

**Al-Nahrain University
College of Medicine
Department of Surgery**



Magnetic resonance imaging findings in pediatric epilepsy patients examined in the MRI unit at Al-Imamain Al- Kadhimain medical city

**A graduation research submitted to the department of
surgery at Al-Nahrain University – College of medicine**

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Dedication

*I dedicate this humble effort
To all my family and my beloved.
Your presence and encouragement
is what drives me forward ..*

Dhualfiqar Basim

April, 2019



Acknowledgment

First, I would like to express my greatest gratitude for my supervisor, **Dr. Ammar Mousa** for his continuous support, valuable informations and guidance throughout the period of this study.

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List of acronyms

Acronym	Meaning
ILAE	International league against epilepsy
BECTS	Benign epilepsy with centro-temporal spikes
IGE	Idiopathic generalized epilepsy
MCD	Malformations of cortical development
FCD	Focal cortical dysplasia
MTS	Mesial temporal sclerosis
MRI	Magnetic resonance imaging
CT	Computed tomography
T1W-FFE	T1 weighted - fast field echo
T2W-TSE	T2 weighted – turbo spin echo
FLAIR	Fluid attenuated inversion recovery
DWI	Diffusion weighted imaging
SWI	Susceptibility weighted imaging
ADC	Apparent diffusion coefficient

Chapter One

Introduction

1. Introduction:

1.1 Basic definitions:

An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain. Epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neuro biologic, cognitive, psychological, and social consequences of this condition (1).

Practically, epilepsy is a disease defined by any of the following conditions (2):

1. At least two unprovoked (or reflex) seizures occurring greater than 24 hours apart.
2. One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years.
3. Diagnosis of an epilepsy syndrome

The term “unprovoked” implies absence of a temporary or reversible factor lowering the threshold and producing a seizure (2). A provoked seizure may be due to structural damage (such as traumatic brain injury, brain tumor, stroke, tuberculosis) or due to metabolic abnormalities (such as alcohol withdrawal and renal or hepatic failure). (3)

An epileptic syndrome is a group of signs and symptoms that share a common pathogenesis, prognosis, and response to treatment. According to the International League Against Epilepsy (ILAE), epilepsy syndromes are divided into two major categories: localization-related syndromes and generalized-onset syndromes (4).

1.2 Classification of Seizures:

Figure 1.1 depicts the basic 2017 seizure classification as proposed by the international league against epilepsy (ILAE). Seizure classification begins with the determination of whether the initial manifestations of the seizure are focal or generalized. The onset may be missed or obscured, in which case the seizure is of unknown onset. A focal aware seizure corresponds to the prior term “simple partial seizure.” A focal impaired awareness seizure corresponds to the prior term “complex partial seizure.” Generalized seizures are divided into motor and non-motor (absence) seizures (5).

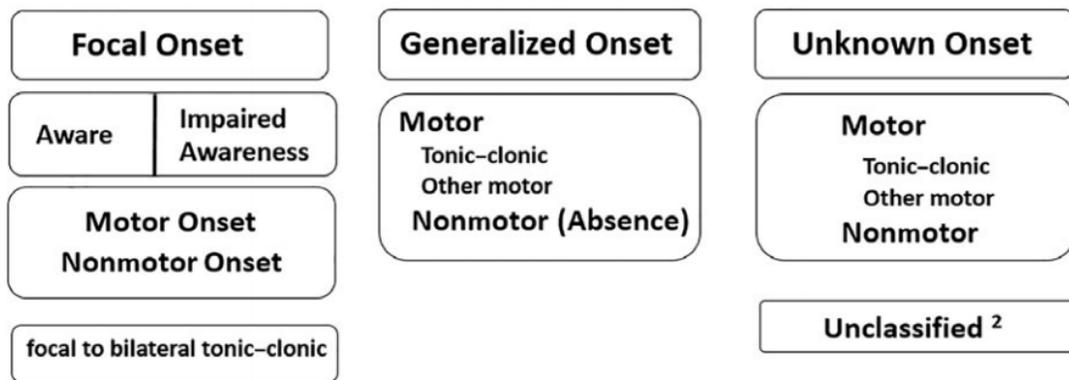


Figure 1.1: The basic ILAE 2017 operational classification of seizure types.

Partial seizures originate in a localized area of the brain, with clinical manifestations based on the area of brain involved and how extensively discharges spread from this “focus”. Generalized seizures begin with abnormal electrical discharges occurring in both hemispheres simultaneously. Generalized seizures also can spread and synchronize via the corpus callosum. A seizure that starts focally and then spreads widely throughout the brain is referred to as secondarily generalized. (6)

1.3 Pathophysiology of seizures:

A seizure occurs due to disruption in the normal balance between excitation and inhibition in part or all of the brain. A seizure can occur when excitation increases, inhibition decreases, or both. Hyper excitability can occur at one or more levels of brain function, including a network of interconnected neurons; the neuronal membrane with its ionic channels, neurotransmitters, and their receptors; or intracellular second messenger cascades. (6)

1.4 Epidemiology:

Epilepsy is a common problem worldwide with an age adjusted prevalence of 4–8/1000 in developed countries. In developed countries, the overall incidence of childhood epilepsy from birth to 16 years is approximately 40 cases in 100,000 children per year.(7)

1.5 Role of neuroimaging in diagnosis of epilepsy:

Structural neuroimaging plays an important role in the evaluation, management, and treatment of the child with epilepsy. The role of neuroimaging is to detect an underlying cerebral lesion that may be causally related to the seizure disorder (8).

Imaging is most useful for children with a suspected or confirmed localization-related or remote symptomatic generalized epilepsy. Early in the course of epilepsy it may be difficult to decide whether a child has a localization-related epilepsy.

Therefore, it is proposed that children with epilepsy undergo neuroimaging if one or more of the following are present (8):

1. If there is any evidence to suggest the epilepsy is localization related (e.g., focal).
2. Abnormal neurologic examination, including focal deficits, stigmata of neurocutaneous, cerebral malformation syndrome, or a history of significant developmental delay, arrest, or regression.
3. Children younger than 2 years, excluding those with simple febrile seizures.
4. Children with characteristics of a symptomatic generalized epilepsy syndrome as focal MRI findings may be found in a substantial proportion of these children.
5. Failure to control seizures, worsening seizures, changes in seizure manifestations, or developmental regression also necessitate neuroimaging if not previously performed.
6. Finally, new onset seizures/epilepsy presenting with evidence for a medical emergency such as increased intracranial pressure or status epilepticus always merit emergency imaging.

Imaging indicated	Imaging not indicated
Localization related seizures ^a	Childhood absence epilepsy
Focal history, abnormal exam, focal EEG abnormalities ^a	Juvenile absence epilepsy
Developmental regression <2 years old	Juvenile myoclonic epilepsy
Symptomatic generalized epilepsy syndrome	BECTS
Increased intracranial pressure	
History of status epilepticus	
Atypical course for BECTS/IGE	
^a Except for BECTS.	

Figure 1.2: Conditions in which imaging is indicated and not indicated for pediatric patients with epilepsy. (9)

BECTS refers to benign epilepsy of childhood with centro temporal spikes. It is one of the most common childhood epilepsy syndromes characterized by partial seizures with a characteristic EEG pattern in the centro temporal region of the brain. BECTS seizures nearly always disappear by age 16.

MRI is considered the imaging modality of choice because of superior anatomic resolution and characterization of pathologic processes. CT confers some advantages with regard to identifying bleeding and calcifications (as found in congenital infection). Studies in adults with childhood onset epilepsy and in children in both recent onset and chronic epilepsy find that MRI identifies more abnormalities than CT. Furthermore CT does not identify abnormalities found on MRI. MRI will identify focal cortical dysplasia, mesial temporal sclerosis, small tumors (such as oligodendrogliomas and gangliogliomas), and vascular malformations (AVM, cavernous angioma); CT will not or may not identify these (10).

