



Original Article

Role of Magnetic Resonance Imaging in evaluation of Intractable Epilepsy

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ABSTRACT

Introduction: Intractable epilepsy is a significant neurological disorder characterized by failure to achieve sustained seizure control despite optimal antiepileptic therapy. Identification of an underlying structural lesion is crucial for appropriate management, particularly in patients being considered for surgical intervention. This study aims to evaluate the role of Magnetic Resonance Imaging (MRI) in detecting and characterizing epileptogenic lesions in patients with drug-resistant epilepsy.

Materials and Methods: This prospective study included 50 patients with clinical suspicion of intractable epilepsy referred to the Department of Radio-diagnosis at a tertiary care hospital in Ahmedabad, India, between August 2024 and August 2025. All patients underwent brain MRI using a dedicated epilepsy protocol, including high-resolution T1-weighted imaging, T2-weighted sequences, FLAIR, diffusion-weighted imaging, and coronal oblique sections perpendicular to the hippocampus. Imaging findings were analysed to identify structural abnormalities and correlate them with clinical presentation.

Results: MRI demonstrated a high diagnostic yield in detecting epileptogenic lesions. Structural abnormalities were identified in the majority of patients using dedicated epilepsy protocols. Mesial temporal sclerosis was the most common finding, characterized by hippocampal atrophy, increased T2/FLAIR signal intensity, loss of internal architecture, and ipsilateral temporal horn dilatation. Other detected abnormalities included focal cortical dysplasia, glial scars, encephalomalacia, low-grade neoplasms such as gangliogliomas and Dysembryoplastic neuroepithelial tumours, as well as sequelae of infections and vascular malformations. Advanced sequences such as 3D T1-weighted volumetric imaging, coronal oblique sections perpendicular to the hippocampus, and high-resolution T2/FLAIR images significantly enhanced lesion conspicuity.

Conclusion: MRI is the imaging modality of choice in the evaluation of intractable epilepsy due to its superior soft-tissue contrast and multiplanar capability. Dedicated epilepsy protocols enhance detection of subtle structural abnormalities, facilitate precise lesion localization, and aid in surgical planning. Accurate MRI assessment plays a pivotal role in optimizing treatment strategies and improving clinical outcomes in patients with drug-resistant epilepsy.

Keywords: Intractable epilepsy, Magnetic Resonance Imaging, Mesial temporal sclerosis, Epilepsy protocol MRI, Focal cortical Dysplasia, Drug-resistant epilepsy.

INTRODUCTION

Epilepsy is one of the most common chronic neurological disorders worldwide, affecting over 50 million people and posing a substantial burden on healthcare systems. Despite the availability of multiple antiepileptic drugs, approximately 20–30%

of patients continue to experience seizures despite optimal medical therapy. This condition, termed drug-resistant or intractable epilepsy, is defined by the International League Against Epilepsy as the failure of adequate trials of two appropriately chosen and tolerated antiepileptic medications to achieve sustained seizure freedom. In such patients, timely identification of an underlying structural substrate is crucial, as surgical intervention may offer the possibility of cure or significant seizure reduction.

Magnetic resonance imaging (MRI) has emerged as the imaging modality of choice in the evaluation of intractable epilepsy due to its superior soft tissue contrast, multiplanar capability, and lack of ionizing radiation. High-resolution MRI plays a pivotal role in detecting epileptogenic structural abnormalities such as mesial temporal sclerosis, focal cortical dysplasia, low-grade glioneuronal tumours, cavernous malformations, and other malformations of cortical development, and sequelae of prior infections or trauma. Among these, mesial temporal sclerosis remains the most common cause of drug-resistant focal epilepsy in adults, characterized by hippocampal atrophy and signal alteration on T2-weighted and fluid-attenuated inversion recovery sequences, and loss of internal hippocampal architecture.

The development of dedicated epilepsy imaging protocols and advances in technology—including high-field strength scanners (3T and above), thin-section volumetric imaging, high-resolution T2-weighted and FLAIR images, and coronal oblique sections oriented perpendicular to the hippocampal axis, susceptibility-weighted imaging, and diffusion tensor imaging—have substantially improved lesion detection rates. Moreover, functional techniques such as functional MRI (fMRI) and MR spectroscopy contribute to presurgical evaluation by aiding in localization of eloquent cortex and metabolic abnormalities, thereby enhancing surgical planning and reducing postoperative morbidity

MATERIALS AND METHODS

Patient selection:

The study comprised of 50 patients referred to department of radiodiagnosis at a tertiary care hospital in Ahmedabad with clinical suspicion intractable epilepsy over a period of 12 months. Patients with implanted electric and electronic devices, (heart pacemakers, especially older types), insulin pumps, implanted hearing aids, neurostimulators, intracranial metal clips, metallic bodies in eye, metallic hip replacements, sutures of foreign bodies were excluded from the study.

