Original Research Article

Study of role of MRI in evaluation of pediatric epilepsy at a tertiary hospital

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Abstract

Background: Epilepsy is a disease characterized by spontaneous recurrence of unprovoked seizures. Many focal lesions/ pathologies responsible for epilepsy are identified with neuroimaging. The imaging modality of choice is MRI because of its superior resolution compared to CT and USG. **Material And Methods:** Present study was Hospital based prospective study, conducted in pediatric patients (age under 12 years) referred from OPD and IPD who presented with epilepsy, underwent MRI study. **Results:** Out of 100 patients, most common age group in our study was 10-12yrs age (33%) followed by 0-3 yrs (29 %). 58 patients (58%) were males and 42 patients (42%) were females. Male: Female ratio 1.3:1. 60patients(60%)presented with Generalized seizures, 29 patients (29%) presented with focal seizures while 11 patients (11%) had an unknown onset. Out of 100 patients studied, 85 patients (85%) had positive findings on MRI while 15 patients (15%) had normal MRI with no detectable lesions. In the study, the most common cause of epilepsy was infection (35%), followed by anoxia and hypoxic ischemic encephalopathy (HIE) (17%), malformations of cortical development (MCD) (8.2%), acquired metabolic disorders and vascular causes (3.5%) each. The most common etiology seen on MRI was infection in all age groups except 0-3yrs age group where anoxia and HIE was most common etiology while infection was second most common etiology. **Conclusion:** MRI plays a significant role in evaluation of pediatric patients presenting with epilepsy and it is the first imaging modality of choice with proper MRI seizure protocol to establish the correct diagnosis, plan the management according to diagnosis as well as helps in prognosis.

Keywords: MRI, pediatric epilepsy, refractory seizures, infectious etiology

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INTRODUCTION

Epilepsy is a disease characterized by spontaneous recurrence of unprovoked seizures. Seizures are time-limited paroxysmal events that result from abnormal, involuntary, rhythmic neuronal discharges in the brain.[1] Epilepsy is one of the most common neurologic conditions, with an incidence of approximately 50 new cases per year per 100,000 population.[2] Diagnosis of seizure involves identifying the symptoms, clinical diagnosis of the case, laboratory evaluation, EEG recording, lumbar puncture in suspected meningitis and neuroimaging.[3,4] Recent advances in neuroimaging play an important role in the diagnosis, management and in guiding the treatment of pediatric epilepsy. Functional neuroimaging provides further information and may show abnormalities even in cases where MRI was normal, thus further helping in the localization of the epileptogenic foci and guiding the possible surgical management of intractable/refractory epilepsy when indicated.[5] Many focal lesions/ pathologies responsible for epilepsy are identified with neuroimaging. The yield of neuroimaging is high even in low risk groups.[6] The imaging modality of choice is MRI because of its superior resolution compared to CT and USG.[7] MRI provides detailed evaluation of small lesion, radiation free. Present study was aimed to study of role of MRI in evaluation of paediatric epilepsy at a tertiary hospital.

MATERIAL AND METHODS

Present study was Hospital based prospective study, conducted in Department of Radiodiagnosis, Vilasrao

Deshmukh Government Medical College, Latur, India. Study duration was of 2 years (July 2018 to June 2019). Study was approved by institutional ethical committee.

Inclusion criteria: All pediatric patients (age under 12 years) referred from OPD and IPD who presented with epilepsy

Exclusion criteria: Claustrophobicpatients, Patients with metallic implants considered contraindicated for MRIimaging. Patients with trauma. Patients with febrile seizuredisorders.

First informed consent was taken from parents/accompanying relatives after giving proper information of the MRIscan. Then patient was screened for ferromagnetic objects and aneurysm clipsetc.

Complete clinical history, birth and vaccination history, family history and past history of patient was noted. The points noted were type of seizure, duration of illness and any associatedcomplaints. Physical examination findings as for evidence of any neurocutaneous stigmata and complete CNS examination findings were noted. Biochemical investigations like complete blood profile, liver and renal function tests, blood glucose levels, blood electrolytes levels as advised by physician were noted. Other laboratory parameters like Biochemical levels forleukodystrophies, Serological studies forinfections and CSF examination.

