A seizure is a paroxysmal alteration in neurologic function resulting from abnormal excessive neuronal electrical activity. The pathophysiologic basis of seizures is loss of normal regulation of neuronal excitation and inhibition, resulting in a state of relative hyperexcitability. Epilepsy is a chronic condition characterized by recurrent seizures unprovoked by an acute systemic or neurologic insult.

MRI plays a pivotal role in every aspect of the diagnosis and management of seizures.

MR techniques are crucial for a complete workup of a patient with seizures because the ultimate goal of treatment is relief from symptoms, whether it be medical or surgical treatment and in the latter case prevention of disastrous postresection complications, such as speech and memory loss.

The International classification of epilepsies and epileptic syndromes classifies clinical epilepsy into two broad categories, idiopathic (primary) and symptomatic (secondary) disorders [1]. Primary epilepsies are genetically transmitted seizures that are not associated with other neurological disturbances or structural pathology and are usually benign. Secondary epilepsies, in contrast, are seizures resulting from a specific pathologic substrate that can be genetic or acquired. There is a third group which includes epilepsy syndromes of uncertain or mixed classification such as neonatal seizures, febrile seizures etc.

Both primary and secondary epilepsies are further divided into generalized disorders (where the brain is diffusely and bilaterally involved) and localization-related or focal disorders (seizures originate from a localized cortical region).

The common causes of seizures can be roughly classified as

1) Idiopathic (no identifiable cause)
   - Usually begin between 5-20 years.
   - Can occur at any age.
   - No other neurologic abnormality.
   - Often family history of epilepsy or seizures.

2) Mesial Temporal sclerosis: In addition to being the lesion most commonly associated with complex partial seizures (60-85% cases) [2], it is also the most common structural abnormality in human epilepsy [3]. It is characterized by pyramidal cell loss and astrogliosis in the mesial temporal lobe, hippocampal formation, amygdala, parahippocampal gyrus and the entorhinal cortex [4].

Although MTS is a bilateral process, one side is affected more than the other in 80% of patients. Usually, the more severely affected side is the origin of the patients seizures. Cases in which both sides are equally affected generally respond less favorably to temporal lobectomy.
MR features of MTS

Primary signs

- A small atrophic unilateral hippocampus (fig.2).

- Hyperintensity on both T2 W and FLAIR images (Fluid attenuated inversion recovery images)(fig.3).

- Loss of the hippocampal internal architecture and that of normal digitations of the head (fig.3).

- Visual assessment of size, architecture and signal intensity changes is quite sensitive, with the eye being able to detect asymmetry of 14% or more [5].

Secondary signs

- Unilateral atrophy of the mamillary body [6], fornix columns (circuit of paper) [7], and the amygdala.

- Increased T2 W signal in the anterior temporal lobe white matter [8] with loss of grey-white demarcation in the ipsilateral anterior temporal lobe (fig.4).

- Unilateral dilatation of the temporal horn (a less reliable secondary sign) [4]

- Unilateral atrophy of the collateral white matter bundle [8].

Volumetric MR Imaging:

Quantitative evaluation of hippocampal volume has been found to marginally increase the sensitivity over visual analysis in detection of hippocampal sclerosis [9].

Measuring size can be accomplished by manually tracing the hippocampus. The normal, ipsilateral hippocampal volume is approximately 2.8 Ml [4].

T2 Relaxometry:

It is used to quantify the T2 signal in the hippocampus. It
measures the decay in signal intensity at different TE s in a series of T2W images acquired in the same slice. In MTS, the relaxation time has proven to be lengthened by 10 milliseconds [4].

Other techniques useful in MTS are diffusion tensor imaging and functional MR imaging.

3) Developmental Disorders: These can be further divided into four groups [12] on the basis of
- Abnormal cell proliferation.
- Abnormal neuronal migration.
- Abnormal cortical organization.
- Unclassified miscellaneous group.

Some of the common abnormalities are:

Focal Cortical Dysplasia is among the most common causes of epilepsy attributable to focal cerebral dysgenesis. 60% FCD is found in temporal lobes [13].

Proton MRS has been widely used in providing insight into the metabolic alterations in epilepsy.

