Role of 1.5T Magnetic Resonance Imaging in First Line Drug Resistant Epilepsy

Dr. Jyoti Gupta[1], Dr. I. D. Desai[2]

[1]Resident Doctor, MD Radiodiagnosis, BJ Medical College, Ahmedabad

[2]Professor, MD Radiodiagnosis, BJ Medical College, Ahmedabad

Abstract: ***A seizure is defined as a paroxysmal alteration in neurologic function due to excessive electrical discharge from the central nervous system. Epilepsy is defined as a condition of recurrent seizures, and medical intractability as recurrent seizures despite optimal treatment under the direction of an experienced neurologist over a 2-3-year period. Many patients don't respond to first line antiepileptic drugs. Many imaging modalities for investigating seizures are available like neurosonogram, computerized tomography (CT scan) of brain, magnetic resonance imaging (MRI) of brain, functional MRI, positron emission tomography (PET), single photon emission computed tomography (SPECT). Of these various modalities MRI is most important modality for evaluation of structural disorders causing epilepsy. So, role of 1.5T MRI is evaluated in 102 patients with refractory epilepsy in age group 0-40 years.***

Keywords: Refractory epilepsy, Radiological imaging, 1.5T MRI, epilepsy protocol imaging

1. Introduction-

Epilepsy is the fourth most common neurological disorder and affects people of all ages. At least fifty million people in the world suffer from recurrent non provoked seizures. Public perception and misunderstanding of epilepsy causes challenges often worse than the seizure. Incidence of epilepsy is clearly higher in developing countries than in developed countries.

A seizure is defined as a paroxysmal alteration in neurologic function due to excessive electrical discharge from the central nervous system. Epilepsy is defined as a condition of recurrent seizures, and medical intractability as recurrent seizures despite optimal treatment under the direction of an experienced neurologist over a 2-3-year period.

The main purposes of neuroimaging in epilepsy patients are to identify underlying structural or metabolic abnormalities that require specific treatment and to aid in formulating a syndromic or etiological diagnosis. Neuroimaging is even more important for those patients who have medically intractable seizures. Advances in technology to localize epileptogenic focus substantially improved the success of surgical treatment.

Structural disorders and metabolic disorders can be associated with seizure and detected on imaging. Structural disorders can be hippocampal or mesial temporal sclerosis, cortical developmental malformations or neuronal migration disorders, phakomatoses, vascular abnormalities, infections, neoplasms and scar epilepsy. However neoplastic causes of epilepsy are excluded in this study.

Many imaging modalities for investigating seizures are available like neurosonogram, computerized tomography (CT scan) of brain, magnetic resonance imaging (MRI) of brain, functional MRI, positron emission tomography (PET), single photon emission computed tomography (SPECT). Of these various modalities MRI is most important modality for evaluation of structural disorders causing epilepsy. So, role of MRI to be evaluated in patients with epilepsy.

2. Literature Survey

Idiopathic/Primary epilepsy- Idiopathic epilepsies are usuallyage-related, genetic disorders unassociated with lesions in the brain or other neurologic disturbances and they remit spontaneously.

Secondary or Symptomatic epilepsy-They result from lesions or disturbances in brain, that produces signs of symptoms besides epilepsy.

Cryptogenic epileptic disorder-That are symptomatic but doesn't have any identifiable cause.

2.1 Classifications

International Classification of Epileptic Seizures (1981)

I. Partial (focal, local) seizures
- a) Simple partial seizures
  - 1) With motor signs
  - 2) With somato-sensory or special sensory symptoms
  - 3) With autonomic symptoms or signs
  - 4) With psychic symptoms
- b) Complex partial seizures
  - 1) Simple partial onset followed by impairment of consciousness
  - 2) With impairment of consciousness at onset
- c) Partial seizures evolving to secondarily generalized seizures
  - 1) Simple partial seizures evolving to generalized seizures

2) Complex partial seizures evolving to generalized seizures
3) Simple partial seizures evolving to complex partial seizures evolving to generalized seizures

II. Generalized seizures (convulsive or non convulsive)

a) Absence seizures
    1) Typical absences
    2) Atypical absences
b) Myoclonic seizures
c) Clonic seizures
d) Tonic seizures
e) Tonic-clonic seizures
f) Atonic seizures (astatic seizures)

III. Unclassified epileptic seizures

Modified Internationsl League Against Epilepsy Classification of EpileptcSydromes (1989)

I. Idiopathic epilepsy syndromes (focal or generalised)

a) Benign neonatal convulsions (familial and non familial)
b) Benign childhood epilepsy
    - With central mid temporal spikes.
    - With occipital spikes
c) Childhood / juvenile absence epilepsy
d) Juvinile myoclonic epilepsy
e) Idiopathic epilepsy , otherwise unspecified.