Findings of EEG and CT scan if done were documented. Very few cases had EEG documentation which was correlated with imaging findings. For MRI, patient was positioned supine on scanning table, immobilization of the patient's head was achieved and the head coil was applied. Patients were subjected to MRI scanning. When necessary, adequate sedation was given by the anaesthetist. Magnetic Resonance Imaging was done with Machine, 1.5 Tesla GE SIGNA MRISYSTEM, Radiofrequency coil—Sense headcoil, excellent resolution, field of view was kept as small as possible (approximately 20 cms), Slice thickness-- usually 3-5 mm, Interslicegap 1mm and Matrix 256 x256. Conventional MR imaging was performed by taking T1W (TE 8.0 ms, TR 480 ms), T2W (TE 102.9 ms, TR 4780 ms), and FLAIR (TE 92.2 ms, TR 8002 ms) sequences in planes as mentioned below. Post gadolinium (dose 0.1mmol/kg) enhanced MRI was performed in Axial and Sagittal planes in selected cases depending on findings on non- contrast study or clinical suspicion. DWI (TE 83 ms, TR 5025 ms) and GRE (Gradient recalled echo) axial performed in all cases. When required, MR spectroscopy, Venous 3D PCA (Phase Contrast Angiography) and MR angiography including TOF was done.

Pulse sequences and imaging planes: T1 Sagittal and Axial Pre and Post contrast. T2 Axial and Coronal. FLAIR Axial. DWI (Diffusion Weighted Imaging) Axial GRE (Gradient Recalled Echo) Axial. T1 Inversion Recovery sequence.

In suspected temporal lobe epilepsy additional sequences are: T1 angled coronal, T1 3D isotropic acquisition. FLAIR angled coronal, T2 angled coronal, MR Spectroscopy, Venous 3D PCA (Phase Contrast Angiography) and MR angiography including TOF if required.

MRI scans were studied with respect to: Number of lesions present., Unilateral or Bilateral, Site, Signalintensity, Any hemorrhage, Calcifications. Surrounding edema, Mass effect, Diffusion restriction, Contrast enhancement/enhancing lesion. Abnormal meningeal enhancement, Atrophy, Hydrocephalus, MR spectroscopy findings. Any other significant positive finding Final diagnosis was based on the medical history, clinical presentation, EEG and CT correlation, followup CSF analysis, pathological, surgical findings when available and\ response to medical therapy. In inconclusive cases it was made by follow up MRI and treatment response. MRI Brain findings were noted and recorded. The management decision, follow up and outcome recorded. Statistical analysis–Data was collected and entered in MS Excel.

RESULTS

This study includes MRI brain evaluation of 100 cases of pediatric patients aged 0- 12 years. Most common age group in our study was 10-12yrs age (33%) followed by 0-3 yrs (29 %). 58 patients (58%) were males and 42 patients (42%) were females. Male: Female ratio 1.3:1.

Table 1: Age And gender Distribution

Age Group (In Years)	Male	Female	Total
0-3	16	13	29
4-6	14	4	18
7-9	11	9	20
10-12	17	16	33
TOTAL	**58**	**42**	**100**

60patients(60%)presented with Generalized seizures, 29 patients (29%) presented with focal seizures while 11 patients (11%) had an unknown onset.

Table 2: Distribution according to types of seizure

Types of Seizure	No. of patients	Percentage (%)
Generalized	60	60
Focal	29	29
Unknown	11	11
TOTAL	**100**	**100**

In table 3, Out of 100 patients studied, 85 patients (85%) had positive findings on MRI while 15 patients (15%) had normal MRI with no detectable lesions. In the study, the most common cause of epilepsy was infection (35%), followed by anoxia and hypoxic ischemic encephalopathy (HIE) (17%), malformations of cortical development (MCD) (8.2%), acquired metabolic disorders and vascular causes (3.5%) each. In our study, total 32 patients reported