MRI protocol for epilepsy is a group of MRI sequences put together to improve sensitivity and specificity in identifying possible structural abnormalities that

underlie seizure disorders (e.g. mesial temporal sclerosis and malformation of cortical development). The sequences performed are as the following **(11)**:

1. Non-focal epilepsy protocol

A good protocol for this purpose involves at least:

- T1 sequence: axial and coronal; in modern scanners it can be replaced by a 3D isotropic acquisition
- FLAIR sequence: axial and angled coronal; in modern scanners it can be replaced by a 3D isotropic acquisition
- Inversion recovery sequences: DIR sequence: 3D isotropic acquisition
- DWI/ADC
- SWI or T2

2. Temporal lobe epilepsy protocol

A good protocol for this purpose involves at least:

- T1 sequence: axial and coronal; in modern scanners it can be replaced by a 3D isotropic acquisition
- FLAIR sequence: axial and angled coronal; in modern scanners it can be replaced by a 3D isotropic acquisition
- T2 sequence: angled coronal
- DWI/ADC
- SWI or T2

1.6 Examples of abnormalities associated with epilepsy in pediatric age group:

1. Focal Cortical Dysplasia: FCD is now recognized as one of the most common causes of seizures in children with intractable epilepsy, accounting for nearly 80% of all surgically treated cases in children under 3 years of age **(12)**.

MRI is the modality of choice to assess patients with possible focal cortical dysplasias. There is much overlap of imaging features between the different types of FCD, and in many instances, no MRI abnormality is evident.

Focal cortical dysplasia is classified into 3 types: **(13)**

Type I – malformation presenting with abnormal cortical layering as a result of abnormal radial migration and maturation of neurons (FCD Type Ia) or disruption of typical 6-layered tangential composition of the cortex with

immature neurons (FCD Type Ib) or compromising both architectural abnormalities (FCD Type Ic).

Type II – malformation resulting from disrupted cortical lamination and specific cytological abnormalities, Type IIa with dysmorphic neurons and Type IIb with dysmorphic neurons and balloon cells.

Type III – malformation connected with different cortical dislamination and cytological abnormalities with main lesion within the same area/lobe.

General features of focal cortical dysplasia include: (14)

- Cortical thickening
- Blurring of white matter-grey matter junction with abnormal architecture of subcortical layer
- T2/FLAIR signal hyperintensity of white matter with or without the transmantle sign
- T2/FLAIR signal hyperintensity of grey matter
- abnormal sulcal or gyral pattern
- segmental and/or lobar hypoplasia/atrophy

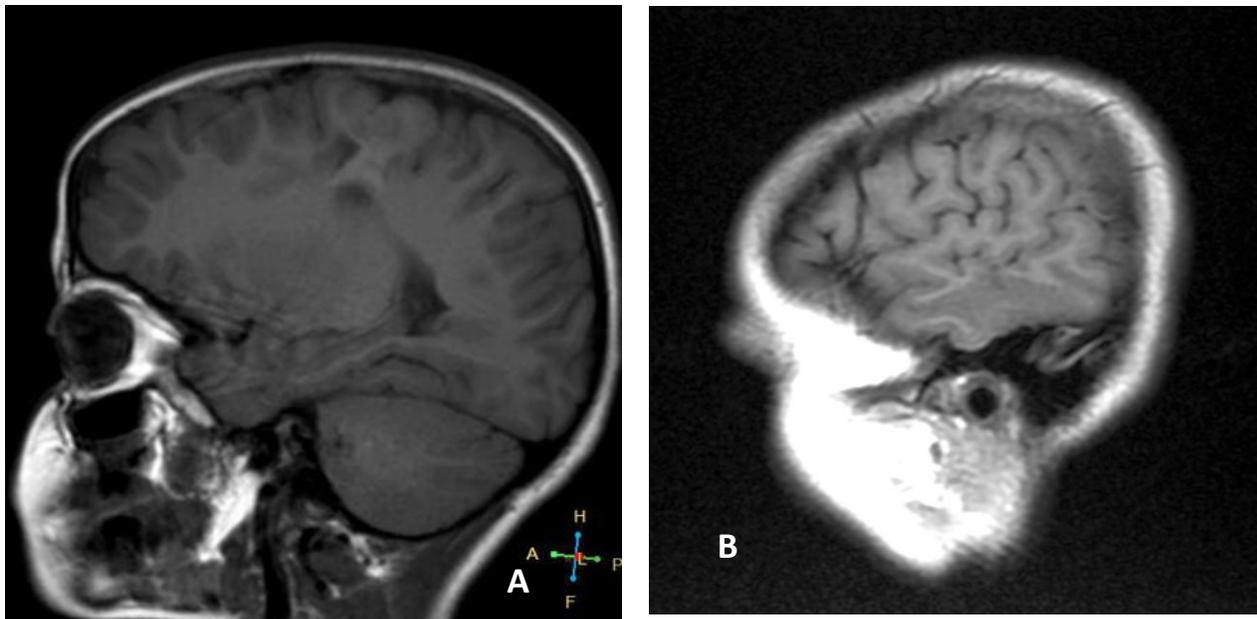


Figure 1.3: Sagittal T1 sections showing FCD type 2 (image A) and type 1 (image B)

2. Tuberos Sclerosis: an autosomal dominant disorder that results in multi organ hamartomas. As many as 90% of patients with tuberous sclerosis have seizures (15).

Central nervous system involvement by tuberous sclerosis is characterized by cortical tubers, subependymal nodules, and subependymal giant cell astrocytomas.

In neonates, the tubers are seen as regions of subcortical T1 hyperintensity and T2 hypointensity, but after about 6 months of age the signal intensity characteristics are reversed (16).



Figure 1.4: Axial FLAIR image shows multiple cortical and subcortical tubers (arrows).

3. Grey matter heterotopia: a relatively common group of conditions characterized by interruption of normal neuronal migration from near the

ventricle to the cortex, thus resulting in "normal neurons in abnormal locations" (17).

Grey matter heterotopias can be divided macroscopically into nodular heterotopias and diffuse heterotopias. Patients most commonly present with partial seizures in the second decade of life (18).

On MRI the heterotopic tissue follows grey matter on all sequences. Their margins are often indistinct. Careful examination of the remainder of the brain is necessary to identify associated anomalies (19).