II. Symptomatic epilepsy syndromes

a) West syndrome (infantile spasms)
b) Lennox- Gastaut syndrome
c) Early myoclonic encephalopathy.
d) Epilepsiapartialis continua
    - Rasmussen syndrome (encephalitic form)
    - Restricted form
e) Aquired epileptic aphasia
f) Temporal lobe epilepsy
g) Frontal lobe epilepsy
h) Posttraumatic epilepsy
i) Other symptomatic epilepsy , not specified

III. Other epileptic syndromes of uncertain or mixed classification

a) Neonatal seizures
b) Febrile seizures
c) Reflex epilepsy
d) Other unspecified

According to Anatomic localization

1) Temporal lobe epilepsies
2) Frontal lobe epilepsies
3) Parietal lobe epilepsies
4) Occipital lobe epilepsies
5) Bi- and multilobar epilepsies

Etiology

I. Inheritant Metabolic disorder

II. Structural abnormality

1. Malformations of Cortical Development Abnormal neuronal and glial proliferation or apoptosis

- microcephaly
- Abnormal proliferation (abnormal cell types)
    a) Cortical hamartomas of tuberous sclerosis
    b) Cortical dysplasia
    c) Hemimeganencephaly

Abnormal neuronal migration

- Lissencephaly
- Heterotopia
    a) Subependymal
    b) Subcortical
    c) Marginal

Abnormal cortical organization

- Polymicrogyria and pachygyria
- Schizencephaly

2. Hippocampus abnormality

- Mesialtemporal lobe sclerosis

3. Vascular malformation

- AV malformation
- Venous malformation (capillary telangiectasia, cavernous)

4. Phakomatoses

- Tuberous sclerosis
- Sturge Weber syndrome
- Neurofibromatosis

5. Cerebral Infections

- Tuberculoma
- Neurocysticercosis
- Cerebral abscess

6. Inflammatory disorder

- Rasmussen encephalitis
- Sarcoidosis
- cerebral vasculitis

7. Scar epilepsy secondary to gliosis in trauma & stroke

8. Intra Cranial Neoplasm

- Glioma
- Neuronal tumors
- Hemopoietic and blood vessel tumors
- Embryonal &neuroblastic tumors
- Germ cell tumors

9. Non-neoplastic lesions

- Epidermoid cyst
- Dermoid cyst
- Colloid cyst
- Neuroenteric cyst
- Porencephalic cyst
- Neuroglial cyst
- Hypothalamic hamartoma

III. Electrolyte disturbances(reduced Na+ &Mn+)

IV. Alcohol and Drug Abuse

Pathophysiology

Enhanced inhibition and disturbed function of certain low-threshold calcium channels in the thalamus cause the abnormal hypersynchronization underlying generalized spike-and-wave discharges associated with absence seizures.

On the other hand, generalized tonic-clonic seizures may result from enhanced excitation in brainstem and neocortical structures.

MTLE is usually associated with hippocampal sclerosis, which appears to be epileptogenic as a result of enhanced excitation and inhibition, resulting in hypersynchronization.

3. MRI Appearnce

I) Malformations of Cortical Development

1) ***Hemimeganencephaly***Hamartomatous overgrowth of part/all of a hemisphere due to defect of cellular organization, neuronal migration**.** Mild, moderate, or markedly enlarged dysplastic hemisphere**,** Dysplastic cortex, abnormal gyri**,** Displaced posterior falx**,** Large lateral ventricle with abnormally shaped frontal horn
2) ***Pachygyria-polymicrogyria-*** Pachygyria consist of focal areas of thickened, flattened cortex which may be associated with white matter hyperintensity on T2.Polymicrogyria consist of diffusely thickened, abnormal cortex with irregular bumpy gyral pattern.
3) **Cortical heterotopias-**Arrested/disrupted neurons along migration path from periventricular germinal zone to cortex**. MR Findings-** Subependymal heterotopia (most common)- Subependymal lesions tend to be nodular and to predominate near the trigone of the ventricle.Band heterotopia ("double cortex")- It exhibit ribbons of ectopic gray matter between cortex and ventricles with white matter on both sides.
   Subcortical heterotopias-Focal Heterotopic Gray matter nodule and Large nodular Heterotopic Gray matter (can mimic neoplasm)
4) ***Lissencephaly(smooth brain)***
   Lissencephaly type 1 : It is characterized by Shallow Sylvian fissure with "hour-glass" cerebral configuration(also called figure of 8 appearance) with thick cortical gray matter with smooth gray-white interface
   Lissencephaly type 2(congenital muscular dystrophy) : Neurons "over migrate" through gaps in external layer of cortex, so give "pebbled" surface of brain.
5) **Schizencephaly**Schizencephaly is characterized by clefts extending from pial surface of cerebral mantle to ventricle lined by cortex.**Types** "Closed-lip" (small defect) or "open-lip" (large defect)**.**Trans-mantle gray matter lined clefts
6) ***Focal cortical dysplasia***-Taylor's classification of focal cortical dysplasia divide it into three types.FCD type I – significant segmental or lobar hypoplasia/atrophy often coexistent with reduced volume of subcortical white matter, which may reveal moderately increased signal on T2-weighted images & FLAIR and decrease on T1W. Focal cortical dysplasia type I is frequently found in the temporal lobe with hippocampal atrophy. FCD type II is characterized by cortical thickening, marked blurring of GM/WM junction, and in some patients with a slight increase of white matter signal on T2-weighted images. Altered white matter signal is often extended towards the ventricle (transmantle sign). It is more often found in frontal lobe.FCD type III is associated with hippocampal atrophy, developmental tumors or arteriovenous malformations.

