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Current Role of MRI in Detecting Epileptogenic lesion of Refractory Epilepsy with Positive EEG in Paediatric Period

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ABSTRACT

Background: Epilepsy affects about 1% of children, with 10–30% developing refractory epilepsy unresponsive to antiepileptic drugs (AEDs). Accurate localization of epileptogenic lesions is critical for management, and magnetic resonance imaging (MRI) is the gold standard due to superior resolution and absence of radiation.

Aim: This study aimed to evaluate the role of MRI in detecting epileptogenic foci in pediatric patients with refractory epilepsy and positive electroencephalography (EEG).

Methods: This cross-sectional observational study was conducted from July 2023 to May 2025 at Menoufia University Hospitals and affiliated centers. Sixty children aged 3–17 years with clinically and EEG-confirmed refractory epilepsy underwent standardized brain MRI. Imaging findings were correlated with demographic data, seizure type, EEG results, and AED use.

Results: MRI abnormalities were detected in 48/60 patients (80%). Common findings included encephalomalacia/gliosis (23.3%), demyelination (20.0%), cortical dysplasia (10.0%), and brain atrophy (10.0%). Lesions predominantly involved the white matter and frontal–parietal regions. A strong correlation was observed between EEG positivity and MRI abnormalities (p=0.000) with all EEG-positive patients showing structural lesions. Additionally, all patients receiving three or more AEDs had MRI-detectable abnormalities compared to 45.5% of those on two drugs (p=0.005).

Conclusion: MRI demonstrated high diagnostic yield in pediatric refractory epilepsy, showing strong agreement with EEG and AED resistance. It played a vital role in identifying epileptogenic lesions, supporting individualized treatment strategies, and guiding surgical decision-making.

Keywords: Pediatric epilepsy, Refractory epilepsy, MRI, EEG, Antiepileptic drugs.

INTRODUCTION

Epilepsy is one of the most frequently seen neurologic disorders within childhood. International League against Epilepsy defines epilepsy as; at least two unprovoked or reflex seizures > 24 h apart. Epilepsy affects 50 million people around the world and half of them start in childhood period [1]. Epilepsy affects 0.5–1% of children, and 10–30% of these children are refractory to medical anticonvulsant therapy [2]. Refractory epilepsy is also known as drug resistant epilepsy or pharmacoresistant epilepsy. Refractory epilepsy is diagnosed when there is failure of adequate trials of two tolerated and appropriately chosen and used antiepileptic drugs to achieve sustained seizure freedom. Epilepsy may be classified as acquired and congenital abnormalities [3].

Generally, diagnosis of seizure involves identifying the symptoms, clinical examination and clinical diagnosis of the cases, laboratory evaluation, EEG recording, lumbar puncture in some cases and neuro-imaging. MRI provides detailed evaluation of small lesion [4].

MRI is found to be the imaging modality of choice for epileptogenic lesion, because of non-utilization of radiation and providing detailed evaluation of small lesions as well as showing the underlying associated various structural brain abnormalities [5]. Therefore, this study aimed to highlight the role of MRI in detecting epileptogenic focus in children suffering from refractory epilepsy with positive EEG.

PATIENTS AND METHODS

This cross-sectional observational study was carried out in the MRI Unit, Radiodiagnosis Department, Shebin El Koum Military Hospital, Mit Khalaf Fever Hospital, and the Pediatric Neurology Department, Menoufia University Hospitals. The research work was carried out between July 2023 and May 2025.

Physicians and medical personnel referred patients, and they were enrolled based on pre-established inclusion and exclusion criteria. MRI of the brain was performed on 60 patients with refractory epilepsy. Diagnosis of epilepsy in children was made on clinical examination, assessment as per the International League Against Epilepsy (ILAE) 2017 criteria, and electroencephalography (EEG).

Inclusion criteria: Clinically diagnosed patients with refractory epilepsy based on EEG and clinical assessment, between the ages of 2–18 years, and of both sexes.

Exclusion criteria: Children with controlled epilepsy, children under 2 years and above 18 years, patients with MRI contraindications in the form of non-compatible implants or claustrophobia, and patients presenting with syncopal or hypoglycemic attacks,

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pseudo-seizures, or drug-induced seizures. Also, children with a history of recent head injury (within a week).

