ORIGINAL ARTICLE

Role of MRI in Epilepsy: A Retrospective Study at POF Hospital, Wah Cantt

Sobia Jaweed Raza[1], Madiha[2], Afreen Anjum[3], Muhammad Farhan Ul Huq[4], Beenish Suhail[5], Junaid Ur Rehman[6]

ABSTRACT

Objective: To establish the role of magnetic resonance imaging (MRI) in the management of epilepsy and to determine the proportion of MRI brain findings in young patients with epilepsy.

Study Design: Retrospective cross-sectional descriptive study.

Place and Duration of Study: Department of Diagnostic Radiology, POF Hospital Wah Cantt, from January 1, 2025 to February 28, 2025.

Materials and Methods: A total of 172 patients were selected by non-probability consecutive sampling. MRI brain images, obtained using the epilepsy protocol, from patients aged 1 to 30 years with a clinical history of epilepsy were retrospectively retrieved from the hospital Picture Archiving and Communication System (PACS) between June 1, 2018, and May 31, 2024. Patient age, gender, and MRI findings were recorded on a predesigned proforma. Data were analyzed using SPSS version 23.0. Age was expressed as mean ± standard deviation, while gender and MRI findings were presented as frequency and percentage. For abnormal MRI findings and the two most common abnormalities 95% confidence interval (CIs) were calculated.

Results: Out of 172 patients, 51.2% were males and 48.8% females, with a mean age of 18 ± 7.29 years. Structural brain abnormalities were detected in 94 patients (54.7%, 95% CI: 47.2%–61.9%), most commonly white matter hyperintensities (15.1%, 95% CI: 10.5%–21.2%) and mesial temporal sclerosis (14.5%, 95% CI: 10.0%–20.6%).

Conclusion: Dedicated MRI brain epilepsy protocol serves as a first-line neuroimaging modality in epilepsy management, as it allows accurate detection of structural lesions that can influence treatment decisions.

Keywords: *Epilepsy, Magnetic Resonance Imaging, Seizure.*

Introduction

Epilepsy is a chronic disorder characterized by a consistent propensity for recurrent and unprovoked seizures, leading to neurobiological, cognitive, psychological, and social problems.[1, 2] It is the most common neurological disorder in the world, accounting for 1% of the global disease burden. According to the WHO, 50 million people worldwide are suffering from epilepsy.[3] The prevalence of epilepsy in Pakistan is 9.99 per 1,000.[4] It is more common in males than females.[4] Its incidence rises until the age of 20, and then declines.[3] Its prevalence is higher in developing countries due to the high incidence of endemics, perinatal complications, and poor healthcare facilities.[5,6]

Early detection of the cause of epilepsy is crucial, especially in young patients as each seizure increases the risk of injury to the developing brain, resulting in permanent behavioral and cognitive disorders.[7] Routine MRI brain sequences (T1, T2 weighted spin echo and Fluid Attenuated Inversion Recovery FLAIR) may miss subtle epileptogenic lesions due to limited slice thickness and suboptimal orientation. However dedicated epilepsy protocols SPACE sequence, (Sampling Perfection with Application optimized Contrast using different flip angle Evolution) provide superior grey white matter contrast, better delineation of cortical architecture, sulcal patterns and multiplanar reconstruction without loss of resolution. Thus, this protocol significantly improves lesion detection and localization and provides help in early diagnosis, management, surgical planning and prognosis.[8]

In Pakistan, very limited local literature is available describing the role of MRI in epilepsy and spectrum

[1,2,4,6]*Department of Radiology*
Wah Medical College, Wah Cantt
[3]*Department of Radiology*
Akhtar Saeed Medical College, Islamabad
[5]*Department of Radiology*
Islamabad Medical and Dental College, Islamabad
Correspondence:
Dr. Sobia Jaweed Raza
Senior Registrar Radiology
Wah Medical College, Wah Cantt
E-mail: sobiajawwad@gmail.com

Received: June 13, 2025; Revised: December 15, 2025
Accepted: December 24, 2025
https://doi.org/10.57234/jiimc.december25.2543

the MRI brain findings in young epileptic patients. This gap in local data limits clinicians' ability to make evidence-based decisions for early diagnosis and management. This study aimed to establish the role of MRI with dedicated epilepsy protocol in epilepsy by determining the proportion of structural brain abnormalities in young patients.