II. Hippocampus Abnormality

Mesial temporal lobe sclerosis(MTS)Mesial temporal lobe or hippocampal sclerosis is characterized by pyramidal and granule cell neuronal loss in the cornuammonis and gyrus dentatus. MTS is the most common pathology associated with temporal lobe epilepsy. On**T1W-IR-** Decreased size of hippocampus, loss of gray-white differentiation in hippocampus. On**T2WI & FLAIR**- Hyperintense signal in the hippocampus. **Quantitative hippocampal volumetry** may increase sensitivity of MTS detection, particularly in patients with bilateral MTS

III. Vascular Malformations

(1)Arteriovenous malformation(AVM)

1) **Parenchymal AVM – On T1WI & T2W-** Signal varies with presence/age of hemorrhage, Tightly packed honeycomb of "flow voids"
   On T2* GRE- "blooming" if hemorrhage present, On **T1 postcontrast and MRA-** Tightly packed anomalous vascular channels with feeding artery and draining vein.Flow related aneurysm and dilated draining veins are associated findings.
2) **Dural AVM/AV fistula- T1WI & T2W**May be normal or Isointense thrombosed dural sinus with or without "flow voids". **T2* GRE** shows Blooming if hemorrhage occurs. On **T1 postcontrast d**iffuse dural enhancement may seen.

(2)Venous malformation

Cavernous malformation- T1WI &T2WI- Variable appearance depending on hemorrhage/stage**,** "Popcorn ball" appearance of mixed hyper-, hypointense blood-containing "locules". On **T2* GRE-** "blooming" is seen with gradient image.

Venous angioma- T1WI & T2W Can be normal if lesion is small. Variable signal depending on size, flow with or without "Flow void"**. Tl postcontrast & MRV**Delineates "Medusa head" and drainage pattern

IV. PHAKOMATOSIS

(1Tuberous Sclerosis Inherited tumor disorder with multi-organ hamartomas, Classic imaging appearance: Calcified subependymal nodules seen as blooming on T2W GRE**,** Subependymal giant cell astrocytoma (SEGA) 15%**,** White matter lesions 70-95%**,** Cortical/subcortical tubers: Early T1 increase, but variable after myelin maturation**,** Streaky linear or wedge-shaped hyperintensities(along radial migration lines from ventricle to cortex)**,** Subependymal nodules enhancement in 30-80%

(2)Sturge-Weber SyndromeA sporadic congenital malformation in which fetal cortical veins fail to develop normally leads to progressive venous occlusion and chronic venous ischemia,**On T1W &T2W-** Early: Transient hyperperfusion leads to accelerated myelin maturationLate: Cortical atrophy with areas of gliosis and calcification**.** ***On*T2* GRE** Tram-track gyral calcifications

V. Infections

(1)Brain abscess Focal pyogenic infection of the brain parenchyma, typically bacterial; fungal or parasitic.**Four pathologic stages-**Early cerebritis, late cerebritis, early capsule, late capsule. On **T1WI & T2WI**Early cerebritis: Poorly marginated, mixed hypointense on T1 &hyperintense on T2 and mass**.** Late cerebritis: Hypointense center, isointense/mildly hyperintense rim on T1 while opposite in T2**.** Early capsule: Rim hyperintense on T1 &hypointense on T2 to white matter; center isointense to CSF**.** Late capsule: Cavity shrinks, capsule thickens. On **DWI**restriction in abscess**. *On* Tl postcontrast** rim enhancement

(2) Tuberculosis-Basal meningitis + extracerebral TB (pulmonary) + parenchymal lesions (tuberculoma) highly suggestive**.TlWI&T2WI- Tuberculoma**:Noncaseating: hypointenseon T1 and Hyperintense on T2WI, Caseating granuloma with solid center: Hypointense or isointense to brain. Caseating granuloma with necrotic center: Hypointense or isointense to brain with central hypointensity on T1 while hyperintense lesion with hypointense rim on T2. **TI Postcontrast**- Nodular or ring enhancement with or without Marked meningeal enhancement(may be nodular), basal prominence