Clinical evaluation was done by taking detailed history and performing thorough clinical examination. The details obtained included the duration of illness, seizure type, secondary injuries due to seizures (e.g., head trauma), and any comorbid associated conditions. A provisional clinico-etiological diagnosis was then established.

For MRI study of the brain, informed consent was obtained from all patients or the guardians in case of children. The procedure, along with potential risks of contrast administration, was explained beforehand. MRI scans were performed on a Toshiba Excelart Vantage 1.5T scanner (Toshiba, Japan) with a standard head coil. Imaging was performed in all patients according to the institutional protocol, with additional advanced sequences performed when required. The MRI sequences included sagittal T1-weighted, axial T1-weighted, axial T2-weighted, axial 3D T1-weighted, axial FLAIR, coronal 3D FLAIR, and intravenous contrast-enhanced images as required. Sedation or general anesthesia was given in certain cases to facilitate examination. The MRI findings were correlated with EEG, computed tomography (CT), laboratory investigations, previous MRI studies, histopathological findings, and follow-up studies wherever possible.

Analysis of data was accomplished by saving MRI images as Digital Imaging and Communications in Medicine (DICOM) files and analyzing them on a dedicated workstation (Aze Virtual Place FujinRaijin 310). The imaging results were correlated with clinical and investigational findings. Subtypes of brain tumors were established through histopathology, follow-up, and laboratory investigations. All findings were recorded systematically, for example, MRI protocols used, clinical presentation, EEG findings, lesion location (temporal & extratemporal, or combined), and nonspecific age-related changes. Data were recorded in Microsoft Excel 2020.

Abnormal MRI findings were categorized into several categories. Congenital and developmental non-vascular abnormalities included atrophic brain changes, periventricular leukomalacia, focal cortical dysplasia (FCD), partial agenesis or thinning of the corpus callosum, Dandy–Walker malformation variant, non-communicating hydrocephalus, and leptomeningeal cysts. Brain tumors were counted individually. Vascular abnormalities included developmental venous anomalies, cavernomas, ischemic and hemorrhagic strokes, bilateral subdural hematomas, vasculitis, and suprasellar aneurysms. Sequelae of previous brain insults included gliosis, scarring, and encephalomalacia. Other categories included mesial temporal sclerosis (MTS), demyelinating disorders including multiple sclerosis (MS), infectious processes including encephalitis and brain abscesses, and miscellaneous findings including posterior reversible encephalopathy syndrome (PRES) and bilateral basal ganglia insults. Finally, age-related changes, including involutional and ischemic white matter changes, were also documented.

Ethical considerations: The study was approved by The Research Ethics Committee of Menoufia University (Approval No. 5/2023 RAD 13). Written informed consents were obtained from all participants or, in the case of minors, from their parents or legal guardians before enrollment. The consent process clearly outlined agreement to participate and to allow publication of anonymized data, with full assurance of confidentiality and privacy. All procedures were conducted in accordance with the ethical principles of the World Medical Association Declaration of Helsinki for studies involving human subjects.

Statistical analysis

Statistical software SPSS version 25 was used to conduct statistical analysis. Descriptive statistics including frequencies, percentages, means, and standard deviations were used to represent demographic and clinical variables of the study sample. Association between categorical variables was assessed using Fisher's exact test, which was used since the sample size was limited and data were not spread out. This analysis determined correlations between EEG findings and MRI lesions, seizure features and MRI status, number of antiepileptic drugs, presence of lesions, and neurological symptom distribution by lesion location. A p-value ≤ 0.05 was considered statistically significant.

RESULTS

The study population included 60 participants aged between 3 and 17 years, with a mean age of 8.70 ± 4.34 years, indicating a primarily pediatric cohort. Males were slightly more represented than females (56.7% vs. 43.3%). This balanced gender distribution minimizes bias related to sex-based neurological vulnerability (Table 1).

Table (1): Demographic and clinical characteristics of the study participants (n = 60)

Characteristic	Category	Frequency (n)	Percentage (%)
Age (years)	Mean ± SD	8.70 ± 4.34	
	Min – Max	3 – 17	
Gender	Male	34	56.7%
	Female	26	43.3%

MRI abnormalities were identified in 80% of patients, with demyelination, gliosis, encephalomalacia, cortical dysplasia and brain atrophy being common findings. Lesion sites were diverse, with white matter and the

frontal-parietal regions most frequently affected. Only 20% had no detectable lesions. This underscored the high rate of structural brain involvement in this patient group (Table 2).