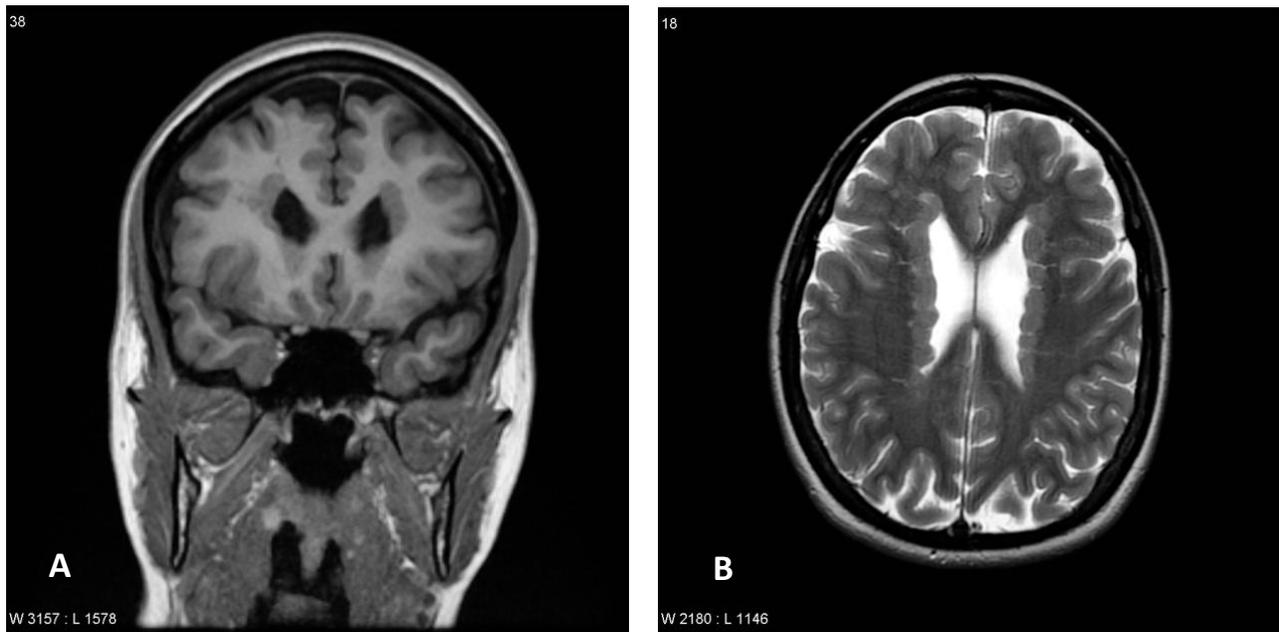


Figure 1.5: Grey matter heterotopia on coronal T1 (image A) and axial T2 (image B)

4. Hemimegalencephaly: is a severe, rare MCD. It can occur on its own or in association with a variety of syndromes, including neurofibromatosis type 1, tuberous sclerosis, and epidermal nevus syndrome. At clinical examination, it is usually dominated by severe and drug resistant epilepsy (20).

Hemimegalencephaly has characteristic MR imaging features. The most notable characteristic is unilateral cortical thickening involving all or part of the cerebral hemisphere.

The involved hemisphere also demonstrates ipsilateral white matter changes secondary to hypermyelination (21).

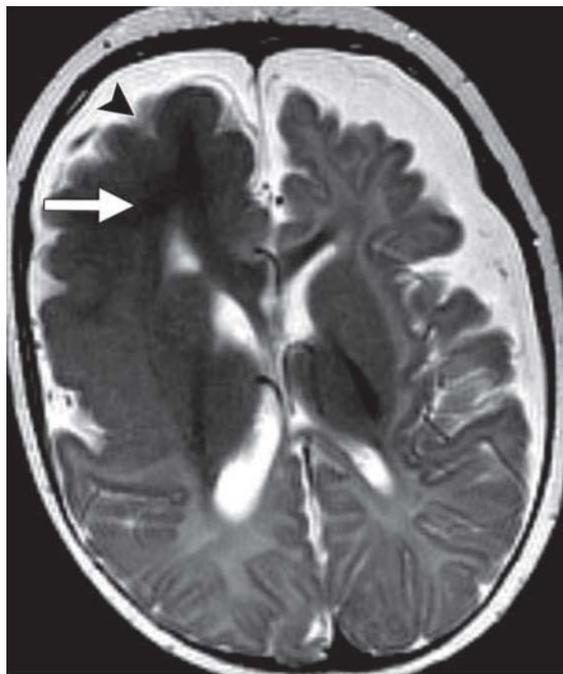


Figure 1.6: Right hemimegalencephaly in a patient with intractable epilepsy. Axial T2-weighted MR image right cortical thickening (arrowhead) and abnormal T2 hypointensity of the subjacent white matter (arrow).

5. Mesial Temporal Sclerosis: MTS is one of the most common causes of epilepsy in the adolescent and young adult population. It is characterized by

neuronal loss with gliosis in the hippocampus and may also involve the ipsilateral fornix and mammillary body (22).

At clinical examination, patients often have a history of a cortical insult such as intracerebral infection, head trauma, or complicated febrile seizures during the first 4–5 years of life (23).

Characteristic MR imaging features of MTS include atrophy of the hippocampus on T1-weighted images and increased signal intensity in the mesial temporal region on T2-weighted images (24).

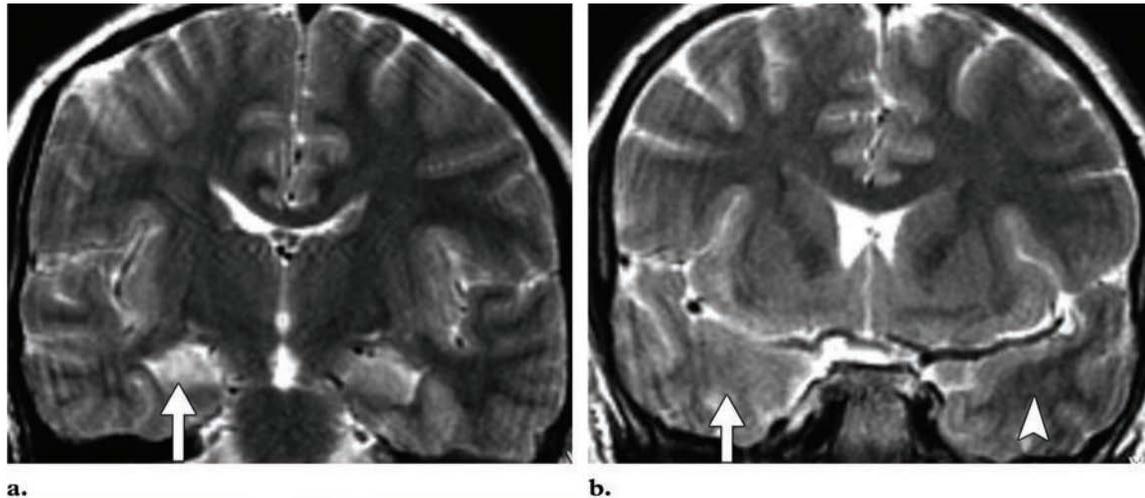


Figure 1.7: Right hippocampal sclerosis in a 15-year-old boy with intractable epilepsy. The images are slightly degraded by motion. **(a)** Coronal T2-weighted MR image shows T2 hyperintensity of the right hippocampus (arrow). **(b)** On a coronal T2-weighted MR image obtained slightly posterior to a, the white matter of the right temporal pole is T2 hyperintense (arrow) relative to the normal white matter on the left side (arrowhead).

6. Neoplasms: neoplasms of the central nervous system in pediatric patients often manifest clinically as seizures. Although any of these neoplasms may result in pediatric epilepsy, certain tumors are characteristically associated with this clinical manifestation. A group of these tumors sharing similar clinical-pathologic features are referred to as epilepsy-associated developmental tumors and include ganglioglioma, gangliocytoma, desmoplastic infantile

ganglioglioma, dysembryoplastic neuroepithelial tumor, and pleomorphic xanthoastrocytoma (25).

At MR imaging, they usually appear as cortically based lesions that are hypo to isointense on T1-weighted images and hyperintense on T2-weighted images (26).