(3)Neurocysticercosis- Intracranial parasitic infection caused by the pork tape worm, Taeniasolium**. Four pathologic stages**: Vesicular, colloidal vesicular, granular nodular, nodular calcified**. T1WI & T2WI**stage: Cystic lesion isointense to CSF eccentric , scolex (hyperintense on T1), Colloidal vesicular stage: Cyst is mildly hyperintense to CSF**,** Granular nodular stage: Thickened, retracted cyst wall; edema decreases**,** Nodular calcified stage: Shrunken, Calcified lesion**. TI Postcontrast-** ring postcontrast enhancement with or without enhancing nodule**. T2* GRE**- Useful to demonstrate calcified scolex

VI. Inflammation

(1)Rasmuseen Encephalitis- Chronic, unilateral inflammation of brain of uncertain etiology Characterized by hemispheric volume loss and difficult to control focal seizure activity**. • T1WI**- Atrophy of involved cerebral hemisphere or lobe noted in late stage.**T2WI**- Early focal swelling of gyri , Late: Atrophy of involved cerebral hemisphere or lobe

4. Metabolic Diseases

I) Mitochondrial disorders

(1)Leigh syndrome-Genetically heterogeneous mitochondrial disorder characterized by progressive neurodegeneration**. T1WI**-Hypointense with variable foci of hyperintensity due to blood or myelin degradation products. **T2WI-** Bilateral, symmetrical hyperintensity of putamen and peri-aqueductal gray matter

(2)MELAS(Mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes)-Inherited disorder of intracellular energy production caused by point mutation in mtDNA. **T1WI-** Acute: Swollen gyri, compressed sulci, Subacute: Band of cortical hyperintensity consistent with laminar necrosis, Chronic: Progressive atrophy of basal ganglia, temporal-parietal-occipital cortex with preservation of hippocampal, entorhinal structures. **T2WI-** Acute: Hyperintense cortex/subcortical WM, Chronic: Multifocal hyperintensities in basal ganglia, deep WM

II)Lysosomal disorders

1)Gangliosidosis(GM2)-Inherited glycosphingolipid lysosomal storage disorder characterized by GM2 ganglioside brain accumulation.Bilateral, symmetric thalamic CT hyperdensity, T2 hypointensity, Tl hyperintensity (infantile GM2)

(2)Metachromatic leukodystrophy (MLD)-Progressive neurodegenerative lysosomal storage disorder due to deficiency of arylsulfatase A (ARSA)**.** Confluent "butterfly-shaped" cerebral hemispheric white matter (WM) hyperintensity on T2W spares subcortical U-fibers, internal capsules.

(3)Krabbe's disease- Progressive autosomal-recessive degenerative leukodystrophy. On **T1WI**Deep, periventricular white matter hypointensity, Thalami, basal ganglia hyperintensity. On **T2WI**- Hypointensity in thalami and basal ganglia, Confluent symmetric deep periventricular white matter hyperintensity and Spares subcortical U-fibers. **T1 postcontrast:** Infantile (late): Enhancing rim between abnormal white matter and unaffected U-fibers

III) Peroxisomal disorders

(1)Zellweger syndrome-Neonatal leukodystrophy resulting from peroxisome Failure.On **T1WI** Profound hypomyelination, hypointensesubependymalcysts.On **T2WI-** Microgyria common in the frontal and temporal regions and Hyperintense white matter and germinolytic cysts.

(2)X-Linked adrenoleukodystrophy-Inherited disorder of peroxisome metabolism due to impaired oxidation of very long chain fatty acids. Peri trigonal white matter (WM) demyelination showing post contrast enhancement isclassical diagnostic feature of adrenoleukodystrophy

IV) Organic and Aminoacidopathies

1) ***Maple syrup urine disease***-It is an inherited disorder of branched chain amino acid metabolism presenting in newborns with neurologic deterioration, ketoacidosis and hyperammonemia. T1 hypointensity and T2 hyperintensity involving Cerebellar white matter, brain stem, globus Pallidus, Thalamus, cerebral peduncles, corticospinal tracts (to cortex). Marked restriction seen as increase signal intensity.
2) ***Urea cycle disorders***-It includes Carbamyl phosphate synthetase deficiency (CPSD), ornithine transcarbamylase deficiency (OTD),argininosuccinatesynthetase / ligase deficiencies (ASD/ALD), arginase deficiency (AD, citrullinemia)**. *I***ncrease signal in deep gray nuclei andinsular/perirolandic cortex
3) ***Glutaric aciduria type 1***-Inborn error of metabolism characterized by encephalopathic crises and resultant severe dystonic-dyskinetic movement disorder**..** Subependymal pseudocysts (disappear by 6 months) and Fronto-temporal atrophy. **T2WI**Increase signal caudate/putamina>globus pallidus which atrophy over time. If severe: White matter (WM), thalami, dentate nuclei may be involved