Image acquisition protocols:

Magnetic Resonance Imaging of Brain:

The patients were made to undergo a standard conventional contrast enhanced MRI brain on Siemens 1.5 T MRI. Brain MRI was performed using the following protocols: Plain sequences included sagittal T1 (Tse), Axial T1, Axial T2, Axial FLAIR, T2 Coronal, Diffusion weighted imaging (DWI) with high B values (2000-3000), Apparent diffusion coefficient (ADC), Susceptibility weighted imaged (SWI).

Harmonized Neuroimaging of Epilepsy Structural Sequences 3D T1W SPGR, 3D FLAIR, Coronal T1 inversion recovery (IR) obtained.

After Gadolinium (Magnevist) administered intravenously (0.2 ml/kg, 0.1mmol/kg) by hand injection: Axial 3D T1 was obtained.

RESULTS

SOCIODEMOGRAPHICS:

A total of 50 patients with clinically diagnosed intractable epilepsy were included in the study.

Age Distribution

The age of patients ranged from 6 to 52 years, with a mean age of 24.8 ± 10.6 years. The majority of patients belonged to the second and third decades of life (58%).

Age Group (years)	Number (n=50)	Percentage (%)
≤10	6	12%
11–20	14	28%
21–30	15	30%
31–40	9	18%
>40	6	12%

Table 1. Age Distribution of patients.

Gender Distribution

Out of 50 patients:

Male: 28 patients (56%)

Female: 22 patients (44%)

Male-to-female ratio was 1.27:1, showing a slight male predominance.

MRI Findings in Intractable Epilepsy (n = 50)

MRI detected structural abnormalities in 40 out of 50 patients (80%), while 10 patients (20%) had normal MRI findings (MRI-negative epilepsy).

MRI Finding	Number (n=50)	Percentage (%)
Mesial Temporal Sclerosis (MTS)	14	28%
Focal Cortical Dysplasia (FCD)	8	16%
Gliosis / Encephalomalacia	6	12%
Low-grade Tumors	4	8%
Neurocysticercosis (calcified/residual)	3	6%
Vascular Malformations (Cavernoma/ AVM)	3	6%
Tuberous Sclerosis Complex	1	2%
Coats Plus Syndrome	1	2%
Normal MRI (MRI-negative epilepsy)	10	20%

Table 2: MRI Findings in Intractable Epilepsy (n = 50)

Imaging Features of Common MRI Findings

Mesial Temporal Sclerosis (MTS) – 14 cases (28%)

Laterality:

Right-sided: 8 cases, Left-sided: 5 cases, Bilateral: 1 case

Imaging characteristics:

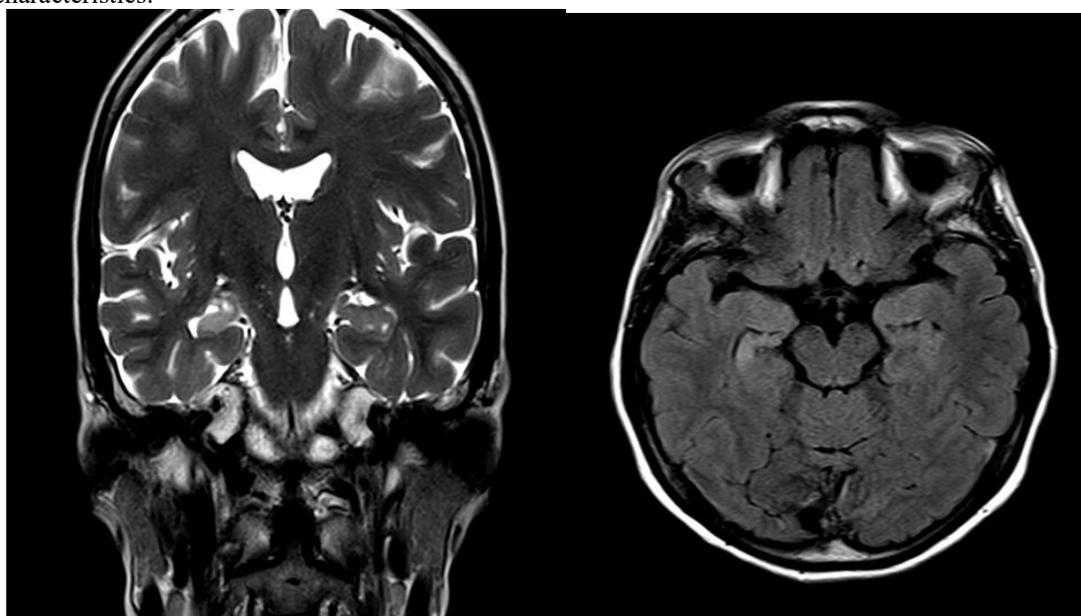


Image 1: Coronal T2W, Axial FLAIR of MRI brain showing Right Mesial Temporal Sclerosis.