Materials and Methods

This retrospective cross-sectional study was conducted in the Radiology Department of POF Hospital Wah Cantt from January 1, 2025 to February 28, 2025 over a period of 2 months after obtaining ethical committee approval (Letter no: IRB/POFH/01-2025/RADIOLOGY/03, dated 01.01.2025). As this study involved retrospective review of preexisting data, informed consent was not required. The data were securely stored to ensure confidentiality and patient identifiers were removed. The minimum required sample was calculated as 15 by using the WHO formula with 95% CI (Z= 1.96), d= 0.05 and a prevalence of epilepsy 9.99 per 1000 [4] however a total of 172 patients were included in the study by non-probability consecutive sampling to enhance study reliability. MRI brain images obtained using the epilepsy protocol from patients aged 1 to 30 years with a clinical history of epilepsy were retrospectively retrieved from the hospital Picture Archiving and Communication System (PACS) between June 1, 2018 and May 31, 2024 over a period of 6 years. Younger patients were prioritized because epilepsy is more common in childhood. MRI brain scans of patients with clinical history of epilepsy older than 30 years or those with a history of seizures due to conditions other than epilepsy were excluded from the study. MRI brain scans without a dedicated epilepsy protocol were also excluded.

After fulfilling the inclusion and exclusion criteria, a total 172 MRI images of the patients were selected. All patients underwent MRI brain on a Siemens Magnetom Aera 1.5T. The dedicated epilepsy protocol used in our hospital includes Sampling Perfection with Application optimized Contrast using different flip angle Evolution (SPACE) T1-weighted sagittal, T2-weighted sagittal and T2-weighted dark fluid sagittal images with MPR planning, ep2d-diff-3scan-trace, T2-SWI axial and contrast-enhanced T1WI when needed. All MRI images were analyzed by a senior radiologist with 15 years of experience to identify brain abnormalities. Findings were recorded on the proforma as the presence or absence of MRI-evident structural brain abnormalities along with the patient's age, gender, and clinical history. If any structural abnormality was present, its complete name and description were recorded according to predefined operational MRI criteria derived from established neuroimaging literature (Appendix A).

The data were entered and analyzed using SPSS version 23. Patient age was expressed as mean ± standard deviation, while gender and MRI brain findings were reported as frequencies and percentages. For abnormal MRI findings and the two most common abnormalities 95% confidence interval (CI) were calculated.

Results

A total of 172 patients were included in the study. The mean age of the patients was 18 ± 7.29 years (median: 18; mode: 30) with an age range of 1-30 years. There were 88 males (51.16%) and 84 females (48.84%) (Table I).

A total of 94 patients had structural brain abnormalities on MRI, giving a frequency of **54.7% (95% CI: 47.3–62.0%).** The common findings were white matter hyperintensities (WMHs) (n = 26; 15.1%, 95% CI: 10.5%–21.2%) and mesial temporal sclerosis (MTS) (n = 25; 14.5%, 95% CI: 10.0%–20.6%).

The other findings were focal cortical dysplasia (FCD) type II (n = 4; 2.33%), pachygyria (n = 2; 1.16%), grey matter heterotopia (GMH) (n = 1; 0.58%), encephalomalacia (n = 8; 4.65%), enlarged amygdala (n = 3; 1.74%), porencephalic cyst (n = 2; 1.16%), ulegyria (n = 2; 1.16%), periventricular leukomalacia (PVL) (n = 2; 1.16%), arachnoid cyst (n=6; 3.49%), chiari I malformation (n=2; 1.16%), chiari II malformation (n=2; 1.16%), intracranial lipoma (n=1; 0.58%), vascular malformations (n = 2; 1.16%), including arteriovenous malformation (AVM) and developmental venous anomaly (DVA), and atrophy related causes including cerebral atrophy (n = 3; 1.74%), cerebellar atrophy (n = 1; 0.58%) and hemicerebral atrophy (n = 2; 1.16%) (Table II).