4) ***Canavan disease***Progressive autosomal-recessive Leukodystrophy. MacrocephalyEarly: Subcortical U-fiber involvement, centripetal white matter involvement**.** Late: Progressive cerebral atrophy and cerebellar dentate involvement
5) **Alexander disease**Rare leukoencephalopathy characterized by macrocephaly and psychomotor regression**.** Diffuse, symmetrical bifrontal white matter (WM) signal abnormality (T2 hyperintensity) and post contrast enhancement

5. Methods

This is a prospective, single-center, observational study. Our institute serves a primary population of approximately 5 million inhabitants. The Department of Imaging & Radiology provides a full range of services of diagnostic imaging to all the patients of hospital. We included 102 patients with epilepsy between the period of June 2014 to September 2016. Each patient was studied in detail with relevant to clinical history, examination and laboratory investigation. They all are examined using magnetic resonance imaging of brain as prime diagnostic modality at our institution.

Inclusion criteria- Patients with any of the following are included in present study. All patients with known case of partial seizures between age ranging since birth to 40 years.Onset of generalized or unclassified seizures in the first year of life, or in adulthood Evidence of a fixed deficit on neurological or neuropsychological examinationDifficulty obtaining seizure control with first-line antiepileptic drugs (AEDs)

Exclusion criteria- All patients with seizures due to neoplasm**.** All patients with alcohol and drug abuse**.** All patients with neonatal asphyxia, perinatal trauma and congenital infections.

Imaging methods- Scanning is done on 1.5 Tesla Phillips Achieva MR machine. Standard brain sequences is composed of an axial T2-W, axial & sagittal T1-W, coronal FLAIR, sagittal T2-W images and DWI (with ADC) for all patients. Routine MRI consists of a short scan time, 3 to 5 mm thick slices with an interslice gap of 2-3 mm. Epilepsy protocol includes T1-IR images 1.5-mm slice thickness with no intervening gap obtained in the coronal oblique plane and coronal & axial FLAIR sequences with a 2- to 3-mm slice thickness and 1-mm interslice gap. Post contrast T1W images(axial, coronal & sagittal) can be taken after administration of gadolinium(0.1 mmol/kg dose) in selected cases. Time of flight angiography and T2* gradient recall echo (GRE) sequence are taken in patients with vascular malformations.

Statestical methods

All the collected data were entered in excel sheet. A binary classification test is used for statistical analysis.

6. Discussion

In this study 102 known epileptic patients with upto 40 years of age who followed inclusion and exclusion criteria were included. After taking proper history, neurological examination and laboratory investigations, all patients underwent MRI of brain with routine and epilepsy protocol. Contrast study was done in specific conditions for further characterization of lesions. Specific treatment and a proper follow up of patients were carried out.

Out of all patients up to 40 years of age with epilepsy, maximum incidence was found inage group of 16 to 20 years (18 patients).Epilepsy was more common in males (58.8%) than females (41.2%) in present study.

Commonest presenting seizure type were complex partial seizure (31.3%) and generalized tonic clonic seizure (33.3%). Focal neurological deficits were present in 26.6 % of patients on neurological examination.

Incidence of structural abnormality on MRI in present study was 67.7 % as compared to 74% in study by L M Li, D R Fish, S M Sisodiya.

Among all the patients with epilepsy causes in descending order are idiopathic (32.3%) followed by Infections(14.7%), Cortical malformation(14.7%), hippocampal pathology (7.8 %), vascular compromises (6.8), metabolic causes (5.8 %) , hydrocephalus (3.9%), gliosis (3.9%), phakomatoses (1.9 %) and AVM(1%).Among epileptic patients with abnormal MRI findings, tuberculoma was most common structural lesion comprising 15%, as comparable with the study- MAGNETIC RESONANCE IMAGING IN CHILD HOOD EPILEPSY, by P.Gulani, A Jena, R.P. Tripathi, A.K. Gupta. This was followed by unilateral hippocampal sclerosis comprising 7.8%.

Among patients with epilepsy, cortical malformations was most common cause of epilepsy in <10 years of age. Hippocampal malformations are common in first two decades of life. While infections were commonest structural abnormality in 11-20 years years of age.

Most common lesion causing partial seizure and partial seizures with secondary generalization is cortical malformations , infections and hippocampal sclerosis. Generalized seizures is most commonly associated with idiopathic epilepsy (39.9%) followed by abnormal white matter hyperintensity and infections (15%).

Most of structural brain abnormalities lead to epilepsy were located in temporal lobe (either isolated or with multilobarpresenation combined - 40 %) followed by parietal lobe (26%). Thus temporal lobe lesions were more epileptogenic.

Abnormal white matter hypointensity on T1W images and hyperintensity on T2W images were most common finding on MRI study comprising 27%%.