N-acetyl - aspartate (NAA) is a marker of metabolically active neurons, and decreased NAA/ creatinine or decreased NAA / creatinine +choline ratios signify neuronal loss and / or metabolic dysfunction. A decrease in these ratios has been shown to lateralize temporal lobe epilepsy in 65% to 90 % of patients with bilateral temporal lobe structural abnormalities on MR [10]. In cases of temporal lobe epilepsy with normal MR studies, NAA ratios can provide lateralizing evidence in at least 20% of patients [11] (fig.6).

In focal transmantle dysplasia, abnormal signal is seen to extend from the cortex to, or almost to, the ventricle [14] (fig.8).
Subcortical heterotopia may occur at any location from the periventricular white matter to the grey-white matter junction. Band heterotopia is found almost exclusively in females, although rare cases in males have been reported [16]. On imaging, white matter zone separates a thin rim of cortex from a broad band of subcortical grey matter.

Grey Matter Heterotopia:

It is the focal collection of ectopic neurons in the cerebral hemispheres and can be of three broad types:

- Subependymal heterotopia appearing as ovoid lesions within the subependymal region. They are isointense with grey matter on all imaging sequences [15](fig.9).

In FCD without balloon cells, the lesion may be evident only as areas of blurring of the cortical white matter junction.

**Fig8** Focal transmantle dysplasia left frontal lobe: Thickened cortex with abnormal signal extending from the cortex to the left frontal horn.

**Fig9** Subependymal heterotopia along the right occipital horn.

**Fig10** Band heterotopia right temporal lobe.

**Fig11** Lissencephaly.
Lissencephaly: -

It includes a group of disorders characterized by a generalized paucity of gyral and sulcal formation known as agyria and pachgyria. Imaging studies reveal a range from the severe cases showing completely smooth brain with sulcal fissures being only the definable fissures to milder cases, in which a few shallow sulci surround broad flat gyri (fig.11).

Polymicrogyria: -

This refers to an abnormal appearance of the cortex with multiple abnormally small convolutions and too few sulci. A variety of etiologic factors has been linked to polymicrogyria including prenatal cytomegalovirus infection, cerebral ischaemia and genetic disorders [17,18]. It maybe unilateral or bilateral, symptomatic or asymptomatic and associated with other anomalies or isolated. On MR, polymicrogyria is seen as thickened cortex with poorly developed sulci and irregular margin of the cortical white matter junction (fig.12). In bilateral perisylvian polymicrogyria, the opercula are dysplastic and incomplete and sylvian fissure is wide and underdeveloped, with the sagittal images showing posterior extension of the sylvian fissure and apparent thickening of the cortex (fig.13).

Schizencephaly:

Schizencephaly describes grey matter lined clefts that extend through the entire cerebral hemisphere from the lateral ventricle to the cerebral cortex [19,20].
Schizencephalic clefts may be "closed" in which only a double layer of cortex is seen extending from the surface to the ventricle. Alternatively, they may be "open" in which there is wide communication between the subarachnoid space and ventricle through a broad hemispheric cleft (fig. 14). These clefts are lined by grey matter. A key feature is the presence of a ventricular dimple. This is almost always present in cases with closed or minimally open lips (fig. 15).

4) Neoplasms And Vascular Malformations:

MR has nearly 100% sensitivity for detecting epileptogenic neoplastic and vascular lesions [21]. Most epileptogenic neoplasms occur in the temporal lobe, in or adjacent to the cerebral cortex. Indolent tumours such as ganglioglioma, dysembryoplastic neuroepithelial tumours, and low grade gliomas are often associated with chronic intractable seizures (fig. 16). In the elderly population, cerebral metastasis is the most frequent neoplastic lesion associated with late onset seizures [22] (fig. 17).

Seizure is the principal clinical manifestation of vascular malformations, occurring in 24% to 69% of arterio-venous malformations [23] and 34% to 51% of cavernous haemangiomas (also referred to as cavernous angiomas or cavernous malformations) [24]. High flow vascular lesions appear as curvilinear signal voids (fig. 18). Cavernous haemangiomas demonstrate a stereotypical appearance of central hyperintensity due to haemoglobin products, surrounded by a hypointense rim resulting from haemosiderin (fig. 19).
5) Infections:

Seizures are common with acute cerebral infections (viral encephalitis and bacterial and aseptic meningitis) as well as those with brain abscesses, parasitic infections, aspergillosis, and other fungal infections. Patients developing acute central nervous infection before four years of age have a higher propensity to develop hippocampal sclerosis [4]. Chronic epilepsy however may result from post-inflammatory glial scarring.