to have infection, tuberculosis (40.6%), encephalitis (9.3%), meningitis (21.8%), meninigo-encephalitis (9.3%), viral infection (12.5%) were common findings. It was noted that Mesial Temporal sclerosis was most common cause of isolated temporal lobe epilepsy. Hippocampal atrophy and secondary changes were found in 100% while Hippocampal T2 and FLAIR Hyper intensity and abnormal architecture were seen in 75% patients. 7 patients had malformations of cortical development. Focal cortical is dyplasia was seen in 2 patients (14.2%) while rest of the pathologies like microcephaly, agyria, lissencephaly, polymicrogyria, heterotopia, schizencephaly were seen in one patient each (7.1%) cortical developmental malformations presented with epilepsy in early age group, particularly in infancy (<1 year of age). Out of 3 patients with acquired toxic-metabolic disorders, 1 patient each had Chronic bilirubin encephalopathy, PRES (posterior reversible encephalopathy syndrome) secondary to hypertension and Medication-Related Leukoencephalopathy. Outof5patientsof neoplasticetiology, 1 patient each had DNET, ganglio-glioma, glioma, pilocystic astrocytoma and glioblastoma multiforme. Vascular pathology was noted in 3patients. 2 patients (66.6%) has arterial infarct and one had Vasculitis. Out of 2 patients presenting with demyelination on MRI, one patient had ADEM while other patient had tumefactive demyelination.

Table 3: Etiology on MRI

MRI findings	No. of patients	Percentage(%)
MRI IMAGING		
Normal	15	15 %
Abnormal	85	85 %
Type of Etiology		
Infection	32	37
Tuberculosis	13	40.6%
Meningitis(excluding tuberculous)	7	21.8%
Viral (Meningitis, encephalitis)	4	12.5%
Encephalitis	3	9.3%
Meningoencephalitis (excluding tuberculous)	3	9.3%
Rasmussen's encephalitis	1	3.12%
Hydatid cyst	1	3.12%
Anoxia and Hypoxic Ischemic Encephalopathy	15	17
Miscellaneous	12	14
Post ictal edema	7	58.3
Hydrocephalus	3	25.0
Arachnoid cyst	1	8.3
Subdural Hygroma	1	8.3
Malformations of Cortical Development	7	8.2
Microcephalywithagyria	1	7.1
Lissencephaly	1	7.1
Polymicrogyria	1	7.1
Focalcorticaldysplasia	2	14.2
Heterotopia	1	7.1
Schizencephaly	1	7.1
Neoplasm	5	5.8
DNET	1	20
Ganglioglioma	1	20
Glioma	1	20
Pilocytic astrocytoma	1	20
Glioblastoma multiforme	1	20
Mesial Temporal Sclerosis	4	4.7
Hippocampal atrophy	4(100%)	
Hippocampal T2 and FLAIR Hyper intensity	3(75%)	
Loss of hippocampal architecture	3(75%)	
Secondary changes (Temporal horn dilatation, caudate nucleus atrophy and hypointensity in T1W images)	4(100%)	
Vascular	3	3.5
Arterial infarct (excluding tuberculous)	2	66.6
Vasculitis	1	33.3

Acquired metabolic disorders	3	3.5
Chronic bilirubin encephalopathy	1	33.33
PRES (posterior reversible encephalopathysyndrome)	1	33.33
Medication-Related Leukoencephalopathy	1	33.33
Demyelinating Diseases	2	2.3
ADEM	1	50
Tumefactive demylination	1	50
Phakomatoses (Tuberous sclerosis)	1	1.1
Inherited metabolic disorders (Metachromatic Leukodystrophy)	1	1.1

In our study, isolated temporal lobe lesion was cause of epilepsy in 6 patients. Out of 6 patients Mesial Temporal sclerosis was found in 4 patients (66.6%), Glioma and ganglioglioma was found 1 patient (16.6%) each.

Table 4: Isolated temporal lobe epilepsy (N= 6)

Pathologies	No. of patients	Percentage (%)
Mesial Temporal sclerosis	4	66.6
Glioma	1	16.6
Ganglioglioma	1	16.6

In our study out of 100 patients, 85 patients showed abnormal finding on MRI. In our study the most common age group showing pathology on MRI was 10-12yrs age, followed by 0-3yrs age group.The most common etiology seen on MRI was infection in all age groups except 0-3yrs age group where anoxia and HIE was most common etiology while infection was second most common etiology. In age group 0-3yrs, there were total 27 patients (31.7%) with positive MRI, common etiologies were anoxia and HIE (8 patients), followed by infection (7 patients), and malformations of cortical development (3 patients). In age group 4-6yrs, total 14 (16.4%) had pathologies on MRI, infection was found to be most common cause (6 patients), followed by acquired metabolic diseases and miscellaneous (2 patients each). In age group 7-9yrs, total 14 (17.6%) had pathologies on MRI, infection was reported as most common cause (7 patients), followed by anoxia HIE and neoplasm (2 patients each). In age group 10-12yrs, total 29 (34.1%) had pathologies on MRI, infection was seen as most common etiology (12 patients), followed by anoxia and HIE (4 patients).