Discussion

In this retrospective cross-sectional study 54.7% of patients had abnormal findings on MRI. The most common findings were white matter hyperintensities (WMHs) and mesial temporal

https://doi.org/10.57234/jiimc.december25.2543

Table I: Demographic Characteristics of Study Participants

Variable	Values
Mean age	18 ± 7.29
Median age	18
Mode	30
Age Range	1- 30 years
Male	88 (51.16%)
Female	84 (48.84%)

Table II: Distribution of MRI Findings in Patients with Epilepsy (n = 172)

MRI Finding	n (%)	95% CI
Normal MRI brain	78 (45.3%)	—
MRI with structural brain abnormality	94 (54.7%)	47.3–62.0
White matter hyperintensities (WMHs)	26 (15.1%)	10.5–21.2
Mesial temporal sclerosis (MTS)	25 (14.5%)	10.0–20.6
Malformations of cortical development (MCD)		
Focal cortical dysplasia (FCD) type II	4 (2.33%)	—
Pachygyria	2 (1.16%)	—
Grey matter heterotopia (GMH)	1 (0.58%)	—
Other causes		
Encephalomalacia	8 (4.65%)	—
Enlarged amygdala	3 (1.74%)	—
Porencephalic cyst	2 (1.16%)	—
Ulegyria	2 (1.16%)	—
Periventricular leukomalacia (PVL)	2 (1.16%)	—
Arachnoid cyst	6 (3.49%)	—
Chiari I	2 (1.16%)	—
Chiari II	2 (1.16%)	—
Intracranial lipoma	1 (0.58%)	—
Vascular malformations		
AVM	1 (0.58%)	—
DVA	1 (0.58%)	—
Atrophy-related causes		
Cerebral atrophy	3 (1.74%)	—
Cerebellar atrophy	1 (0.58%)	—
Hemicerebral atrophy	2 (1.16%)	—

sclerosis (MTS). The mean age of patients was 18 years with almost equal gender distribution.

Literature review shows that studies on childhood epilepsy typically report abnormal MRI findings in around 40% of cases,[9,10] our slightly higher proportion (54.7%) may be explained by the fact that we considered not only abnormalities directly causing epilepsy but also those associated with it thereby providing a comprehensive picture of

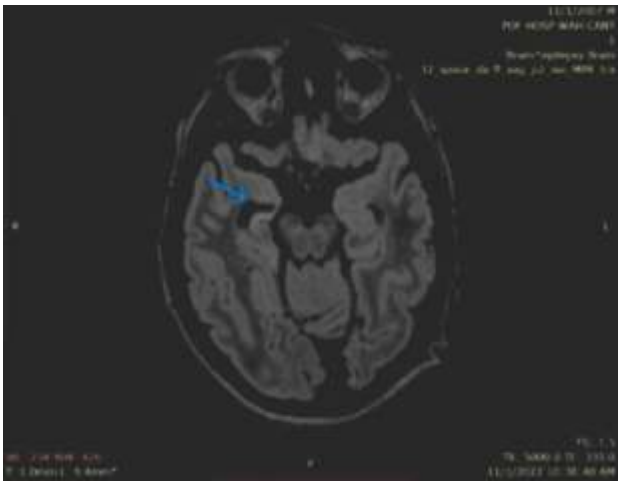

Fig 1.Right mesial temporal sclerosis evident as hippocampal volume loss with mildly dilated temporal horn of lateral ventricle (arrow)

Appendix A

Table: Operational Definitions of Structural MRI Abnormalities

structural brain changes and highlights the clinical value of MRI in detecting both overt epileptogenic lesions and subtle abnormalities that may influence disease course, management and prognosis.

In our study, WMHs were the most common MRI abnormality in epileptic patients. Similar findings have been reported in literature showing higher WMH burden in epileptic patients as compared to healthy controls.[11] WMHs in epilepsy are believed to result from chronic white matter hypoperfusion or blood-brain barrier damage during fits.[12] Thus WMH in young epileptic patients should not be taken as incidental findings although their prognostic value remains uncertain, their higher occurrence warrants future studies focusing on relationship between WMH, seizure outcome, cognitive decline and treatment response.