Imaging characteristics:

Coronal T2W, Axial FLAIR images of temporal lobes show volume loss of right hippocampus with T2 and FLAIR hyperintense signals. There is loss of right hippocampal digitations. Minimal dilatation of ipsilateral temporal horn is noted.

Focal Cortical Dysplasia (FCD) – 8 cases (16%)

Most common location:

- I. Frontal lobe (5 cases)
- II. Temporal lobe (2 cases)
- III. Parietal lobe (1 case)

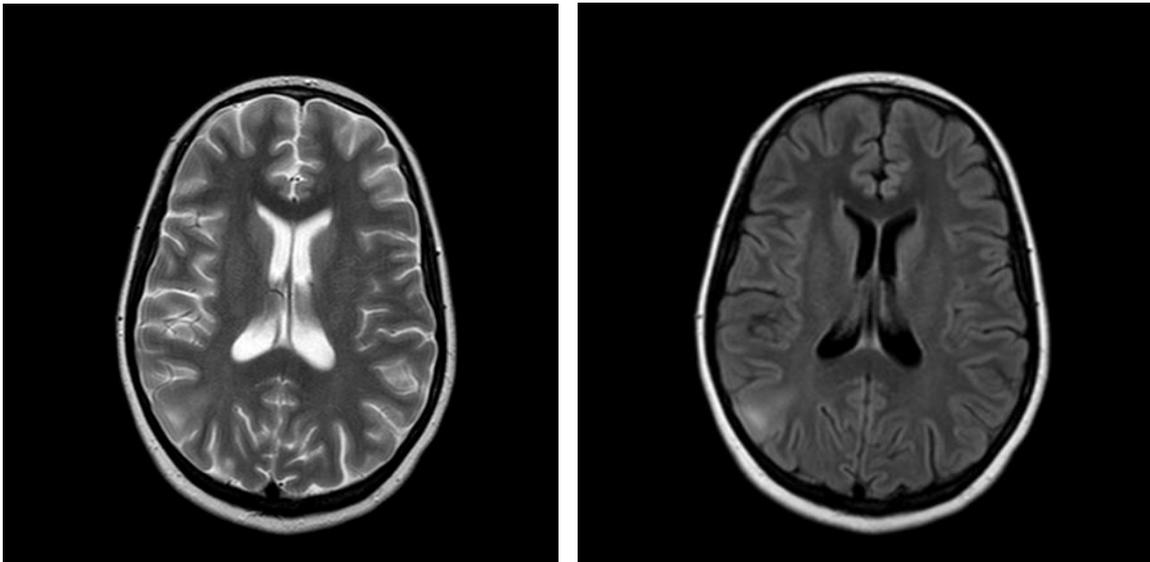


Image 2: Axial T2W, Axial FLAIR of MRI brain showing Focal Cortical Dysplasia (FCD) in right parietal region. Imaging features:

Axial T2W and FLAIR image shows a focal area of thickened cortex in right parietal region with blurring of the grey/white matter junction and abnormal increased signal on T2W and FLAIR sequences consistent with dysplastic cortex and extends from cortex to ventricle giving trans mantle sign.

3. Gliosis / Encephalomalacia – 6 cases (12%)

Etiology:

Post-traumatic: 3

Post-infective: 2

Perinatal insult: 1

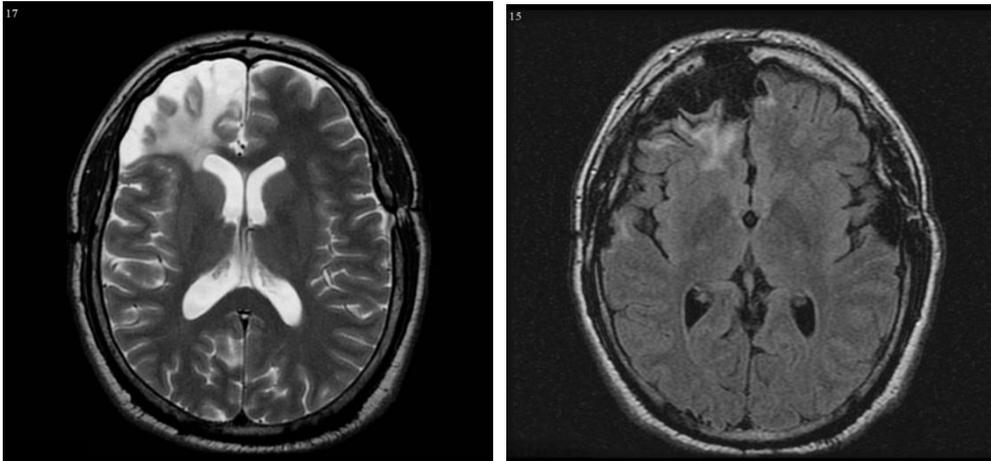


Image 3: Post traumatic Encephalomalacia with gliosis

Imaging features:

T2/FLAIR hyperintensity with volume loss involving right frontal lobe.

Ex vacuo dilatation of adjacent ventricle

4. Low-Grade Tumors – 4 cases (8%)

Types identified:

- i) Dysembryoplastic neuroepithelial tumour (DNET) – 2
- ii) Central neurocytoma – 1
- iii) Low-grade astrocytoma – 1

DNET (Dysembryoplastic neuroepithelial tumour)

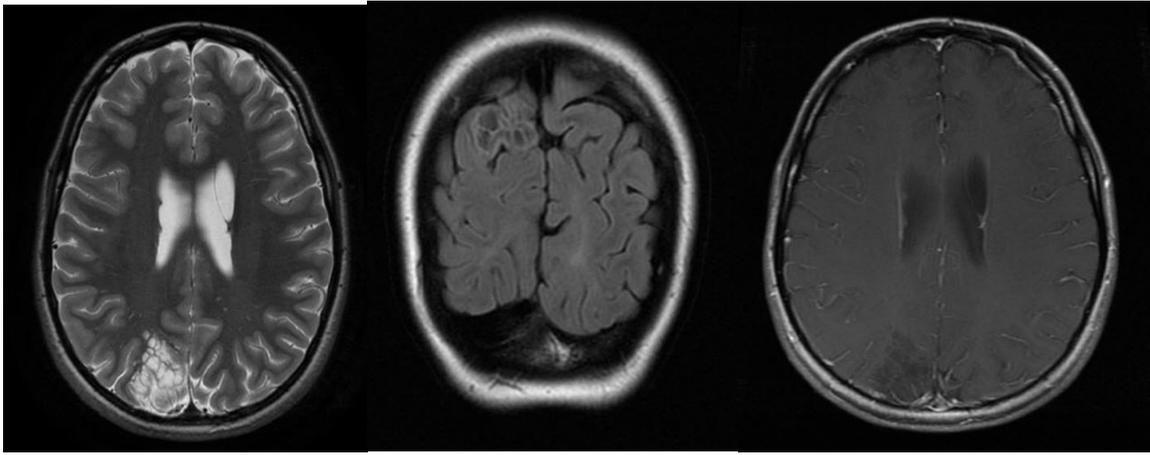


Image 4: Bubbly DNET in right parietal lobe

Imaging characteristics: Axial T2W image shows cortical based “bubbly” appearing mass in right parietal lobe. The coronal FLAIR image shows characteristic ring like hyperintense signal (bright rim sign). No perifocal oedema and no contrast enhancement are present.

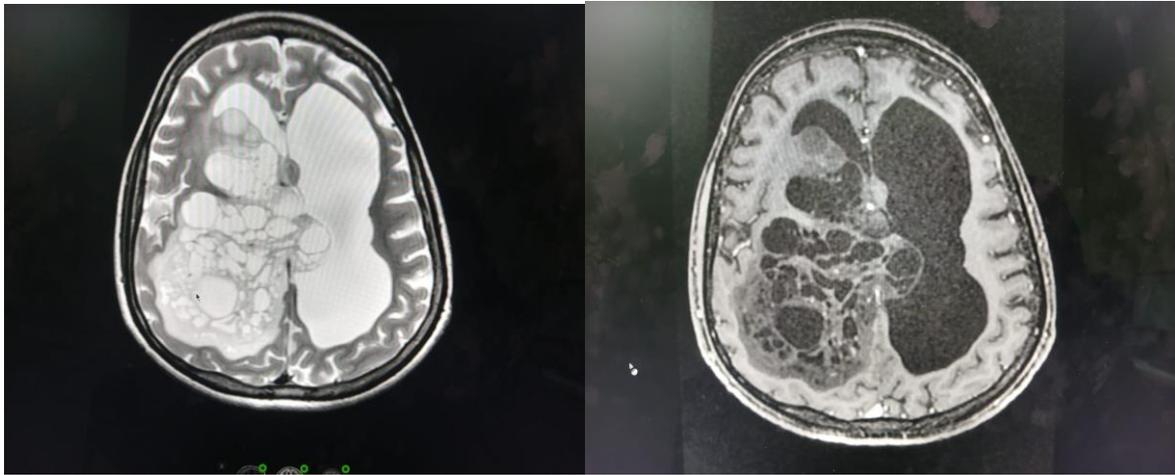


Image 5: Central Neurocytoma

Imaging features:

Large, Intraventricular, multilocular, bubbly/Swiss cheese like, complex cystic, T2W Heterogeneously hyperintense lesion in right lateral ventricle in the region of foramen monro, attached to septum pellucidum, displacing it to left lateral aspect and extends across midline causing compression over third ventricle with gross dilatation of bilateral lateral ventricle.

5. Neurocysticercosis – 3 cases (6%)

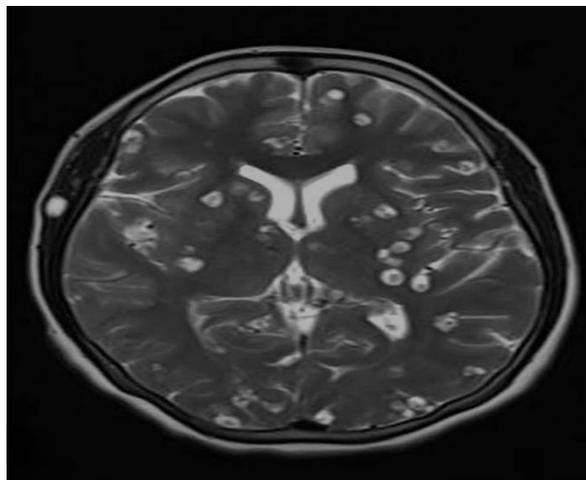


Image 6: Axial T2W shows multiple tiny neurocysticercosis.

Imaging features:

Axial T2W image show multiple cystic lesions with hypointense scolex within giving dot sign noted in bilateral cerebral hemisphere.

No perilesional oedema, s/o vesicular stage of neurocysticercosis.

6. Vascular Malformations – 3 cases (6%)

Cavernoma – 2 cases

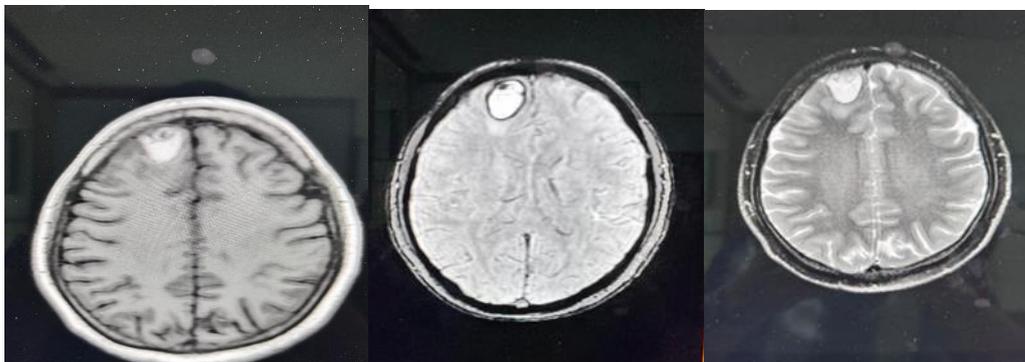


Image 7 : Zabramski Type I cavernous malformation

Imaging features: Well, defined altered signal intensity area noted in right frontal region, appears iso to hypointense on T1W, heterogeneously hyperintense on T2W images and shows blooming on SWAN image. Surrounding this lesion hyperintense T1W, T2W area noted which does not suppress on fat suppressed image, findings s/o cavernoma with surrounding haemorrhage. (According to Zabramski classification cerebral cavernomas – type I)