Table 5: Distribution of various types of etiologies according to age group (n=85)

Type of Etiology	0-3 Yrs	4-6 Yrs	7-9 Yrs.	10-12 Yrs.	TOTAL
Mesial Temporal Sclerosis	2	1	1	0	4
Malformations of CorticalDevelopment	3	1	1	2	7
Phakomatoses	0	0	0	1	1
Inherited metabolic disorders	1	0	0	0	1
Acquired metabolic disorders	0	2	1	0	3
Anoxia and Hypoxic Ischemic Encephalopathy	8	1	2	4	15
Infection	7	6	7	12	32
Demyelinating Diseases	0	0	0	2	2
Neoplasm	0	1	2	2	5
Vascular	1	0	0	2	3
Miscellaneous	5	2	1	4	12
TOTAL	**27**	**14**	**15**	**29**	**85**

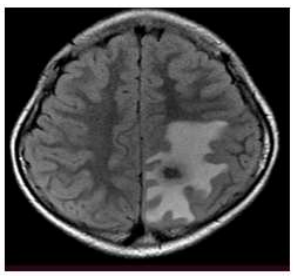

Figure 1: AXIAL FLAIR

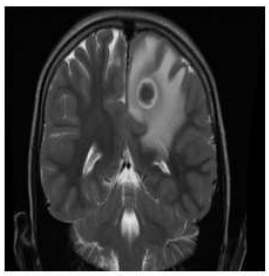

Figure 2: COR T2

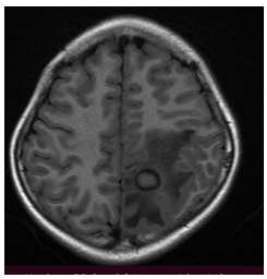

Figure 3: AXIAL T1

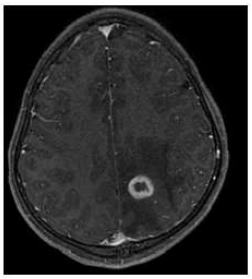

Figure 4: AXIAL T1 FAT SAT POST CONTRAST

A well-defined T1 isointense and T2 hypointense lesion showing peripheral ring like enhancement without diffusion restriction with moderate adjacent vasogenic edema seen in left parietal lobe

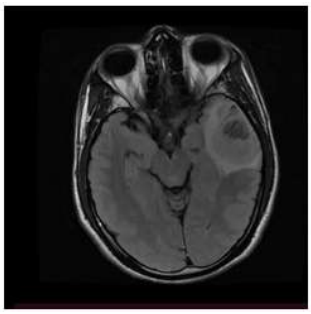
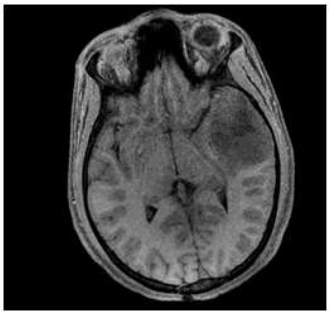
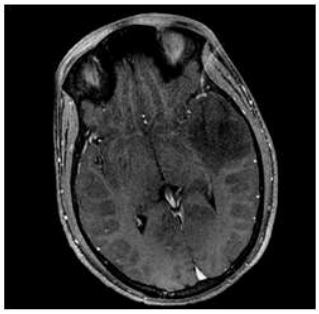

Figure 5: AXIAL FLAIR **Figure 6:** T1 FAT SATPRECONTRAST **Figure 7:** T1 FAT SATPOSTCONTRAST

FLAIR hyperintentense lesion in left temporal lobe with minimal adjacent edema without post contrast enhancement