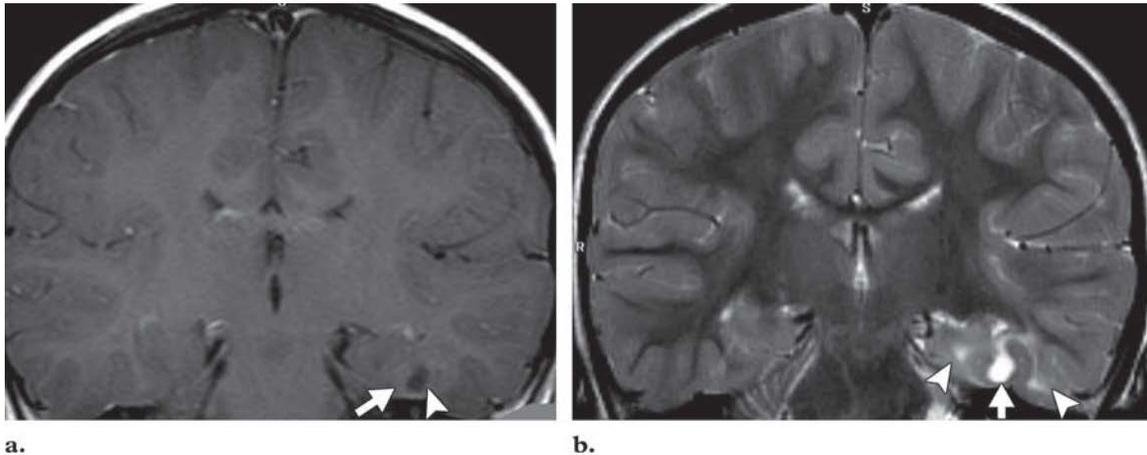


Figure 1.8: Left temporal lobe ganglioglioma and FCD. **(a)** Coronal contrast material–enhanced T1-weighted MR image shows a cortically based lesion in the left anterior temporal pole with a nonenhancing solid component (arrow) and a cystic component (arrowhead). **(b)** Coronal T2-weighted MR image shows areas of T2 hyperintensity (arrowheads) surrounding the lesion (arrow), findings that proved to be FCD associated with a ganglioglioma at pathologic analysis. Perilesional edema with ganglioglioma is rare.

7. Sturge-Weber Syndrome: Sturge-Weber syndrome is a rare neurocutaneous disorder. It is clinically characterized by epilepsy, progressive mental retardation, and facial telangiectatic nevi, often in the distribution of a trigeminal nerve division. Approximately 75%–90% of patients with Sturge-Weber syndrome have epilepsy (27).

MR imaging is considered to be the standard of reference for the imaging of Sturge-Weber syndrome. One of the most important signs is leptomeningeal enhancement with gadolinium-based contrast agents (28). On T1-weighted MR images, enlargement and abnormally avid enhancement of the ipsilateral choroid

plexus may also be seen (29). T2- weighted images are used to detect areas of gliosis and cerebral atrophy likely related to chronic ischemia (30).

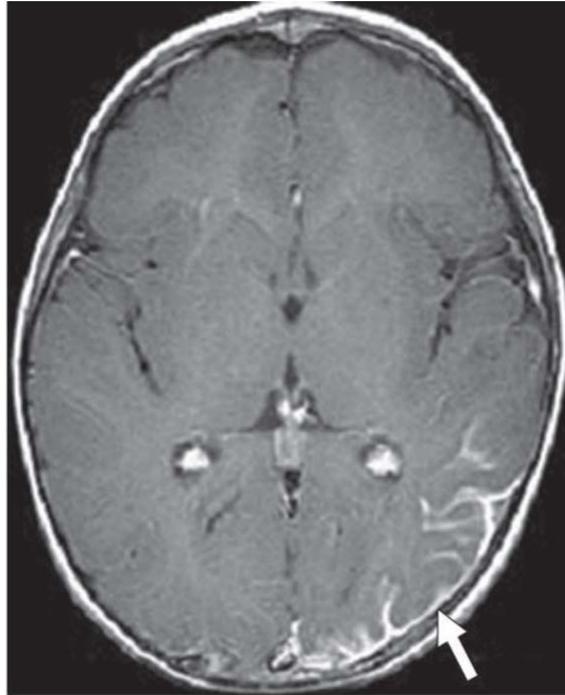


Figure 1.9: Sturge-Weber syndrome. Axial contrast-enhanced T1- weighted MR image shows leptomeningeal enhancement in the left temporo-occipital lobe (arrow).

Chapter Two

Patients and methods

2. Patients and methods:

This cross sectional study included 20 patients and was conducted in the MRI unit at Al-Imamain Al-Kadhimain medical city. The data were collected from October, 2018 to March, 2019. The minimum age of the patients in the study sample was 9 months and the maximum age was 15 years with the mean age being 7.8 years.

2.1 Patients:

22 patients were referred from the neurology outpatient and the pediatric outpatient clinics to the MRI unit at Al-Imamain Al-Kadhimain medical city. All the patients were clinically diagnosed with epilepsy and they presented with at least one of the following: focal seizure, generalized tonic clonic seizures, myoclonic seizures, delayed growth or focal neurological deficit. They underwent MRI examination and 2 of the patients were excluded from the study for not fulfilling the complete MRI epilepsy protocol. The 20 remaining patients were included in the study. 12 of them were males and 8 were females.

Exclusion criteria:

- Not completing the sequences of the MRI epilepsy protocol.
- Poor quality MRI images.
- Patients with contraindication for MRI (e.g. patients with metallic shells or implants)

2.2 Imaging technique:

The MRI imaging was carried out using a 3 Tesla MR scanner Philips system (Netherland) and 1.5 Tesla MR scanner Siemens system (Germany).

Each patient was examined while lying supine in the MRI machine. Some of the smaller age group patients underwent general anesthesia before the examination. Examinations were performed using head coil, the use of head coils provides excellent anatomical details and enhances the quality of the MRI images. Abnormal MRI findings were registered and included: abnormalities in the hippocampal formation, grey matter heterotopia, focal cortical dysplasia and tumors.

The following sequences were done for all patients:

- T1 weighted fast field echo images (T1W-FFE) in coronal and axial planes, with the following parameters: Repetition time (TR) = 255.4, Echo time (TE) = 4.6. Slice thickness was 5 mm.

- T2 weighted turbo spin echo images (T2W-TSE) in axial plane with the following parameters: Repetition time (TR) = 2806.9, Echo time (TE) = 80. Slice thickness was 5 mm.
- T2 weighted FLAIR images in coronal and axial planes with the following parameters: Repetition time (TR) = 11000, Echo time (TE) = 120. Slice thickness was 5 mm.
- Inversion recovery images in coronal and axial planes with the following parameters: Repetition time (TR) = 3998.8, Echo time (TE) = 16. Slice thickness was 5 mm.
- Diffusion weighted images (DWI) in axial plane with the following parameters: Repetition time (TR) = 3340.6, Echo time (TE) = 98.1.

Complementary sequences included SWI for detection of calcifications.

2.3 Image interpretation:

For each patient the exam was loaded on a CD and reviewed on a personal computer using the software RadiAnt DICOM viewer 4.6.9. Image interpretation was done by the researcher and a specialist radiologist (the supervisor).

2.4 Statistical analysis:

The data were analyzed using Microsoft Office Excel software (2016 version) and represented in the form of figures and charts.

An example of the Questionnaire used in our study

Patient's name	
Age	
Gender	
Date of examination	
Clinical presentation:	
MRI findings:	
✓ Abnormal signal intensity	
✓ Ventricular system	
✓ Shifting of midline structures	
✓ Posterior fossa structures	

Chapter Three

Results

3. Results:

20 patients were included in this study, of whom 12 (60%) were males and 8 (40%) were females. Figure 3.1 depicts the gender distribution of the study population.