Table (2): Distribution of MRI findings and lesion sites among study participants (n = 60)

Category	Finding / Site	Frequency (n)	Percentage (%)
MRI Finding	Normal	12	20.0%
	Brain atrophy	6	10.0%
	arachnoid cysts	6	10.0%
	Demyelination	12	20.0%
	Encephalomalacia/ Gliosis	14	23.3%
	subdural hygroma	2	3.33%
	hemorrhage	2	3.33%
	cortical dysplasia	6	10.0%
	Total MRI Findings	**60**	**100.0%**
Lesion Site	Nothing	12	20.0%
	White matter only	12	20.0%
	frontal lobe one side	4	6.67%
	frontal lobes both side	6	10.0%
	parietal lobes one side	2	3.33%
	Parietal lobes both side	6	10.0%
	Occipital lobe one side	6	10.0%
	Occipital lobes both side	4	6.67%
	Temporal lobe one side	6	10.0%
	Ventricular system	2	3.33%
	Total Lesion Sites	**60**	**100.0%**

A highly significant association was observed between positive EEG findings and MRI abnormalities (p = 0.000). All participants with positive EEGs showed MRI abnormalities, whereas 75% of those with normal MRIs had negative EEGs. This strong correlation suggests that EEG can be a reliable indicator of underlying structural pathology in this study (Table 3).

Table (3): Relationship between previous EEG findings and MRI results (n = 60)

Previous EEG Findings	MRI Abnormal	MRI Normal	Total	p-value
Negative EEG	4	12	16	0.000*
Positive EEG	44	0	44	
Column Total	48	12	60	

Fisher's Exact Test,* statistically significant.

Patients on three or more AEDs had a 100% rate of detectable MRI lesions, while those on two drugs showed fewer lesions (45.5%). This suggests a direct relationship between lesion burden and drug-resistant epilepsy, with higher AED use reflecting more severe or structurally evident disease (p = 0.005). **Table 4**

Table 4. Relationship between number of AEDs and MRI findings

No. of AEDs	MRI Lesion Detected	%	No Lesion Detected	%	Total	p-value
(two drugs)	10	45.5 %	12	54. 5%	22	0.00 5
(three drugs)	26	100.0 %	0	0.0 %	26	
(four/five drugs)	12(6+6)	100.0 %	0	0.0 %	12	
Total	48	80. 0%	12	20. 0%	60	

Fisher's Exact Test,* statistically significant

Table 5: Most common clinical symptoms by lesion site (n = 60)