Out of 8 patients with hippocampal pathology diagnosed on epilepsy protocol with volumetry, only 1 patients were diagnosed with routine protocol. While 15 patients with cortical malformation were diagnosed on epilepsy protocol with or without volumetry, only 5 patients were picked up on routine protocol. So, only 13% of hippocampal pathology, 33% of cortical malformation were diagnosed on Routine MRI Protocol study compared to Epilepsy Protocol with or without volumetry. Thus epilepsy protocol with or without volumetry is more sensitive then routine protocol to diagnose hippocampal pathology and cortical malformations.

Out of 102 patients presented with epilepsy 43.1% of patients responded to antiepileptic drugs, 38% patients were refractory to antiepileptic drug therapy. 27.4% of patients required specific medical management and 7.8 % of patients treated with surgical management. 1 % of patients were expired. Rest of the patients could not be traced for follow-up

Table 1: Agewise Distribution of Lesion

Age Group(in years)	Number of patients
Up to 5	18
6-10	15
11-15	17
16-20	18
21-25	13
26-30	9
31-35	6
36-40	6
TOTAL	102

Table 2: Sexwise Distribution of Lesion

Sex	*No. of Patients*	*Percentage*
Male	60	58.8%
Female	42	41.2%

Table 3: Agewise Distribution of Patients with Respect to Sex

Age Group (in years)	*Male*	*Female*	*Total*	*Percentage (male)*
Up to 5	12	6	18	66.6 %
6-10	8	7	15	53.3%
11-15	13	5	18	72.2 %
16-20	7	11	17	41.2 %
21-25	5	8	13	38.4%
26-30	7	2	9	77.8 %
31-35	3	3	6	50%
35-40	6	0	6	100 %
TOTAL	60	42	102	

Table 4: Clinical Presentation

Clinical presentation	*No. of cases*	*Percentage*
Partial Seizure (1) Simple (2) Complex	37 12 25	36.2% 11.8% 24.2%
Partial seizure with secondary generalization	32	31.3%
Generalized seizure (1) Absence Seizure (2) Generalized tonic clonic seizure (3) Myoclonic	34 1 33 -	33.3% 1% 32% Nil
Focal neurological Sign	26	25.4%

Table 5: Distribution and Frequency of Various Lesions

Lesion	*Total cases*	*% incidence*
Cortical malformation	15	14.7%
Schizencephaly	5	4.9 %
Lissencephaly	3 + 1	3.9%
Cortical heterotopias	2	1.9%
Focal cortical dysplasia	7	6.8%
Hippocampal pathology	8	7.8 %
Unilateral hippocampal sclerosis	8	7.8%
Bilateral hippocampal sclerosis	Nil	
Infection	15	14.7 %
Tuberculoma	11	10.7%
Neurocysticercosis	3	2.9%
Meningitits	1	1%
Vascular Malformation	1	1%
Pail AVM	1	1%
Abnormal white matter	1	1%
Nonspecific hyperintensity	2	1.9%
Phakomatosis	2	1.9%
Tuberous sclerosis	2	1.9%
Gliosis/ Scar Epilepsy	3	2.9%
H/O cerebro vascular accident	2	1.9%
Vascular compromise	6	5.9 %
Cerebral atrophy	4	3.9 %
Metabolic causes	6	5.8 %
Hydrocephalus	4	3.9 %
Normal MRI study	33	32.3 %
Total	102	100%

Table 6: Distribution of Lesion According to Etiology

Etiology	No. of cases	Percentage (%)
Idiopathic	33	32.3
Cortical malformation	15	14.7
Hippocampal pathology	7	7.8
Phakomatosis	2	1.9
Vascular malformation/ vasculitis	1	1
Infection	15	14.7
Scar Epilepsy(Gliosis)	3	2.9
Inheritant metabolic disease	2	1.9
Acquired metabolic disease	4	3.9
Non specific white matter Hyperintensity	2	1.9

Table 7: Age Wise Distribution of Various Lesions in Epilepsy

Age (in years)	Idiopathic	Cortical malformation	Hippocampal Pathology	Phakomatosis	Vascular malformation Vascular compromise	Infection	Gliosis	Abn. White matter Hyperintensity	Hydrocephalus	Cerebral trophy	Total
<5	2	5	2	1	2	2	-	-	-	-	18
5-10	3	5	1	1	2	2	-	-	-	-	15
11-20	14	4	2	-	3	4	1	1	2	4	35
21-30	9	1	1	-	2	5	1	1	2	-	22
31-40	5	-	2	-	1	2	1	-	-	-	12
Total	33	15	8	2	10	15	3	2	4	4	102