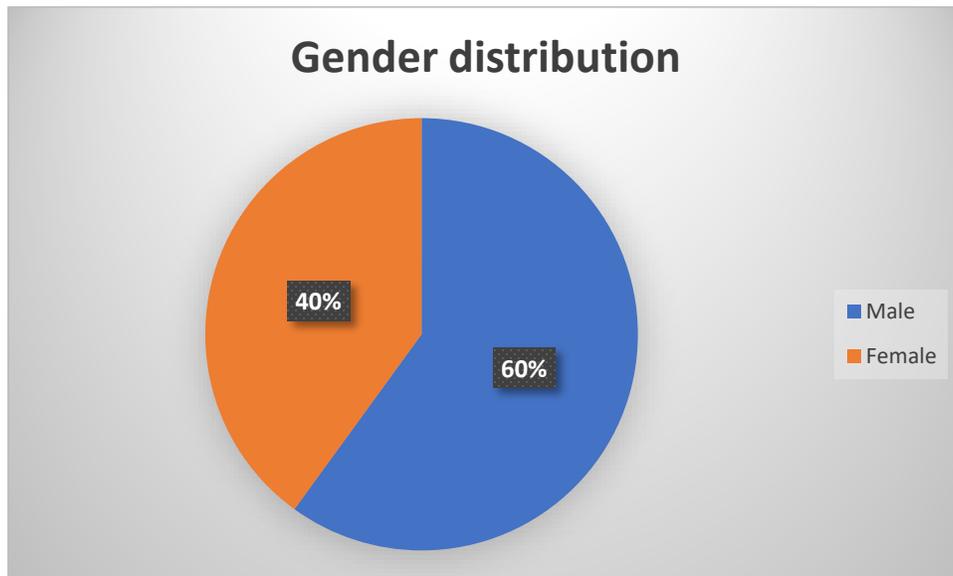


Figure 3.1: The gender distribution of the study sample

The age of the patients in the study population ranged from 9 months to 16 years with the mean age being 7.8 years. The patients were divided into 3 age groups: infants (<1 year), children (1-10 years) and adolescents (11-17 years).

Figure 3.2 shows the distribution of the study population according to age group. 13 patients were in the age group 1-10 years, 6 patients were in the age group 11-17 years and only one patient was <1 year old.

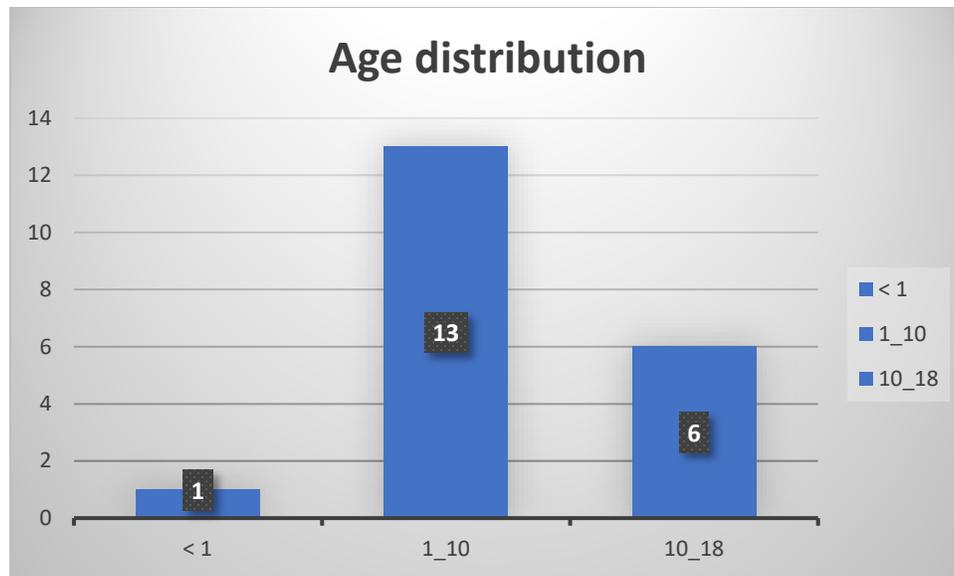


Figure 3.2: Age distribution of the study sample

The clinical types of seizures that the patients presented with are depicted in figure 3.3. 16 patients (80%) presented with focal seizures and 4 patients (20%) presented with generalized tonic clonic seizures.

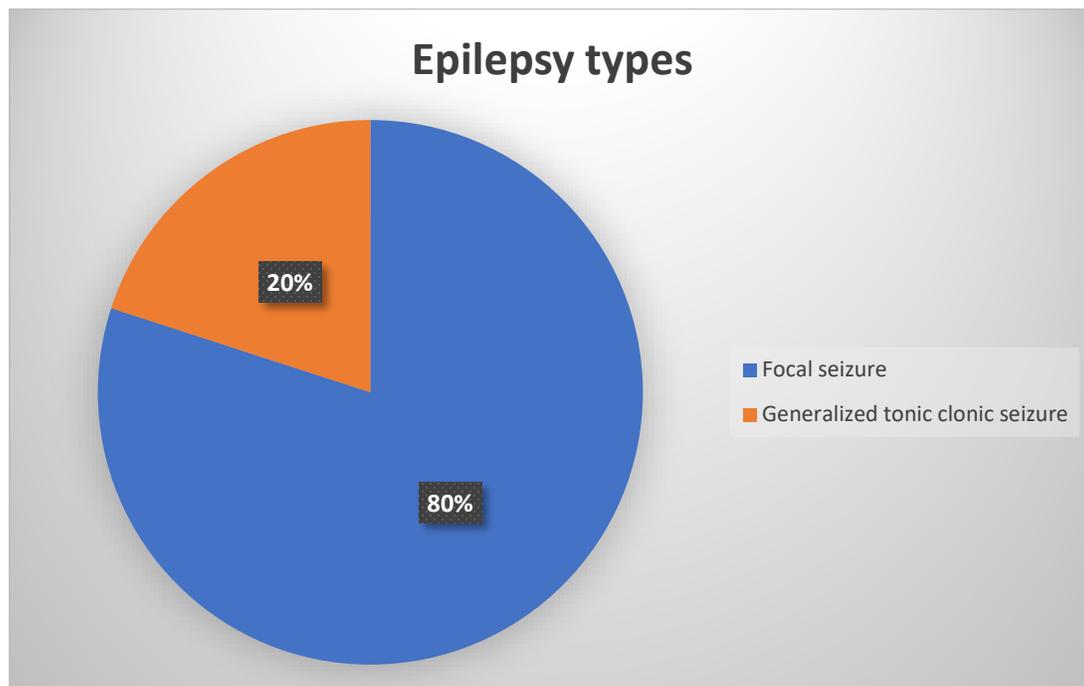


Figure 3.3: distribution of clinical types of epilepsy among the study population

Abnormal MRI findings were obtained in 5 (25%) of the study population while 15 patients (75%) had normal MRI findings (figure 3.4). Of these findings, mesial temporal sclerosis was the commonest being found in 2 patients (10%), followed by focal cortical dysplasia in 1 patient (5%), periventricular heterotopia in 1 patient (5%) and tumor in 1 patient (5%). These results are depicted in figure 3.5