In certain developing regions of the world, neurocysticercosis has been reported to be the most common cause of new onset partial seizures. Inflammation surrounding the cysticercosis manifests as acute seizure disorder. In the inflammatory stage provoked by the dying parasite, the cerebral lesions of cisticercus appear as small enhancing rings on CT and MR with variable degree of oedema in surrounding brain [25](fig.20). In vivo proton MR spectroscopy has been described in a case of a large cysticercus cyst [26]. The metabolites observed are lactate, succinate, acetate, alanine and an unassigned resonance at 3.3ppm.

In the developing world, tuberculomas account for 15-50% of the intra-cranial tumours seen [27]. Symptoms are often limited to seizures and correlates of intra-cranial pressure. The MR features of the individual tuberculoma depend on whether the granuloma is non-caseating or caseating with a solid center, or caseating with a liquid center [27]. The non-caseating granuloma is usually iso/hypointense on.
T1W and hyperintense on T2W images. These granulomas show homogeneous enhancement after injection of contrast agent. The caseating solid granulomas appear relatively isointense/hypointense on T1W images with isointense/hyperintense rim and isointense to hypointense on T2W images(fig.21). These lesions show rim enhancement on post-contrast T1W imaging. The granulomas with central liquefaction of caseous material appear centrally hypointense on T1W and hyperintense on T2W images and show rim enhancement after contrast administration(fig.22). MR Spectroscopy has been found to be specific for intracranial tuberculomas when combined with imaging. Intracranial tuberculomas are characterized by a spectral pattern that primarily involves long chain lipids, with a 0.9 to 1.6ppm peak range, associated with a virtual absence of all brain metabolites normally present [27](fig.23).

6) Neurocutaneous Syndromes (Phacomatosis):

Recurrent seizures are the most common and clinically important manifestation of Sturge-Weber syndrome, also known as encephalotrigeminal angiomatosis [28]. This syndrome consists of a facial port-wine nevus in the trigeminal nerve distribution, leptomeningeal angiomatosis, epilepsy, mental retardation, and other neurologic deficits. The brain involvement is usually unilateral with characteristic tram-tack gyriiform calcification appearing as linear low signal on MR. The involved hemisphere is atrophic, often with overlying calvarial thickening.

Tuberous sclerosis is an inherited systemic disease with prominent cutaneous and CNS manifestations with the classic triad of adenoma sebaceum, epilepsy, and mental handicap. The diagnostic findings include the presence of multiple sub-ependymal nodules and multiple cortical tubers.

7) Miscellaneous Abnormalities:

The pathologic mechanisms for post-traumatic seizures include deposition of tissue haemosiderin, which is a potent epileptogenic agent, and cortical gliosis [29]. Gliosis usually appears as a region of increased signal change on T2W images, often associated with volume
loss (fig.24). Haemosiderin appears hypointense on T2W weighted images and gradient images.

Beyond age 50, stroke is the most frequent cause of seizures [30]. Delayed - onset seizures after an acute stroke carry much greater risk of developing into chronic epilepsy [22].

Eclampsia is defined as the development of convulsions in pregnant women with hypertension and proteinuria. Studies of women with eclampsia disclose multiple foci of cortical and subcortical white matter edema, primarily in the occipital lobes. [31] (Fig.25).

![Figure 25](image.png)

Fig25) Thirty-two weeks pregnant women with eclampsia: Subcortical white matter oedema in both occipital lobes.

Perinatal / neonatal hypoxic-ischaemic insult is a common cause of seizures in neonates (Fig. 26).

![Figure 26](image.png)

Fig26) Right germinal matrix haemorrhage in a neonate having seizures.

Other less common causes of epilepsy include infantile hemiplegia and rasmussen's encephalitis.

Accurate diagnosis of the cause of seizure in a patient is crucial for finding an effective treatment. MRI has been shown to be highly sensitive and specific in identifying the underlying pathology in partial epilepsy. MRI may determine patient selection for surgery and directly affects the presurgical evaluation and operative strategy. Therefore MRI should be performed early to avoid unnecessary medication in patients with resectable intracranial mass lesions.

**BIBLIOGRAPHY:-**