Table 8: Anatomical Localization of Different Lesions in Epilepsy

	FL	PL	TL	OL	ML	D	Total
Hippocampal pathology	-	-	8	-	-	-	8
Cortical malformation	-	4	1	2	7	1	15+1
Schizencephaly	-	1	-	2	2	-	5
Lissencephaly	-	-	-	-	2	1	3
Cortical heterotopia	-	-	-	-	2	-	2
Focal cortical dysplasia	2	3	1	-	1	-	7
Phakomatosis	-	-	--	-	1	1	2
Tuberous sclerosis					1	1	2
Vascular malformation/ vascular compromise	1	2	2	-	5	-	10
Infection							15
Meningitis	-	-	-	-	1	-	1
Tuberculoma	3	3	-	-	2	3	11
Neurocysticercosis	1	1	1	-	-	-	3
Gliosis	-	-	-	-	-	3	3
Abn. White matter hyperintensity	-	2	-	-	-	-	2
Hydrocephalus	-	-	-	-	-	4	4
Atrophy	-	-	-	-	-	4	4
Metabolic	-	-	1	-	3	2	6
Total	7	17	14	4	27	20	

FL- frontal lobe ; TL-temporal lobe ; PL-parietal lobe ; OL-occipital lobe ; ML-multiple lobe ; D-diffuse.

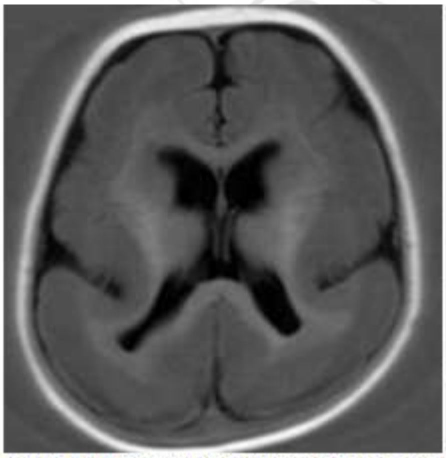
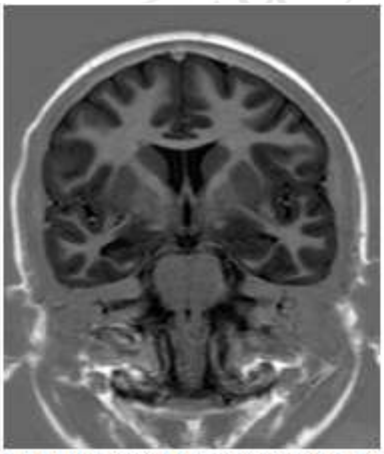
Table 9: Role of Epilepsy Protocol In Various Lesions in Epilepsy

LESION	Routine protocol	Epilepsy protocol+/-volumetry
Cortical malformation	5	10
Schizencephaly	2	3
Lissencephaly	-	3+1
Cortical heterotopias	-	2
Focal cortical dysplasia	3	4
Hippocampal pathology	1	7
Unilateral hippocampal sclerosis	1	7
Phakomatosis	1	1
Tuberous sclerosis	1	1
Vascular compromise	6	3
Vascular Malformation	1	-
Pail AVM	1	-
Infection	11	4
Tuberculoma	9	2
Neurocysticercosis	2	2
Meningitis	1	-
Gliosis(Scar Epilepsy)	2	1

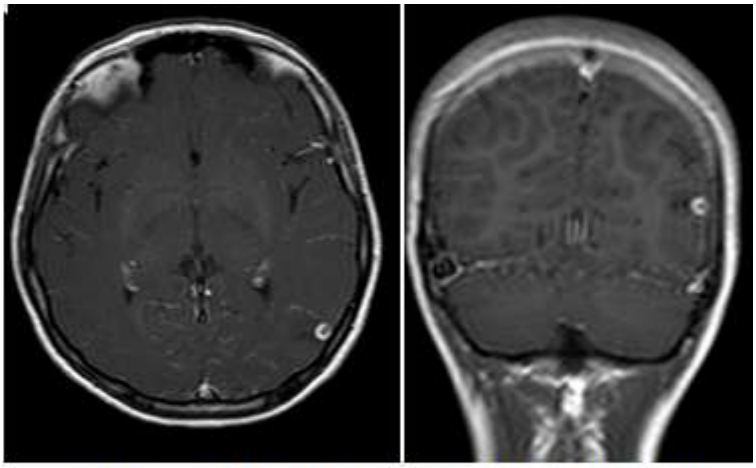
Abnormal white matter	2	-
Nonspecific hyperintensity	3	3
Hydrocephalus	4	-
Cerebral atrophy	4	2
Cyst	-	1
Metabolic	3	3
Normal study	-	33
Total	41	61