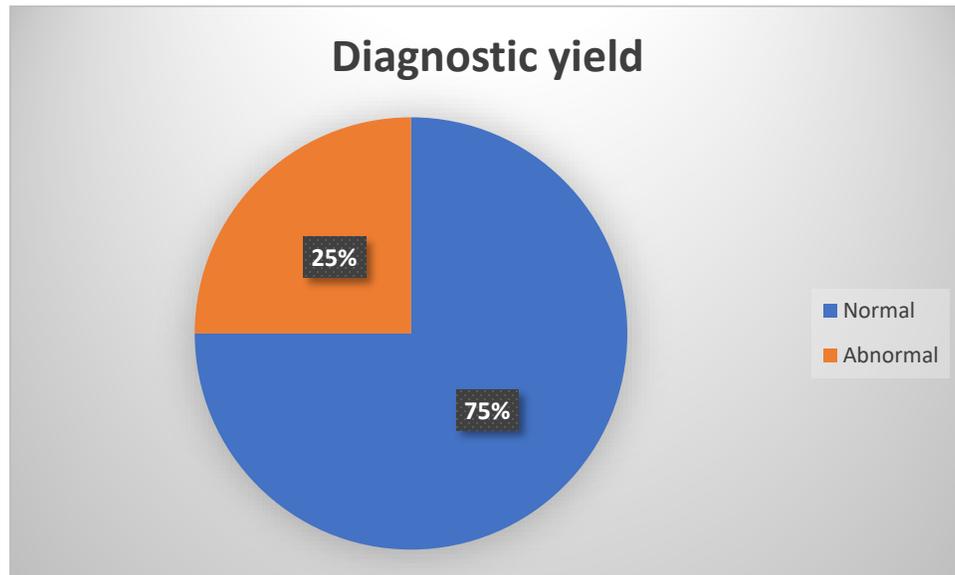


Figure 3.4: The diagnostic yield of the study

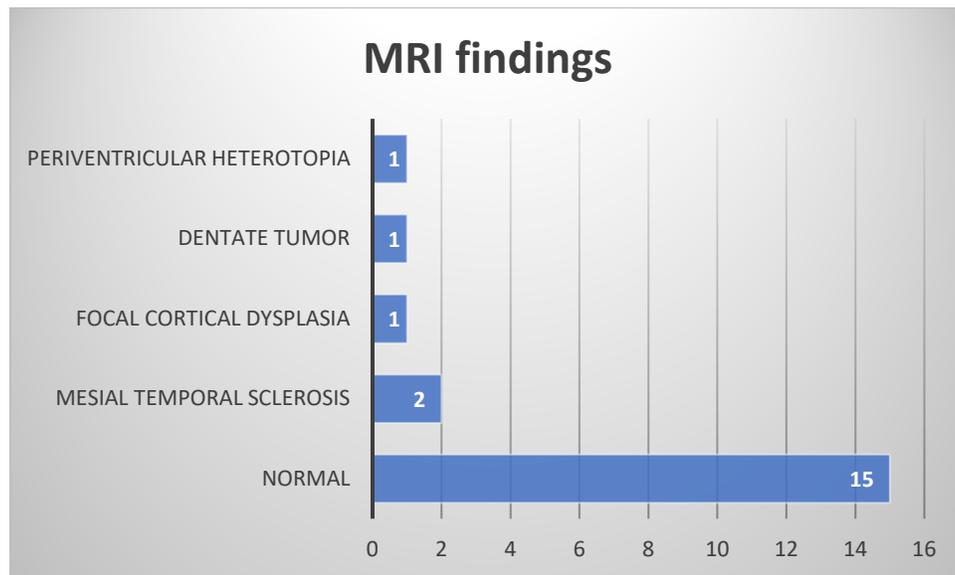


Figure 3.5: The MRI findings among the patients of the study sample

When correlated with epilepsy types, all the abnormal MRI findings were present in patients presenting with focal seizures accounting for 31.25% of all the patients with focal seizures. Normal findings were shown in the rest (68.75%). All the patients with generalized tonic clonic seizures revealed normal MRI examination (100%) and none of them had an abnormal MRI. (Figure 3.6)

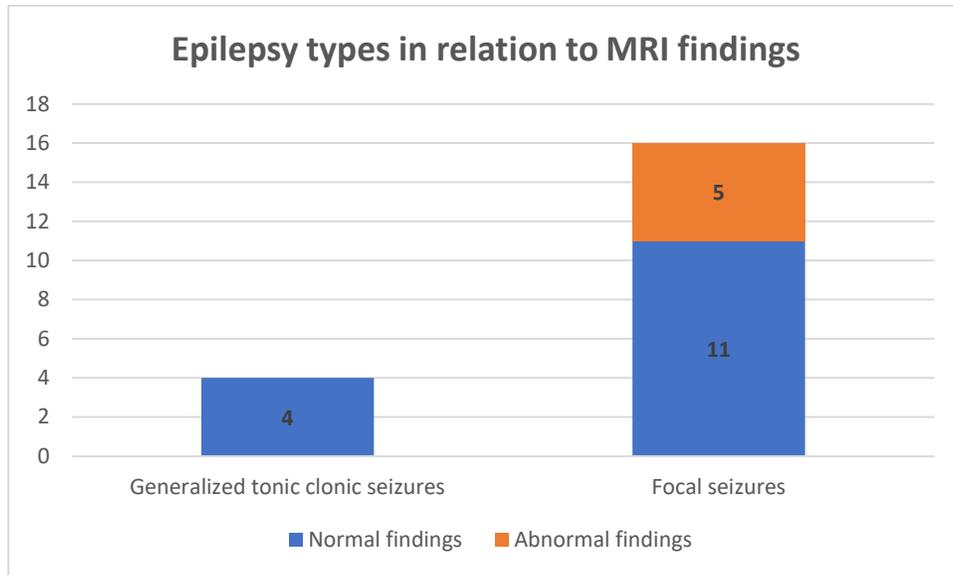


Figure 3.6: Distribution of abnormal MRI findings according to the type of epilepsy