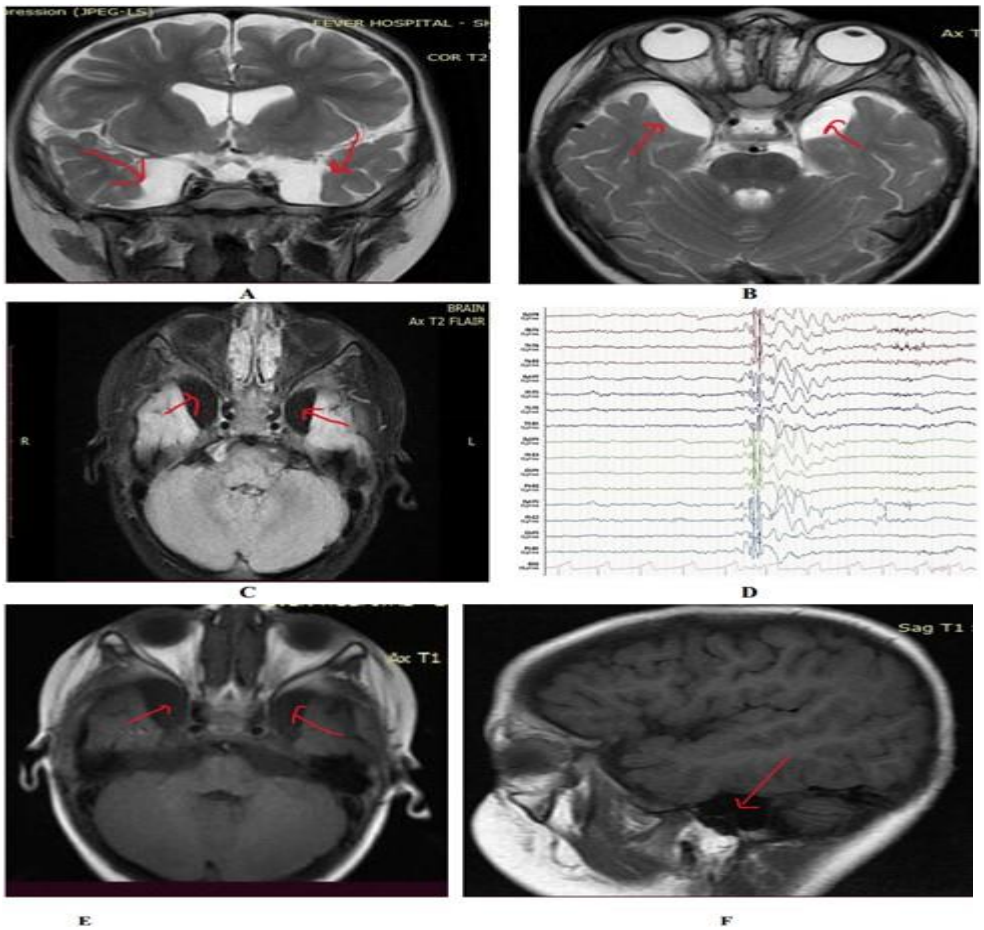
Symptom	frontal lobes both side	Parietal lobes both side	Temporal lobe one side	parietal lobes one side	Nothing Detected	frontal lobe one side	Occipital lobes	Ventricular system	White matter only	Total (Yes)	p-value
Spasm	6	4	6	2	0	6	6	0	6	36	0.001
Atonic Seizures	6	4	6	0	12	6	6	0	0	40	0.001
Loss of Consciousness	6	0	6	2	6	6	6	0	0	32	0.001
Staring	6	4	0	0	6	6	6	0	0	28	0.001
Falling	6	0	6	2	0	6	6	0	0	26	0.001
Muscle Rigidity/Tremor	6	0	6	2	12	6	6	0	0	38	0.001
Eye Movement	6	4	0	2	6	6	6	0	0	30	0.001
Tongue Biting	6	4	6	2	0	6	6	0	6	36	0.001
Convulsion	6	4	6	0	12	6	6	0	0	40	0.001
Muscle Weakness	6	4	0	0	12	6	6	0	0	34	0.001
Muscle Stiffness/Spasm	6	4	6	2	0	6	6	0	6	36	0.001
Headache/Confusion	6	4	6	2	6	6	6	0	0	36	0.001
Memory Loss	6	0	6	2	0	6	6	0	6	32	0.001
Personality Change	6	4	6	0	12	6	6	0	0	40	0.001

Fisher's Exact Test,* statistically significant.