Arteriovenous malformation – 1 case

7. Tuberos Sclerosis Complex – 1 case (2%)

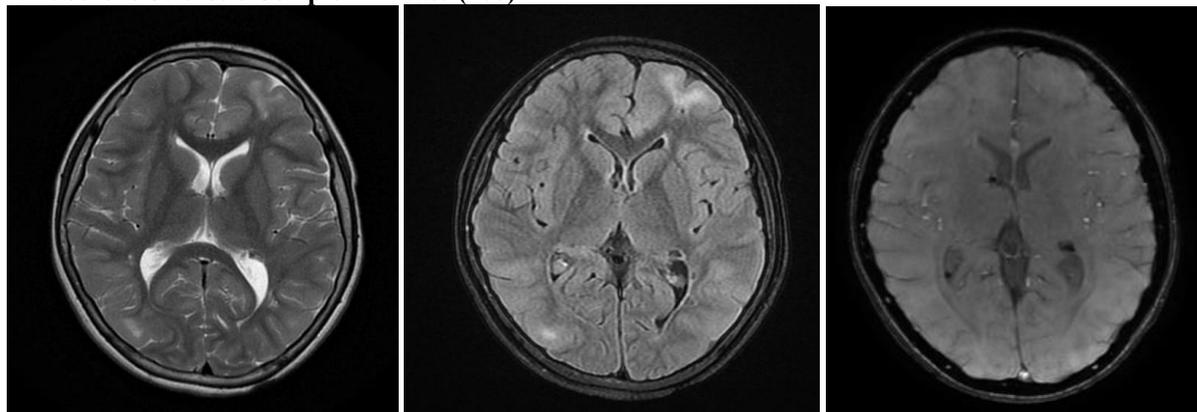


Image 8: Tuberos sclerosis

Imaging features:

Bilateral Multiple cortical tubers (T2/FLAIR hyperintense lesions)

Blooming on SWI images, along bilateral lateral ventricles s/o Subependymal nodules

Radial migration lines in white matter

No evidence of subependymal giant cell astrocytoma in this case

8. Coats Plus Syndrome(Cerebro-retinal microangiopathy with calcification and cysts-CRMCC) – 1 case (2%)

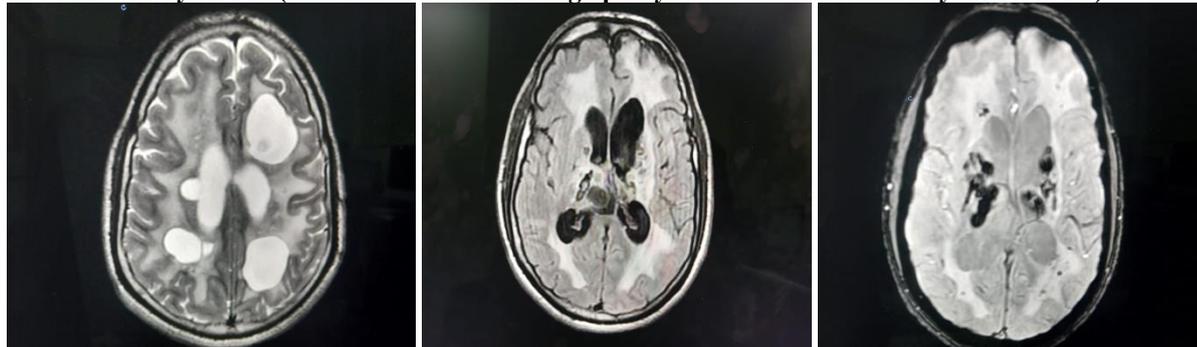


Image 9: Coats Plus Syndrome

Imaging features:

Intracranial calcifications (susceptibility blooming)

Leukodystrophy-like white matter changes (T2W, FLAIR hyperintensity)

Parenchymal cysts

Cerebral atrophy

Associated clinical history of retinal vascular abnormalities.

Summary of MRI Findings

MRI demonstrated abnormalities in 80% of cases, reinforcing its high diagnostic yield in intractable epilepsy.

Mesial temporal sclerosis (28%) remained the most common etiology.

Developmental malformations (including FCD and tuberous sclerosis) comprised a significant proportion.

Rare syndromic causes such as Coats plus syndrome were identified in a small subset.

MRI-negative epilepsy accounted for 20% of cases

DISCUSSION

Intractable epilepsy represents a significant clinical challenge, with early identification of structural abnormalities playing a crucial role in therapeutic decision-making. In the present study of 50 patients with drug-resistant epilepsy, MRI demonstrated structural abnormalities in 80% of cases, highlighting its substantial diagnostic yield in this population. This detection rate is comparable to previously reported studies evaluating dedicated epilepsy MRI protocols, which have documented lesion identification rates ranging between 70–85% in refractory epilepsy cohorts.

The MRI examination plays a key role for the screening of epileptogenic lesions in patients with epilepsy, this may be conducted either through MRI technique alone or in combination with some other imaging techniques such as MRI, MR spectroscopy. The performance of medical examination of epileptogenic lesions with these additional approaches provides an extremely valuable information about the epileptogenic lesions and its exact location in the brain, these additional approaches with MRI not only help the surgeons to perform epileptogenic lesional surgery but also help the radiologists to make their outstanding reports. In spite of the availability of these extraordinary functional imaging approaches but still the applicability of these approaches are limited. There have been several reasons but the most valid reason is the involvement cost in these techniques while applying for the analysis of anatomic epileptogenic lesions. Several investigators have reviewed all these additional techniques with MRI and all have concluded that these additional techniques are highly sensitive, more powerful, and accurate when performed with MRI.