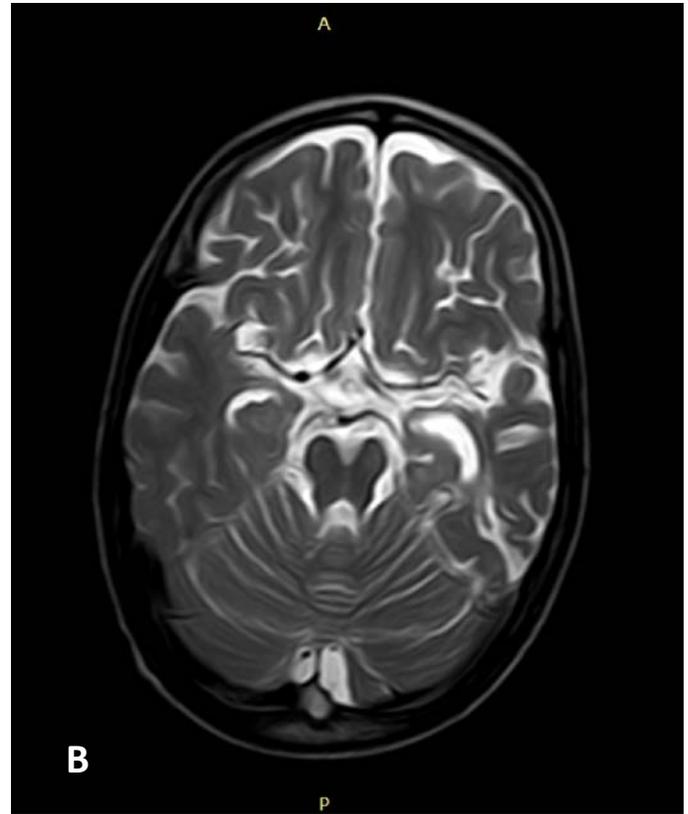
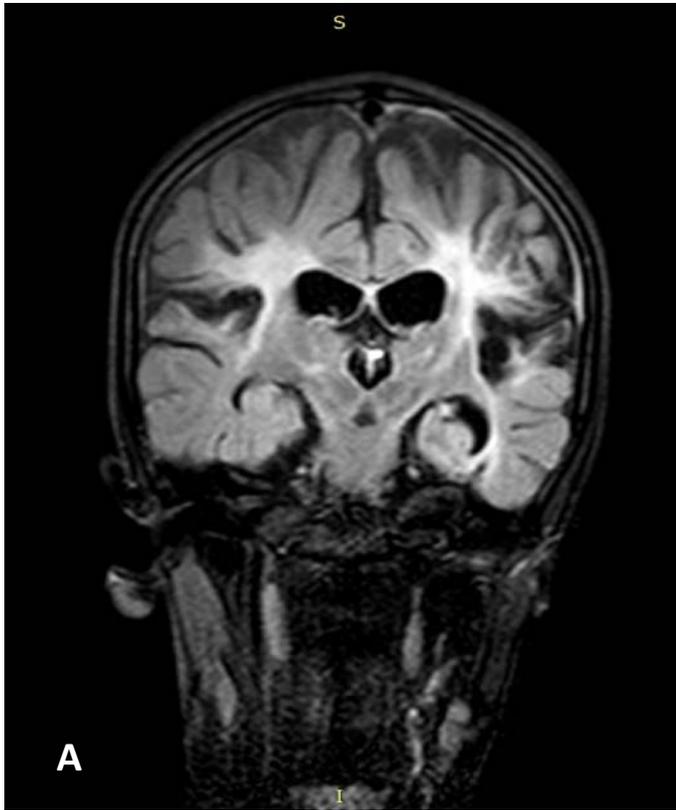
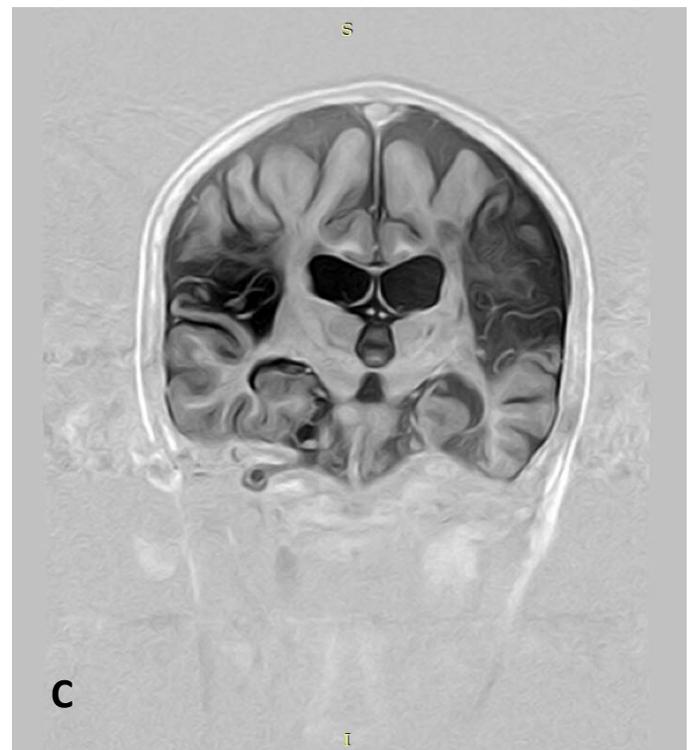


Figure 3.7: Left mesial temporal sclerosis in a 9 yrs. Old male
(A) T2W FLAIR image in coronal section
(B) T2W TSE image in axial section
(C) T1W inversion recovery image in coronal section



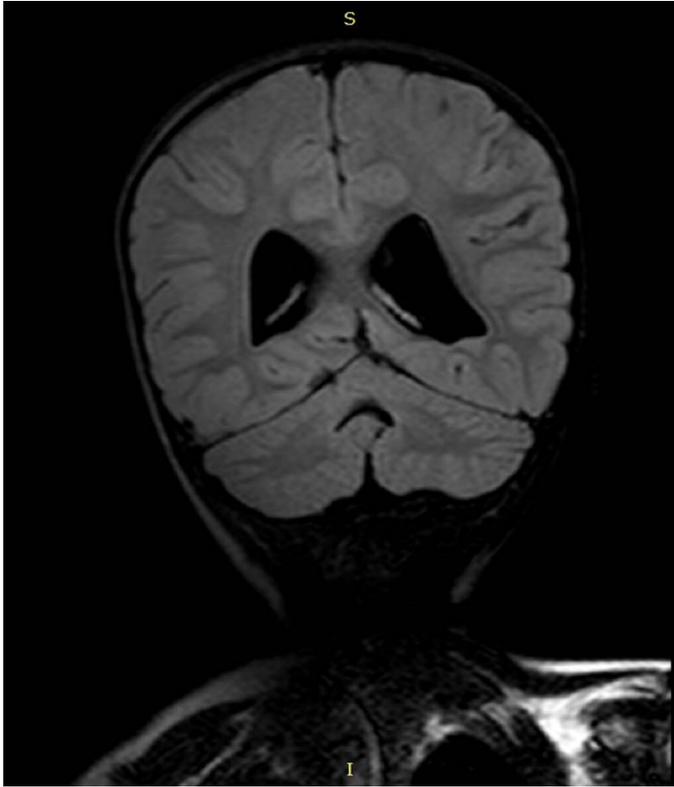
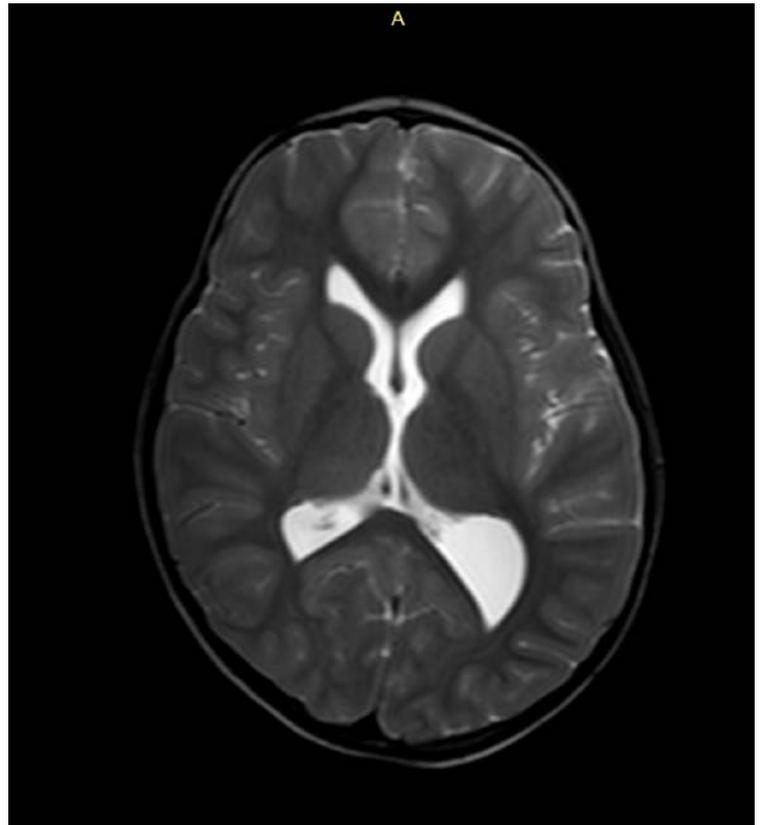