Case presentations

CASE 1

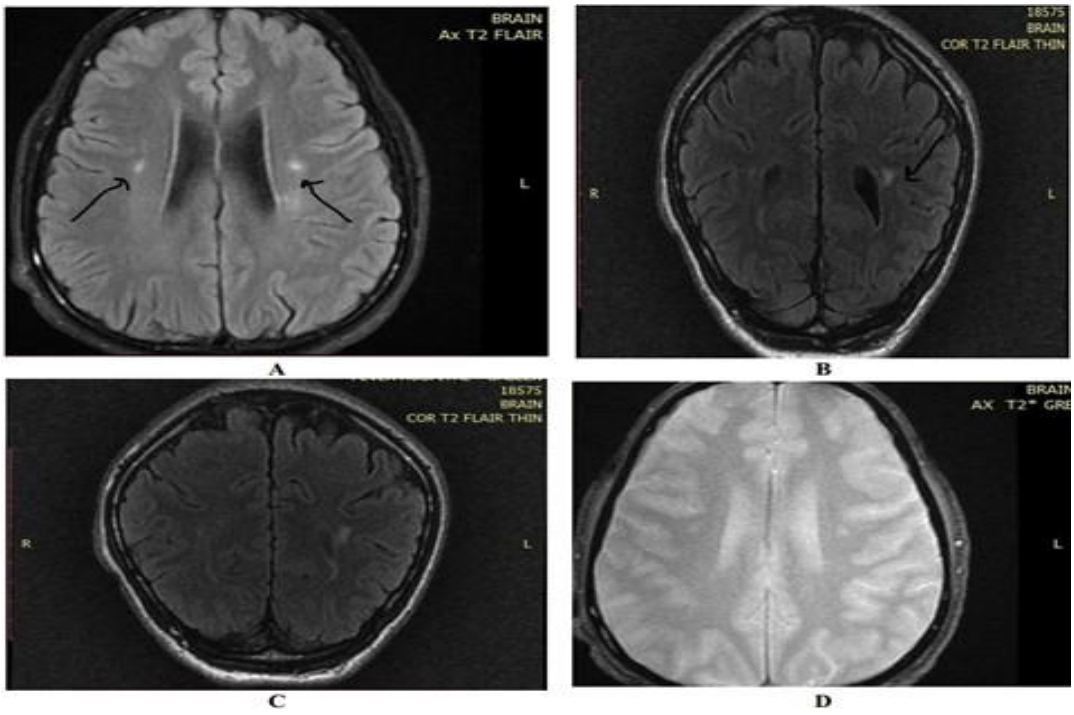
- Clinical presentation: A 3 -years old boy who had chronic seizures despite taking anti-seizure medication and abnormal EEG (D) (generalized poly spike-wave activity followed a few slow waves).
- **Imaging findings:**
  Coronal T2W (A), axial T2W (B) showed bitemporal hyperintense signal however axial FLAIR (C) showed hypo intense signal at bitemporal regions. Axial T1 (E) and sagittal T1 (F) showed hypointense lesion at the same area. As they follow CSF on all sequences, wall is very thin and displacement of surrounding tissues **….** Central atrophic changes with bitemporal arachnoid cysts

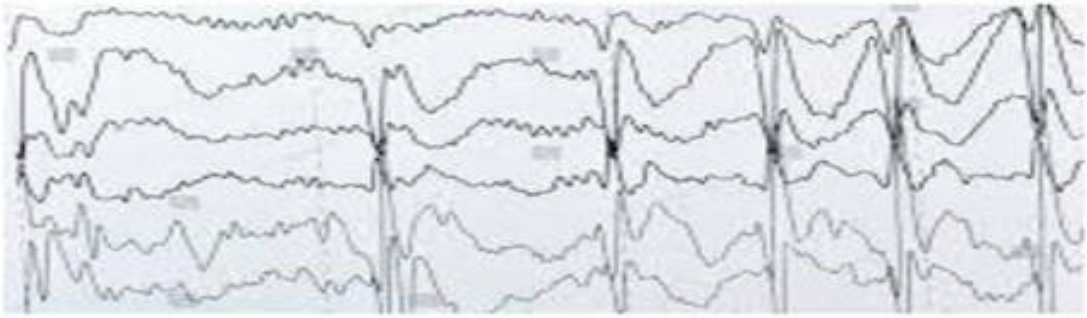


CASE 2

- Clinical presentation: male 17 years with delayed milestones and seizures and abnormal EEG (E).
- **Imaging findings:**

Axial FLAIR (A), coronal FLAIR (B & C)-Axial T2* GRADIENT (D); focal high signal intensity periventricular white matter plaques with two lesions spots BILATERL (black arrow) near to the ventricular system at parietal lobes … white matter demyelination



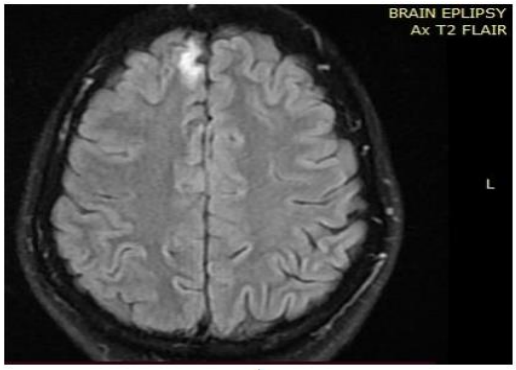


E

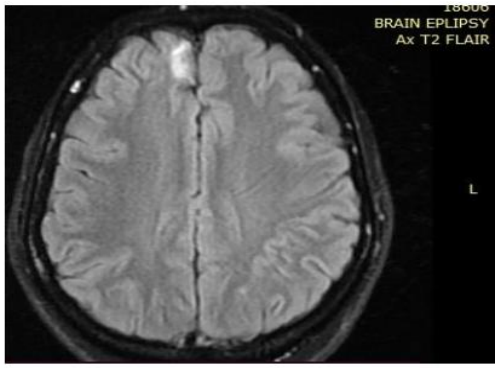
CASE 3

- Clinical presentation: female 13 years old with focal seizures, cognitive and learning difficulties, and sometimes weakness and abnormal EEG (H).
- **Imaging findings:**