In a study conducted by Abdelgawad et al. [1] on children with non-lesional epilepsy in Minya, Egypt, reported that volumetric MRI is extremely useful in the analysis of non-lesional pharmaco-resistant childhood epilepsy.

In another study, Samia et al. [2] from Nairobi, Kenya, reported that the conventional MRI has potential to detect multiple clinical comorbidities in childhood epilepsy.

Furthermore, Bernasconi et al. [3] reported that the structural MRI is very useful for the detection of non-invasive lesions in patients with epilepsy.

Moreover, Ponnatapura et al. [4] from Karnataka, India, reported that MRI with dedicated seizure protocol is best for the detection of epileptogenic lesions in one half of the new-onset seizures in epilepsy patients.

In another study, functional MRI found to be an important technique for the detection of hemodynamic and microstructural alterations in epileptogenic lesions.

John S. Duncan et al. [5] in their study of MRI in diagnosis and management of epileptomas demonstrates that eloquent cortex can be identified with functional MRI (fMRI), with cautions about the precise location and extent of critical cortex.

Demographic Correlation

The majority of patients in our study were young adults, with a mean age of 24.8 ± 10.6 years, and a slight male predominance (56%). Similar age distributions have been reported in studies evaluating refractory epilepsy, where the disease predominantly affects individuals in the second and third decades of life. The higher proportion of patients from rural and lower socioeconomic backgrounds may reflect delayed access to specialized neurological care, potentially contributing to chronicity and refractoriness.

MRI Abnormalities: Comparison with Literature

Mesial Temporal Sclerosis

Mesial temporal sclerosis (MTS) was the most common abnormality in our cohort (28%). This finding is consistent with global data identifying MTS as the leading cause of temporal lobe epilepsy and a major contributor to drug-resistant focal epilepsy.

The term MTS was introduced by Falconer et al. [6] to describe neuronal loss and gliosis in the anterior temporal lobes, as observed in surgical samples from a cohort of 100 patients receiving surgery for intractable TLE.

Cavanagh and Meyer [7] reported a higher frequency of MTS in adults who experienced early onset seizures before the age of 4, with a stronger association in those who had experienced status epilepticus. Building upon this, Babb and Brown [8] hypothesized that early seizures could lead to neuronal damage and reactive gliosis in the hippocampus. This sparked a debate in the medical literature regarding the relationship between MTS and epilepsy. Two main hypotheses were proposed. One suggested that early febrile seizures in childhood can cause damage to the hippocampus, thus being the cause of MTS, while the other proposed that a predisposed hippocampus in a child suffering prolonged febrile seizures was the fundamental cause.

Studies have shown that hippocampal atrophy and T2/FLAIR hyperintensity are the most reliable MRI markers of MTS. The predominance of unilateral involvement in our study mirrors findings from surgical epilepsy series, where unilateral MTS is more frequent than bilateral disease.

The relatively high prevalence of MTS in our cohort underscores the importance of thin-section oblique coronal imaging perpendicular to the hippocampal axis, particularly on 1.5T systems.

Focal Cortical Dysplasia

Focal cortical dysplasia (FCD) accounted for 16% of cases and was the second most common abnormality.

Focal cortical dysplasia is a malformation of cortical development, which is the most common cause of medically refractory epilepsy in the paediatric population and the second/third most common aetiology of medically intractable seizures in adults. Both genetic and acquired factors are involved in the pathogenesis of cortical dysplasia. Distinct classification schemes have been proposed to define the relevant imaging and histological features of FCD. [9]. Numerous classifications of the complex structural abnormalities of focal cortical dysplasia have been proposed - from Taylor et al. [10] in 1971 to the last modification of Palmini [11] classification made by Blumcke [12] in 2011. In general, three types of cortical dysplasia are recognized. Type I focal cortical dysplasia with mild symptomatic expression and late onset, is more often seen in adults, with changes present in the temporal lobe. Clinical symptoms are more severe in type II of cortical dysplasia usually seen in children. In this type, more extensive changes occur outside the temporal lobe with predilection for the frontal lobes. New type III is one of the above dysplasia with associated another principal lesion as hippocampal sclerosis, tumour, vascular malformation or acquired pathology during early life.

Detection rates vary depending on MRI field strength and protocol optimization. Subtle cortical thickening, blurring of the grey-white junction, and the trans mantle sign were key diagnostic features in our study.