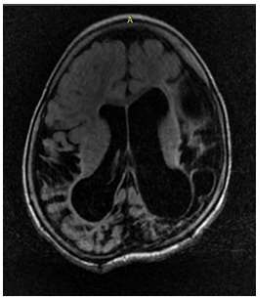
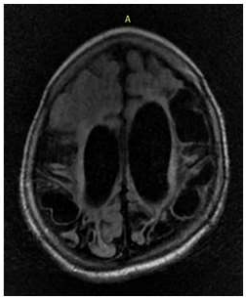

Figure 8: AXIAL FLAIR AXIAL FLAIR

Cystic changes in bilateral subcortical white matter with adjacent gliosis and atrophy

DISCUSSION

In present study, out of 100 patients maximum number of patients in the study were in the age group of 10-12 years (33%) followed by 0-3 years (29%). The mean age in our study was 6.2yrs. Similar findings were noted by Gulati P *et al.*,[8] in which maximum patients were in the age group 6-12 years. However the mean age our study is slightly lesser than the study by Wongladarom S *et al.*,[9](mean age was 7 years and 5 months). In our study of 100patients, 58% were males and 42% were females. Male : Female ratio was1.2:1. Our study correlates with the study done by Sanghvi JP *et al.*,[10]in which 60.5% were males and 31.7% werefemales. It also correlates with the study done by Amirsalari S *et al.*,[11]in which there were 57.7% boys and 42.5% girls. In our study of 95 patients, maximum number of patients, 60% presented with Generalized seizures, 29% had focal seizure and 11 % were unknown type at presentation. Our study has similar results as that of study done by Rasool A *et al.*,[12]in which 276 patients were studied. In this study it was found that generalized seizures constituted the major seizure group (42%), followed by partial seizures (31.2%) and complex febrile seizure (23.2%). Our study also correlates with the study done by Chaurasia R *et al.*,[13]in which generalized seizures accounted for the major number of patients seen in 76.7%. In this study of total 100 patients, 85 patients (85%) had abnormal MRI findings. Our study is comparable with the study done by Kuzniecky R *et al.*,[14]in which MRI revealed abnormalities in 84% of patients. Resta *et al.*,[14] reported positive MRI in 51.3%, Wang *et al.*,[15]in 41.7% and Chang *et al.*,[16]in 48.9%. Our study shows a higher percentage, probably because of strict exclusion criteria's, which shows that patient selection, plays an important role in MR positivity rates. In our study, In the study, the most common cause of epilepsy was infection (35%), followed by anoxia and hypoxic ischemic encephalopathy (HIE) (17%), miscellaneous causes (14%) and malformations of cortical development (MCD) (8.2%). Similar findings were noted by Aarti Aanand*et al.*,[18]where 95 children under the age of 12 years were studied, infection (29.8%) was the most common etiology followed by anoxia and hypoxic-ischemic encephalopathy. Ojaswi B khandediya *et al.*,[19] also noted that infection was the most common etiology followed by Mesial temporal sclerosis and focal cortical dysplasia. In present study, isolated temporal lobe lesion was cause of epilepsy in 6 patients, and Mesial Temporal sclerosis (4 patients) and Glioma and ganglioglioma (1 patient each) were common causes. Our study correlates with study done by J D Grattan Smith *et al.*,[20]in which mesial temporal sclerosis was most common cause of temporal lobe epilepsy seen in 30 out of 53 children (57%), followed by tumours in 8 (15%), cavernous angioma in 1(1.8%) and ectopic gray matter in 1(1.8%) ofpatients. Sales LV *et al.*,[21] in which out of 31 patients with temporal lobe epilepsy, most common pathology was mesial temporal sclerosis seen in 9 (29.0%), dysplasia in 8 (25.8%), tumors in 2(6.4%), arachnoid cyst