Mesial temporal sclerosis (MTS) and malformations of cortical development (MCD) that include FCD type II, pachygyria and GMH were also frequent findings in MRI brain of patients in our study. Literature review shows that MTS is the most common structural abnormality associated with focal epilepsy, particularly temporal lobe epilepsy (TLE) (Fig 1).[13] MRI epilepsy protocol (SPACE sequences) remains the gold standard for detection of MTS and MCD due to improved grey white matter differentiation and multiplanar reconstruction.[14] Both MTS and FCD are surgically treatable

Abnormality	MRI-Based Operational Definition	Reference no.
White Matter Hyperintensities (WMHs)	T2WI and FLAIR hyperintense foci, showing no diffusion restriction, located in deep and periventricular white matter	11
Mesial Temporal Sclerosis (MTS)	Hippocampal atrophy, T2WI and FLAIR hyperintensity and widening of the temporal horn	13
Focal Cortical Dysplasia (FCD Type II)	Cortical thickening, blurring of the grey–white junction with radial hyperintensity from cortex to ventricle on T2WI sequences	15
Pachygyria	Cortical thickening with broad and reduced number of gyri and shallow sulci; abnormal grey –white differentiation on T1WI and T2WI sequences.	17
Grey Matter Heterotopia (GMH)	Nodules or bands of grey matter located along lateral ventricles or deep white matter, isointense to cortex on all sequences.	17
Malformations of Cortical Development (MCD)	Group of structural cortical abnormalities from disord ered neuronal migration, including FCD, pachygyria and GMH	17
Encephalomalacia	CSF signal intensity areas with volume loss and surrounding gliosis seen as T1WI hypointensity and T2WI and FLAIR hyperintensity, replacing brain parenchyma	18
Enlarged Amygdala	Amygdala enlargement with intact architecture and with or without T2WI and FLAIR hyperintensity.	19
Porencephalic Cyst	Well defined CSF signal intensity cyst within brain parenchyma communicating with ventricle or subarachnoid space and adjacent gliosis.	20
Ulegyria	Cortical thinning and scarring in deep sulci with preserved gyri	20
Periventricular Leukomalacia (PVL)	Periventricular T2WI /FLAIR hyperintensity with loss of periventricular white matter volume	20
Intracranial lipoma	Fat signal intensity mass on all sequences	20
Cerebral Atrophy	Generalized or regional reduction in cortical volume with widened sulci and ventriculomegaly, assessed visually, usually no altered signal intensity on T1WI and T2WI images.	20
Cerebellar Atrophy	Volume loss of cerebellar hemispheres or vermis with prominent folia confirmed on sagittal and axial sequences.	20
Hemicerebral Atrophy	Unilateral cerebral volume loss with ipsilateral ventricular dilation, assessed visually.	20
Vascular Malformations (AVM/DVA)	AVM: Cluster of flow voids with early venous filling on contrast MRI. DVA: Radial medullary veins converging into a single enlarged collecting vein, show blooming on SWI	21,22
Arachnoid cyst	Well-defined, extra -axial, CSF -intensity lesion without enhancement or restricted diffusion, and causing variable mass effect on adjacent brain structures.	23
Chiari I malformation	Caudal descent of one or both cerebellar tonsils ≥5 mm below the foramen magnum on midsagittal T1WI and T2WI	24
Chiari II malformation	Complex hindbrain malformation characterized by downward displacement of the cerebellar vermis, brainstem, and fourth ventricle small posterior fossa, and beaking of the tectum and associated hydrocephalus	24

https://doi.org/10.57234/jiimc.december25.2543

pathologies.[15] MRI also aids in surgical planning and can lead to excellent outcomes, achieving seizure free life in well selected patients.[16,17]

Encephalomalacia is a well known cause of epilepsy and has been associated with poor prognosis in refractory cases.[18] Enlarged amygdala may be a part of temporal lobe epilepsy subtype or represent a nonspecific finding.[19] Porencephalic cyst, ulegyria, PVL and hemicerebral atrophy are well established causes of epilepsy.[20] Among vascular malformation AVMs[21] are strongly associated with epilepsy whereas role of DVA[22] is less frequent. The association between epilepsy and arachnoid cyst[23] and chiari malformations[24] remains controversial in literature. While less frequent in our cohort, their presence highlights the diverse range of structural etiologies detectable on MRI in young epileptic patients.