Table 10: Follow Up and Further Management

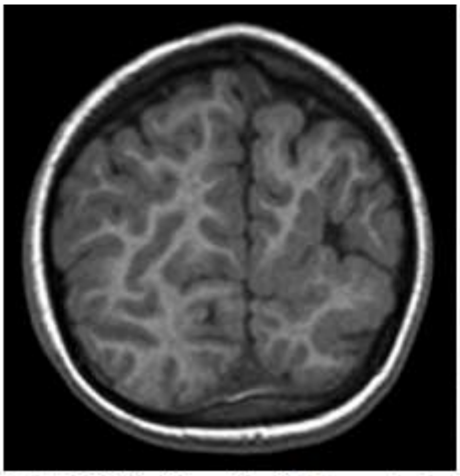
Type of Lesions	Anti epileptic drugs	Specific Medical Mx	Surgical Mx	Expired	Follow-up not available	Total
Cortical malformation	8	-	3	1	4	15
Schizencephaly	5	-		-	-	5
Lissencephaly	5	-	-	1	-	5
Cortical heterotopias	2	-	-	-		2
Focal cortical dysplasia	-	-	3	-	4	7
Hippocampal pathology	8	-	-	-	-	8
Unilateral hippocampal sclerosis	8	-	-	-	-	8
Infection	-	15	-	-	-	15
Tuberculoma	0	11	-	-	-	11
Neurocysticercosis	0	3	-	-	-	3
Meningitis		1	-			1
Vascular Malformation	-	-	1	-	-	1
Pail AVM	-	-	1	-	-	1
Vascular compromise	3	6				9
Nonspecific hyperintensity	1	-	-	-	1	2
Phakomatosis	2	-	-	-	-	2
Tuberous sclerosis	2	-	-	-	-	2
Gliosis(Scar Epilepsy)	2	1	-	-	-	3
Hydrocephalus	-	-	4	-	-	4
Cerebral atrophy	4	-	-	-	-	4
Metabolic		6				6
Arachnoid cyst	1	-	-	-	-	1
Normal MRI study	17	-	-	-	16	33
TOTAL	44	28	8	1	21	102

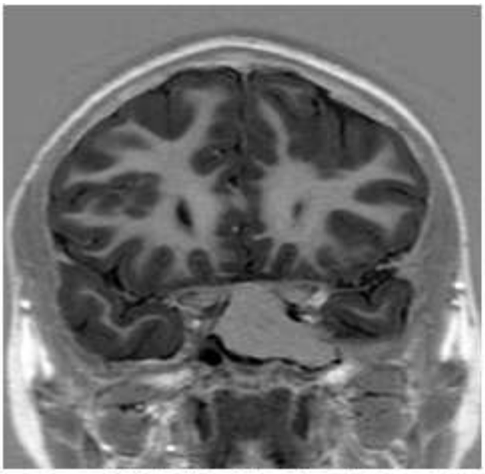
Axial T1WI of classical lissencephaly Coronal T1W- IR of right sided mesial Temporal Sclerosis



Axial T2WI and Coronal T1 post contrast images of Tuberculoma



Axial T1WI of closed lip Schizencephaly



Coronal T1W-IR of Focal cortical dysplasia

7. Conclusion

The imaging of epilepsy is to differentiate primary from secondary epilepsy and also to diagnose and localize structural abnormality that will guide management decisions and reduce morbidity. Benefitting from superior soft tissue contrast, true multiplanar capability, absence of bone artifact and potential for high resolution, MRI has evolved into the unrivaled imaging standard to differentiate primary from secondary epilepsy and to identify underlying structural abnormalityMRI reveals significant number of structural abnormalities in patients with focal seizures, symptomatic generalized seizures, refractory epilepsy, patients with focal neurological deficit and patients with early(infants) or late(adult) onset of epilepsy. Thus incidence of structural abnormality is more in this group of patients. Structural abnormalities are more common with partial seizure then generalized seizures. Underlying cause of epilepsy varies significantly with age.Epilepsy protocol has better yield, sensitivity and specificity than routine protocol in patients with hippocampal pathology and cortical malformation. In order to maximize the potential of the technique, the protocol should be crafted to the needs of the individual epilepsy patient.Thus we conclude that MRI is most important & reliable structural neuroimaging modality to evaluate the patients with epilepsy.

8. Further Scope

MRI has a major impact on management of epileptic patients. MRI identify causative lesion and localize it, thus helps to identify further line of management (medical or surgical). MRI is useful to plan surgical management and for post-operative followup. However, the disadvantages of MRI are its unavailability for larger number of patients, higher cost, and the requirement for longer time periods for scanning. MRI can not be performed in patients with cochlear implant, pacemaker and metallic implants. Children with benign idiopathic generalized epilepsy,uncomplicated febrile convulsions and a normal neurological exam usually do not require imaging studies. Newer imaging techniques like fMRI and SPECT imaging may aid to the diagnosis.

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Author Profile

Dr. Jyoti Gupta has completed her MBBS and MD Radiodiagnosis from BJ Medical College and Civil Hospital , Ahmedabad. Secured Gold medal in MD exit exams.

Dr. I. Desai, Professor and PG Guide in department of Radiodiagnosis, B. J. Medical College and Civil Hospital, Ahmedabad.