Although higher field strength imaging (3T and above) has been shown to improve detection of subtle dysplasia, our findings demonstrate that optimized 1.5T protocols remain effective in identifying clinically significant cortical malformations.

Gliosis and Encephalomalacia

Post-traumatic, post-infective, and perinatal gliosis accounted for 12% of cases. Similar rates have been reported in developing regions, where infectious aetiologies and perinatal insults remain prevalent contributors to structural epilepsy. Chronic gliotic changes were characterized by volume loss and T2/FLAIR hyperintensity without diffusion restriction, consistent with established imaging descriptions.

Li W, Wang X, Wei X, Wang M et al [13] in their study of Susceptibility-weighted and diffusion kurtosis imaging to evaluate encephalomalacia with epilepsy after traumatic brain injury demonstrates that with the advent of new MRI techniques, the evaluation of mild to moderate posttraumatic encephalomalacia as an indicator of future epileptic activity is possible. SWI and DKI could be used to assess the microstructural changes around the encephalomalacia, and therefore be used to evaluate risk of developing epilepsy at 1 year.

Tumors and Vascular Malformations

Low-grade tumours (8%) and vascular malformations (6%) together comprised a significant surgically remediable group. Dysembryoplastic neuroepithelial tumours and gangliogliomas are well-recognized epileptogenic neoplasms, particularly in young patients. Their characteristic cortical location and minimal enhancement patterns were consistent with prior imaging studies.

First described in 1988 by C. Dumas-Duport, [14] Dysembryoplastic neuroepithelial tumours (DNTs) are benign lesions affecting young people and are clinically characterized by drug-resistant partial seizures and normal neurologic examination. Dumas-Duport et al define the clinical-radiologic criteria of DNT as follows: 1) partial seizures, with or without secondary generalization, beginning before the age of 20 years, 2) no neurologic deficit or stable congenital deficit, 3) cortical location of the lesion as best demonstrated by MR imaging, and 4) neither mass effect nor peritumoral oedema findings at imaging.

According to study by Fernandez C, et al. [15] some neuroradiologic features may be helpful to support the diagnosis of DNET: presence of “septations,” triangular pattern of distribution, and absence of contrast enhancement, cortical location, “multinodular bubble-like” morphology on T2/FLAIR, absence of significant oedema or mass effect.

Cavernoma and arteriovenous malformations demonstrated susceptibility blooming and flow voids, respectively, aligning with classical MRI features described in neuroradiology literature.

Neurocutaneous and Rare Syndromic Etiologies

One case each of tuberous sclerosis complex and Coats plus syndrome was identified. Although individually rare, recognition of such syndromic associations is critical due to their systemic implications. The imaging appearance of cortical tubers and subependymal nodules in tuberous sclerosis is well documented.

Kingswood et al.[16] in their study demonstrated that epilepsy was reported in (83.5%) of patients with tuberous sclerosis and demonstrates that major CNS manifestations of tuberous sclerosis is cortical tubers, subependymal nodules, subependymal giant cell astrocytoma.

Similarly, intracranial calcifications, cysts, and Leukodystrophy-like white matter changes are characteristic of Coats plus syndrome.

MRI-Negative Epilepsy

In our study, 20% of patients had normal MRI findings. Published data indicate that MRI-negative epilepsy can range from 15–40%, particularly when subtle cortical dysplasia or microstructural abnormalities are present. This highlights the limitations of conventional structural MRI and the potential role of advanced imaging techniques such as volumetry, post-processing analysis, diffusion tensor imaging, and functional imaging in selected cases.

Clinical Implications

The presence of an MRI-visible lesion is strongly associated with improved surgical outcomes in refractory epilepsy. Our study reinforces the pivotal role of dedicated epilepsy MRI protocols in:

Identifying surgically remediable lesions.

Localizing epileptogenic substrates.

Guiding presurgical planning

Reducing the proportion of MRI-negative cases. Even at 1.5T, optimized thin-section imaging significantly enhances diagnostic yield.

CONCLUSION

MRI is the imaging modality of choice in the assessment of intractable epilepsy due to its superior soft-tissue resolution and ability to detect subtle epileptogenic lesions. The use of dedicated epilepsy protocols enables precise identification and characterization of structural abnormalities responsible for seizure generation. MRI plays a crucial role in lesion localization, surgical candidacy assessment, and preoperative planning, particularly in temporal and extratemporal epilepsy. Accurate MRI evaluation contributes significantly to improved surgical outcomes, reduced seizure burden, and optimized patient management. Hence, MRI is an indispensable component of the comprehensive diagnostic and therapeutic approach to patients with drug-resistant epilepsy.

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