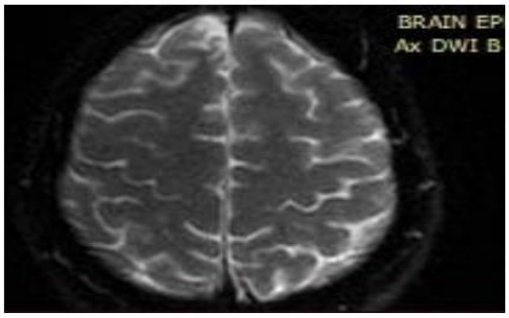
Axial FLAIR (A & B)… Axial DWI (C)…..Coronal T2 (D) …coronal FLAIR (E)…3D SAGITAL T2(F)…T1 (F**).** There was hyperintense lesion in right cortical frontal lobe in FLAIR sequences... Increased T2 signal intensity… 3D T2 showed blurring area at cortical rt frontal lobe…T1 showed cortical thickening at the same area…mostly right frontal cortical dysplasia.



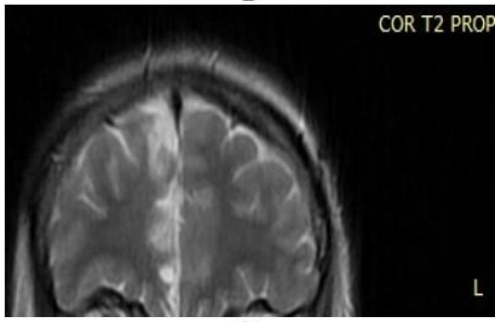
A



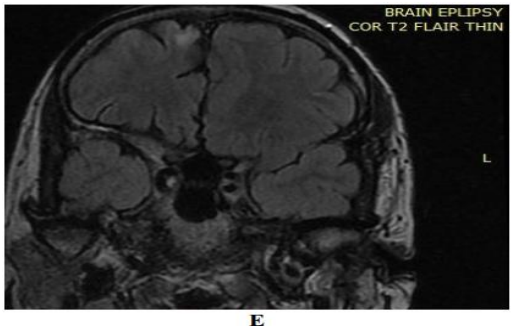
B



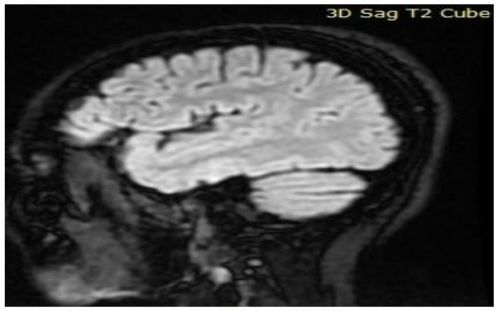
C



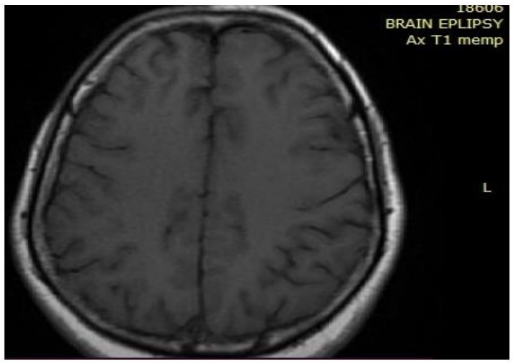
D



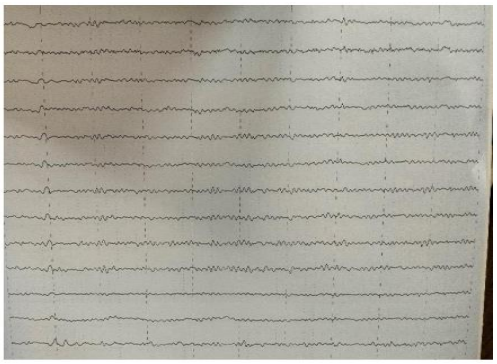
E



F



G



H

DISCUSSION

Epilepsy is one of the most prevalent childhood neurological conditions. Causes are either congenital or acquired and diagnosis depends on clinical assessment, EEG, laboratory tests, and neuroimaging, with MRI being the preferred modality due to high resolution and absence of radiation exposure, with potential to identify subtle epileptogenic lesions [6].