As it was a single-center retrospective study, the findings may not be generalizable to broader populations. Furthermore, findings were not correlated with clinical data such as EEG findings, seizure type and patients were not followed after imaging. Future prospective, multicenter studies integrating clinical, imaging and follow up outcome data are recommended to better clarify the role of MRI findings in guiding epilepsy management.

Conclusion

Dedicated MRI brain epilepsy protocol serves as a first-line neuroimaging modality in epilepsy management, as it allows early detection of structural lesions that can influence treatment decisions. This is particularly important in young patients, where early diagnosis can impact long-term outcomes.

Acknowledgement: Authors acknowledge Dr. Nadia Gul (HOD Radiology department POF hospital Wah Cantt) for general support.

Disclaimer: Authors declare that this study has not been published or under consideration for publication in any scientific journal nor a part of M.Phil or PhD dissertation.

Conflict of Interest: Authors declare that they have no any conflict of scientific or financial interest with any person or institution.

Funding Disclosure: No funding has been received for conduction of this study.

REFERENCES

1. Sarmast ST, Abdullahi AM, Jahan N, Abdullahi MI, Ahmed MI, Salisu AD, et al. Current classification of seizures and epilepsies: scope, limitations, and recommendations for future action. *Cureus*. 2020 Sep 20;12(9):e10549. doi:10.7759/cureus.10549.
2. Sirven JI. Epilepsy: a spectrum disorder. *Cold Spring Harb Perspect Med*. 2015 Sep 1;5(9):a022848. doi:10.1101/cshperspect.a022848.
3. Zhang Y, Kong X, Lv J, Yang C, Li X, Yang X, et al. Analysis of the global burden of disease study highlights the global, regional, and national trends of idiopathic epilepsy epidemiology from 1990 to 2019. *Prev Med Rep*. 2023;36:102522. doi:10.1016/j.pmedr.2023.102522.
4. Khatri IA, Iannaccone ST, Ilyas MS, Abdullah M, Saleem S. Epidemiology of epilepsy in Pakistan: review of literature. *J Pak Med Assoc*. 2003 Dec;53(12):594-7.
5. Mogal Z, Aziz H. Epilepsy treatment gap and stigma reduction in Pakistan: a tested public awareness model. *Epilepsy Behav*. 2020 Feb;102:106637. doi:10.1016/j.yebeh.2019.106637.
6. Biset G, Abebaw N, Gebeyehu NA, Estifanos N, Birrie E, Tegegne KD. Prevalence, incidence, and trends of epilepsy among children and adolescents in Africa: a systematic review and meta-analysis. *BMC Public Health*. 2024 Mar 12;24(1):771. doi:10.1186/s12889-024-18236-z.
7. Rozensztrauch A, Kołtuniuk A. The quality of life of children with epilepsy and the impact of the disease on family functioning. *Int J Environ Res Public Health*. 2022 Feb 17;19(4):2277. doi:10.3390/ijerph19042277.
8. Bernasconi A, Cendes F, Theodore WH, Gill RS, Koepp MJ, Hogan RE, et al. Recommendations for the use of structural magnetic resonance imaging in the care of patients with epilepsy: a consensus report from the ILAE Neuroimaging Task Force. *Epilepsia.* 2019;60(6):1054-68. doi:10.1111/epi.15612.
9. Gauci MC, Gauci R, Ruggieri M, Polizzi A, Praticò AD. Clinical, genetic, EEG, neuroimaging insights and conservative treatment in pediatric focal epilepsy: a retrospective observational study. J Clin Med. 2025;14(7):2234. doi:10.3390/jcm14072234.
10. Elmi AM, Ibrahim AA, Hassan MS, Osman FAO, Çelik C, Dirie AM, et al. Magnetic resonance imaging findings and their association with electroencephalographic data in pediatric epilepsy. *Int J Gen Med.* 2024;17:123-130. doi:10.2147/IJGM.S356789.
11. Mao YT, Goh E, Churilov L, McIntosh A, Ren YF, O'Brien TJ, et al. White matter hyperi ntensities on brain magnetic resonance imaging in people with epilepsy: a hospital-based study. *CNS Neurosci Ther.* 2016 Sep;22(9):758-63. doi:10.1111/cns.12571.
12. Uslu N, Uysal E, Karagöz E, Yılmaz M, Yıldız Y, Demir S, et al. Relationship of white matter hyperintensities with clinical features of seizures in patients with epilepsy. *Neurol Sci*. 2022 Jan;43(1):357-63. doi:10.1007/s10072-021-05554-6.
13. Engel J Jr. Mesial temporal lobe epilepsy: what have we learned? Neuroscientist. 2016 Apr;22(2):182-96. doi:10.1177/1073858415593869.
14. Jehi L. The epileptogenic zone: concept and definition.

