive focal epilepsy. The study concluded that MR-negative and PET positive epilepsy may be a surgically remediable syndrome distinct from hippocampal sclerosis positive focal epilepsy. With a patho physiologic basis that primarily involves lateral temporal, neocortical rather than mesial temporal structures.

A study by Salla Lamusuo et al. concluded that (18 F) FDG PET reveals temporal hypometabolism in patients with temporal lobe epilepsy even when quantitative MRI & histopathological analysis show mild hippocampal damage. Aristides A. et al. (2001) conducted a study regarding MR spectroscopy metabolite images for the laterisation of unilateral non lesiona tempora lobe epilepsy. The study concluded that reading of metabolite images is a feasible and fast means of non-invasive evaluation of patients with Temporal Lobe epilepsy who are candidates for surgery and enables lateralization in some patients with MR negative images.

In our study, 24 patients with focal epilepsy had a history of febrile convulsions. Wen-Chan Wu et al. conducted a study in 2005 on hippocampal changes in children with temporal lobe epilepsy with or without febrile convulsions. They used MR volumetry and MR spectroscopy to evaluate the changes. The study concluded that children with T.L.E. and an early history of febrile convulsions tend to have a lower hippocampal volume and (NAA/Cr+Cho) ratio than do TLE children without a history of febrile convulsions. The TLE with febrile convulsions group seemed to have increased vulnerability in the contralateral hippocampus. Van Landingham KE et al. conducted a study about Magnetic resonance imaging evidence of hippocampal injury after prolonged focal febrile convulsion and demonstrated that prolonged and focal complex febrile convulsion produced acute hippocampal injury that evolved into hippocampal atrophy. [14]

Conclusion

Various pathological processes may cause focal epilepsy. Focal epilepsy is a disorder mainly affecting the adolescent age group. There is a definite male preponderance. Febrile convulsions in childhood are associated with hippocampal sclerosis. Aura is a common clinical feature associated with temporal epilepsy.

MRI is an effective tool for detecting pathologies causing focal or partial epilepsy, except in some cases of non-lesional focal epilepsy. Coronal FLAIR images are superior to axial or coronal T2-weighted images for the detection of hippocampal sclerosis. There will be future scope for functional MRI, MRI and PET fusion or MRI and interictal SPECT fusion in cases of non-lesional MRI-negative focal epilepsy.

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