Our study was exclusively pediatric with a mean age of 8.7 years and a mild male predominance (56.7%). This is in line with **Nikodijevic *et al.*** [7], who also mentioned that refractory epilepsy occurring in childhood has a tendency to occur in late adolescence and late childhood and that the most significant etiologies are hippocampal sclerosis and other structural lesions. Similarly, **Chaurasia *et al.*** [8] discussed pediatric epilepsy with symptomatic causes such as infections and perinatal injury, further emphasizing that demographic age factors affect the epileptogenic substrate.

Our study showed that the majority of patients required polytherapy, where three AEDs were the most common, which is a sign of the drug-resistant group. This aligns with **Ahmed *et al.*** [9], who wrote that children who were brought in for surgical consideration have in general failed multiple antiepileptic therapies. In the same vein, **Gauci *et al.*** [10] documented a widespread presence of complex polytherapy in refractory cases in pediatric patients, suggesting heterogeneity of response due to genetic and structural causes. Conversely, **Tews *et al.*** [11] evaluated children after their first afebrile seizure and found that risk for recurrence grew higher with younger ages, as opposed to our study where participants were largely school-age and already drug-resistant. Moreover, **Ramli *et al.*** [12] pointed out that prevalence and imaging strategy vary greatly with age. However, the similarity of our data with large multicenter series such as the Epilepsy Surgery Commission [13] assures us that polytherapy and male predominance confirming demographic features of refractory pediatric epilepsy across cohorts.

Our study revealed that 80% of the children presented with MRI abnormalities, of which the most common were encephalomalacia/gliosis (23.3%), demyelination (20%), and cortical dysplasia (10%). Lesions were most frequently limited to white matter and frontal–parietal regions, and only 20% of the children had normal scans. The results emphasized the widespread structural pathology in pediatric refractory epilepsy.

Our study is consistent with **Nikodijevic *et al.*** [7], who reported a high MRI detection rate in structural etiologies, particularly hippocampal sclerosis, and confirmed that MRI significantly surpassed CT in localizing of epileptogenic lesions. **Chaurasia *et al.*** [8] also reported a similarly broad spectrum of abnormalities ranging from encephalomalacia to gliosis, demyelinating lesions and malformations showing that our described pattern of mixed etiologies is optimally in keeping with other pediatric series. Shaikh [14] confirmed this too by documenting cortical dysplasia, gliosis, hippocampal sclerosis, and vascular malformations as common pediatric MRI findings. Additional, evidence in support of our study comes from higher-end imaging. **Ahmed *et al.*** [9] demonstrated that high-field 3T MRI was capable of detecting subtle focal cortical dysplasias missed and **Radhakrishnan *et al.*** [15] demonstrated that re-imaging utilizing optimized protocols disclosed further cortical abnormalities not apparent on standard MRI. Similarly, **Bernasconi & Bernasconi** [16] emphasized the role of advanced MRI coils and techniques for reducing false negatives, emphasizing technology as central to improved lesion detection. Concurrently, there were also some variations. For example, **Kim** [17] documented a pediatric series wherein MRI was also negative, but stereo-EEG later revealed hidden epileptogenic networks a finding that contrasted with our very high positive detection rate. **Gauci *et al.*** [10] also highlighted that a large proportion of pediatric refractory patients remain MRI-negative, particularly for subtle signal FCD, and suggested that our signal difference could be explained by selection bias or application of more sophisticated imaging. **Alshafai *et al.*** [18] also reported MRI to be less sensitive for astrocytic inclusion detection compared to cortical dysplasia detection, noting that radiological yield is affected by histopathology.