in 1 (3.2%) and choroid cyst in 1 (3.2%). In present study, 32 patients reported to had infection, tuberculosis (40.6%), encephalitis (9.3%), meningitis (21.8%), meninigo-encephalitis (9.3%), viral infection (12.5%) were common findings. Out of 13 patient of tuberculosis, 8 patients (61.5%) had leptomeningeal enhancement, 3 patients (23.07%) had tuberculoma, and 1 patient (7.6%) each of hydrocephalus and encephalitis. In Gulati P, Jena A.N. *et al.*,[8]study, out of 345 patients with abnormal MRI, tuberculoma was the most common etiology and was seen in 98 (28.4%), followed by neurocysticercosis in 86(24.9%). In Chaurasia R *et al.*,[22] study, most common cause of epilepsy was CNS tuberculosis (30.3%), followed by Neurocysticercosis (11.0%) and Encephalitis (7.9%). However, our study is in discordance with Parihar Ravi Kumar *et al.*,[23] study, in which most common etiology was neurocysticercosis (55.81%) followed by tuberculoma (29.91%). In study by Mittal GK *et al.*,[24] out of 54 patients with Malformations of cortical development, Focal cortical dysplasia was the most common pathology reported in 16 patients (29.6%), next was Schizencephalyin8(14.8%),Polymicrogyriain8(14.8%),D NETin6(11.1%).Similar findings were noted in present study. For etiology in age group 0-3 years, our study correlates well with Khreisat WH *et al.*,[25]study, most common etiological factor found in this study was perinatal asphyxia seen in 55%, followed by CNS infection in 15%, anomalies of central nervous system in (9%), head injuries in (8%), congenital and prematurity in(5%).

For etiology in older age group, our study correlates with Parihar Ravi Kumar *et al.*,[23]study, in which children in the age group of 28days to 18 years with partial seizures were studied. 6 patients (66.6%) in age group of 28days-5years, 18 patients (85.7%) in age group of >5-10years and 12 patients (92.3%) in age group >10-18years had infection as the most common etiology. Thus infection had major burden in causing epilepsy with increasing age group. For etiology in older age group, our study also correlates with Gulati P *et al.*,[8]study, in which 170 children with chronic seizures were studied. Age distribution was done as follows: 0-1 year, 1-3 year, 3-6 year and 6-12 year. The etiologies were classified into Infections (tuberculomas, neurocysticercosis, meningitis), atrophy, vascular and miscellaneous causes. Infection was most common etiology in 6-12 years age group seen in 51.1%, followed by miscellaneous in 16.4%. In age group 0-1, 1-3 and 3-6 years, infection was seen in 4.7%, 4.1% and 3 %respectively. MRI plays an invaluable role in the evaluation of pediatric patients with seizure disorder. Accurate diagnosis of cause of seizure is important for treatment decision. With its high spatial resolution, excellent inherent soft tissue contrast, multi-planar imaging capability and lack of ionizing radiation; MRI has emerged as a versatile tool in imaging of pediatric patients with seizures. MRI not only identifies specific epileptogenic substrates, but also helps in determining specific treatment and predicts prognosis. Employing appropriate imaging protocols and reviewing the images in systemic manner helps in the identification of subtle epileptogenic structural abnormalities. MR imaging is superior neuroimaging with no radiation exposure and could be the first investigation of choice in epileptic syndrome, developmental cortical malformations, mesial temporal sclerosis. Its ability in identifying subtle lesions, location and extent of the lesions is excellent.

CONCLUSION

MRI plays a significant role in evaluation of pediatric patients presenting with epilepsy and it is the first imaging modality of choice with proper MRI seizure protocol to establish the correct diagnosis, plan the management according to diagnosis as well as helps in prognosis. MRI helps in evaluation of patients presenting with refractory seizures undiagnosed by other imaging modalities and children with newly diagnosed epilepsy especially those with abnormal neurological examination, focal seizures or focal EEG abnormalities.

REFERENCES

1. Shneker, B. F., Fountain, N. B. and Orlowski, J. M. Epilepsy. Disease-a-Month 49, 426–478 (2003).
2. Weinstein, S. Seizures and epilepsy: An overview. Epilepsy Intersect. Neurosci. Biol. Math. Eng. Phys. 65–77 (2016).
3. Tolaymat, A., Nayak, A., Geyer, J. D., Geyer, S. K. and Carney, P. R. Diagnosis and management of childhood epilepsy. Curr. Probl. Pediatr. Adolesc. Health Care 45, 3–17 (2015).
4. Khan, A., Lim, H. and Almubarak, S. Importance of prompt diagnosis in pediatric epilepsy outcomes. Seizure 80, 24–30 (2020).
5. Shaikh, Z., Torres, A. and Takeoka, M. Neuroimaging in pediatric epilepsy. Brain Sci. 9, 1–14 (2019).
6. Coryell, J. et al. Neuroimaging of early life epilepsy. Pediatrics 142, (2018).
7. Gaillard, W. D. et al. Guidelines for imaging infants and children with recent-onset epilepsy. Epilepsia 50, 2147–2153 (2009).
8. Gulati P , A Jena, Tripathi RP, G. A. Magnetic resonance imaging in childhood epilepsy. Indian pediatr. 1991 jul;28(7):761-5.
9. Wongladarom S, Laothamatas J, Visudtibhan A, S. P. Magnetic resonance imaging in epileptic paediatric patient. Review of experience of ramathibodi hospital. . J Med Assoc Thai 87:1092–9., (2004).
10. Cinalli, G. et al. Hydrocephalus in aqueductal stenosis. Child's Nerv. Syst. 27, 1621–1642 (2011).
11. Roy, A., Vinayan, K. and Kumar, A. Idiopathic intracranial hypertension in pediatric population: Case series from India. Neurol. India 61, 488–490 (2013).