Epilepsy Curr. 2018 Jul-Aug;18(4):258-262. doi:10.5698/1535-7597.18.4.258.
15. Lerner JT, Salamon N, Hauptman JS, Velasco TR, Hemb M, Wu JY, et al. Assessment and surgical outcomes for mild type I and severe type II cortical dysplasia: a critical review and the UCLA experience. *Epilepsia.* 2009 Jun;50(6):1310-35. doi:10.1111/j.1528-1167.2008.01998.x.
16. Sisodiya SM. Malformations of cortical development: burdens and insights from important causes of human epilepsy. *Lancet Neurol.* 2021 Jan;20(1):48-60. doi:10.1016/S1474-4422(20)30395-1.
17. Severino M, Geraldo AF, Utz N, Tortora D, Pogledic I, Klonowski W, et al. Definitions and classification of malformations of cortical development: practical guidelines. *Brain*. 2020 Oct 1;143(10):2874-94. doi:10.1093/brain/awaa174.
18. Zhong Y. Screening of risk factors for poor prognosis in patients with refractory epilepsy secondary to encephalomalacia. *Comput Math Methods Med*. 2022 Jul 4;2022:5720102. doi:10.1155/2022/5720102.
19. Reyes A, Thesen T, Kuzniecky R, Devinsky O, McDonald CR, Jackson GD, et al. Amygdala enlargement: temporal lobe epilepsy subtype or nonspecific finding? *Epilepsy Res*. 2017 Apr;132:34-40. doi:10.1016/j.eplepsyres.2017.02.019.
20. Cendes F, Theodore WH, Brinkmann BH, Sulc V, Cascino GD. Neuroimaging of epilepsy. *Handb Clin Neurol*. 2016;136:985-1014. doi:10.1016/B978-0-444-53486-6.00051-X.
21. Ogaki A, Ikegaya Y, Koyama R. Vascular abnormalities and the role of vascular endothelial growth factor in the epileptic brain. *Front Pharmacol*. 2020 Feb 4;11:20. doi:10.3389/fphar.2020.00020.
22. El Meskine T, Li J, Reise J, Toffa DH. Epilepsy and cerebral developmental venous anomalies (DVAs): a systematic review. *Epileptic Disord*. 2023 Apr;25(2):209-17. doi:10.1002/epd2.20020.
23. Nikolić I, Kovačević M, Janković M, Vojinović R, Kostić V, Sokić D. The association of arachnoid cysts and focal epilepsy: a hospital-based case-control study. *Epilepsy Res.* 2017;138:56-61. doi:10.1016/j.clineuro.2017.05.014.
24. Pascual-Castroviejo I, Pascual-Pascual SI, Velázquez-Fragua R, Vázquez-López M. Chiari type I and type II malformations and epilepsy: a comprehensive review. *Childs Nerv Syst.* 2008;24(11):1359-1366. doi:10.1007/s00381-008-0699-5.

CONFLICT OF INTEREST

Authors declared no conflicts of Interest.

GRANT SUPPORT AND FINANCIAL DISCLOSURE

Authors have declared no specific grant for this research from any funding agency in public, commercial or nonprofit sector.

DATA SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon request.