Our study demonstrated a statistically significant correlation between EEG and MRI findings: All the patients with positive EEG had MRI lesions, and 75% of patients with normal MRI had negative EEG ($p = 0.000$). Such high concordance is evidence of the predictive value of EEG for structural pathology in children with refractory epilepsy. Our study concurs with **Nikodijevic *et al.*** [7], who indicated that focal epileptiform EEG abnormalities tended to correlate with MRI lesion sites, thus enhancing presurgical assessment.

Likewise, **Ahmed *et al.*** [9] illustrated that EEG and video-EEG directed focused MRI re-imaging led to the detection of concordant lesions and enhanced surgical planning. **Tews *et al.*** [11] also confirmed that EEG patterns in conjunction with good MRI results were more predictive of the development of epilepsy than either test alone.

Shaikh [14] and the Epilepsy Surgery Commission [13] also confirm that our observation that EEG abnormalities are strongly correlated with MRI lesions in focal epilepsies and that concordance is critical for

surgical candidacy. However, **Carrette & Stefan** [19] showed that co-registration of MRI and MEG had better detection of epileptogenic areas compared to EEG, while **Alshafai *et al.*** [18] introduced higher EEG–MRI–MEG concordance in cortical dysplasia compared to astrocytic inclusion. **Radhakrishnan *et al.*** [15] also proved that combining EEG, MEG, and MRI greatly enhanced diagnostic yield.

Not all reports are completely concordant, however **Chaurasia *et al.*** [8] found that although most EEG-abnormal patients had corresponding MRI lesions, normal EEG could not reliably exclude structural pathology, which was slightly different from our own experience of having very strong parallelism. **Gulomov *et al.*** [20] also found mismatches in some pediatric epilepsy syndromes e.g., normal EEG but abnormal MRI, or abnormal EEG but normal MRI suggesting that joint interpretation is required.

In addition, **Kim *et al.*** [17] highlighted EEG–MRI correlation limitations for MRI-negative cases, in which localization of epileptogenic zones was possible only through invasive stereo-EEG.

We found that there was significant correlation between heightened AED burden and MRI abnormalities because all three or more AED-treated patients showed detectable lesions. This implies that structural disease severity can be the cause of pharmacoresistance.

Our study is supported by **Chaurasia *et al.*** [8] and **Nikodijevic *et al.*** [7], whose studies revealed high rates of MRI abnormalities in intractable or multi-drug-resistant patients, particularly hippocampal sclerosis and gliotic lesions. Similarly, **Shaikh *et al.*** [14] and **Gulomov *et al.*** [20] revealed that structural lesions have a high correlation with refractory epilepsy and the use of multiple AEDs without full seizure control.

Bernasconi & Bernasconi [16] also confirmed higher detection rates of lesions in drug-resistant patients and hence our result. However, **Ahmed *et al.*** [9] showed that AED resistance can be present even with an initially negative MRI since small lesions only appear on specialized high-resolution imaging. **Gauci *et al.*** [10] also emphasized heterogeneity of AED-resistant epilepsy in which a few patients are MRI-negative despite clinical drug resistance. In like manner, **Kim *et al.*** [17] documented drug-resistant children with non-lesional MRI, once again suggesting that despite our result being generally consistent, structural lesions are far from the sole cause of inordinate AED burden.

LIMITATIONS: It was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. MRI scans were performed on a 1.5T system without advanced techniques such as 3T or functional imaging, which could have reduced sensitivity for subtle lesions. Histopathological confirmation was not available for most cases, restricting definitive lesion characterization. In addition, the study design was cross-sectional, preventing assessment of long-term outcomes or the predictive value of MRI findings for treatment response. Finally, selection bias may have been introduced by including only EEG-positive refractory patients, which may overestimate the true diagnostic yield of MRI.

CONCLUSION

MRI was valuable in evaluating pediatric refractory epilepsy, especially when EEG reveals epileptogenic activity. In this study, MRI identified structural abnormalities in 80% of cases, most often in white matter and frontal-parietal regions. A strong correlation between EEG positivity and MRI findings confirmed MRI's value in localizing epileptogenic foci. Clinical features such as spasms, tongue biting, stiffness, memory loss, and falls were significantly linked to MRI lesions serving as useful clinical indicators. More use of antiepileptic drugs was also related to lesion presence and is indicative of more disease severity. In general, MRI is crucial for identifying structural pathology, directing individualized therapy, and informing surgical planning for better outcomes in children.

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