12. Rasool A, Choch SA, Wani NA, Ahmad SM, I. Q. Role of EEG and neuroimaging in first onset afebrile and complex febrile seizures in children from Kashmir. Journal of paediatric Neurosciences. 2012; 7(1):9-15.
13. Rachna Chaurasia, Shuchi Singh, Sachin Mahur, P. S. Imaging in paediatric epilepsy, Spectrum of abnormalities detected on MRI. J Evolv Med Dent Sci 2013; 19: 2, 3377–3387.
14. Kuzniecky, R. et al. Magnetic resonance imaging in childhood intractable partial epilepsies: Pathologic correlations. Neurology 43, 681–687 (1993).
15. Chang T, Acosta MT, Rosser T, Conry JA, Pearl PL, Weinstein SL, Kolodgie M, Johnson P, Vezina LG, Dubovsky EC, Galliard WD. (2002) Neuroimaging in children during the acute evaluation of new onset seizures. Ann Neurol 52:S134.
16. Resta M, Palma M, Dicuonzo F, Spagnolo P, Specchio LM, Laneve A, Bellomo R, Lauriero F, La Selva L. (1994) Imaging studies in partial epilepsy in children and adolescents. Epilepsia 38:1187-1193.
17. Wang PJ, Liu HM, Fan PC, LeeWT, Young C, Tseng CL, Huang KM, Shen YZ. (1997) MRI in symptomatic /cryptogenic partial epilepsies of infants and children. Zhonghua Min Guo Xiao Er Ke Yi Xue Hui Za Zhi 38:127-136
18. Anand1, A., Disawal2, A., Bathwal3, P. and Ashwini Bakde. Magnetic Resonance Imaging Brain in Evaluation of Pediatric Epilepsy. Int. J. Sci. Res. 5, 8–14 (2017).
19. Khandediya, O. B., Mani, S. S., Kapoor, P. and Singh, V. A. iMedPub Journals Spectrum of MRI Findings in Pediatric Epilepsy : Medical and Surgical Causes of Epilepsy in Children and its Radiological Correlation Case Study Keywords : 22, 5–8 (2021).
20. J D Grattan-Smith, A S Harvey, P M Desmond and C W Chow. Hippocampal sclerosis in children with intractable temporal lobe epilepsy: detection with MR imaging 1993; 161:1045-8.
21. Sales LV, Velasco TR, Relative frequency, clinical, neuroimaging, and postsurgical features of pediatric temporal lobe epilepsy. 2006 Oct; 39(10):1365-72. Epub 2006 Aug 22.
22. Rachna Chaurasia, Shuchi Singh, Sachin Mahur, P. S. Imaging in paediatric epilepsy, Spectrum of abnormalities detected on MRI. J Evolv Med Dent Sci 2013; 19: 2, 3377–3387.
23. Parihar, R. K., Gupta, A. K., Saini, G. and Dev, G. Role of magnectic resonance imaging of brain in paediatric patients with partial seizures. JK Sci. 14, 60–64 (2011).
24. Mittal GK, Ganguly G, Bhattacharyya KB, Pandit A, Biswas A, Roy A, et al. Epilepsy patients with malformations of cortical development: Experience from a tertiary care centre in Eastern India. J Pediatr Neurol 2014; 12:117-26.
25. Khreisat WH. Clinical profile of epilepsy during the first two years of life. Pak J Med Sci 2006; 22:5.

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