Figure 2: Heterotopia

(A) T2W FLAIR

(B) T1W inversion recovery

(C) T2W-TSE



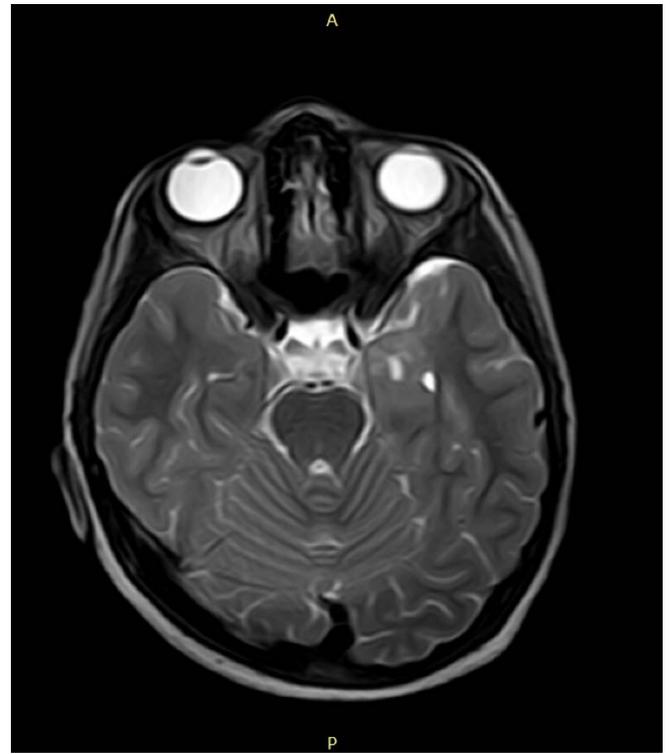
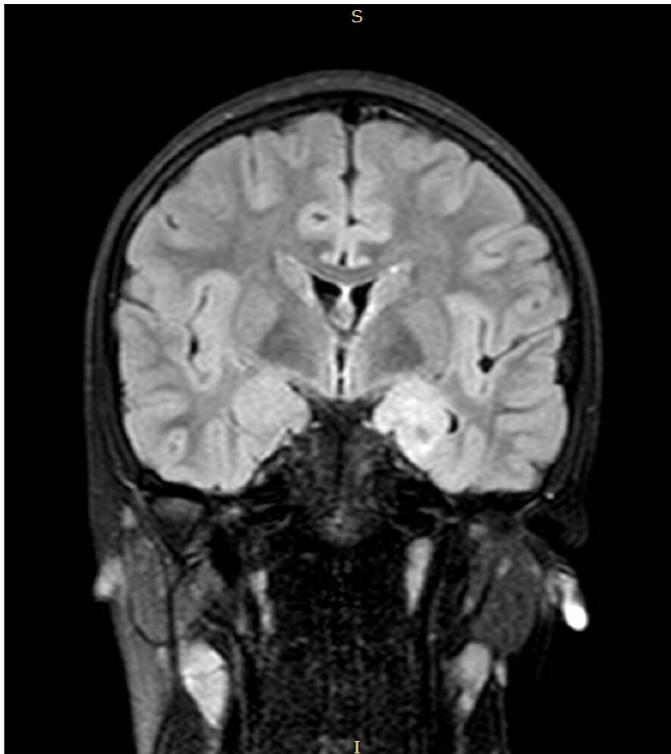
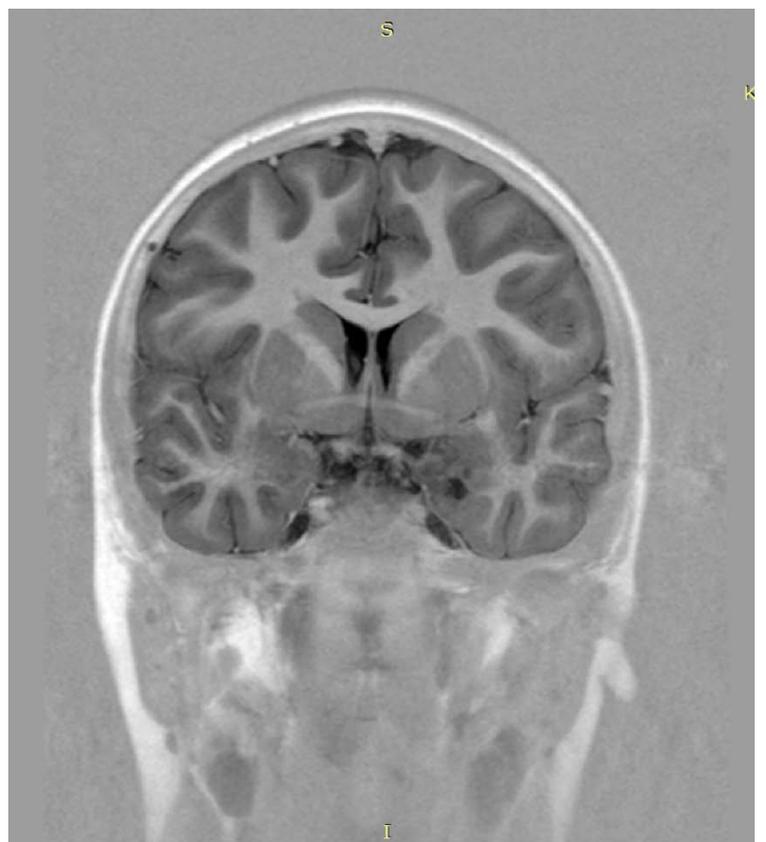


Figure 3.9: dentate gyrus tumor in a 9 yrs. old male

(A) T2W FLAIR coronal section

(B) T2W-TSE axial section

© T1W inversion recovery in coronal section



Chapter Four

Discussion

4. Discussion:

Epilepsy is a common problem worldwide with an age adjusted prevalence of 4–8/1000 in developed countries. (31) The data on epilepsy prevalence in Iraq are insufficient but a study conducted in Baghdad revealed a prevalence of 8.2/1000.(32) Near to 57% of patients who demonstrate with their first epileptic seizure are younger than 25 years, and most of them is 15 years or younger (33).

MRI has been found to be efficient in the evaluation of brain structures and imaging the possible pathologies in the etiology of seizure and other abnormalities in childhood epilepsy. (34)

Most of the patients in our study were in childhood age group (1-10 years) accounting for 65% of the study population.

In our study, the overall MRI abnormalities comprised 25% of the study population which is close to results obtained in a study conducted by Susan A. et al in Iran (35) (28.5%) and a study by T. Dura´ et al in Spain (36) (29.3%) but much lower than another study conducted by Vandana V. et al in India (37) where abnormalities were reported in 48.1% of the patients. Another study conducted by Shinnar and his colleagues (38) on 218 children with first unprovoked seizures and revealed MRI abnormalities in only 12.7% of the patients. This wide range of difference between the studies could be attributed to the different inclusion and exclusion criteria among the different studies.

Regarding the abnormal MRI findings in our study, mesial temporal sclerosis was the commonest finding (10%) followed by heterotopia, focal cortical dysplasia and neoplasm (5% for each). These results were identical to the results reported by Vandana V. et al (37), but they differ from other studies in which other abnormalities were more prevalent such as cerebral atrophy, white matter abnormalities, ventricular enlargement... etc.

Mesial temporal sclerosis has the most common association with intractable temporal lobe epilepsy (TLE). Up to 75-90% of patients with epilepsy due to mesial temporal sclerosis can be cured by anterior temporal lobectomy (39). This signifies the importance of detecting such an abnormality on imaging, although it appears to be underdiagnosed on imaging as it is seen in 65% of autopsy studies for patients with epilepsy.(40)

In our study, focal seizures were more common than the other types of seizures being present in 75% of the study population. This is consistent with the general presentation of such an age group as reported by Hauser et al. (41)

When correlated with the types of epilepsy, the MRI abnormalities found in our study were more prevalent in patients with focal seizures than the other types of seizures which is not surprising for focal seizures and is reported also from other studies as in a study conducted by Kolk et al. (42)

Chapter Five

Conclusion

5. Conclusion:

Magnetic resonance imaging is the modality of choice to evaluate the structural anomaly underlying the seizure disorders and to assess the potential need for surgery.

Focal seizures are the commonest type of seizures occurring in pediatric age group and they are associated with abnormal MRI findings more than other types of seizures.

Mesial temporal sclerosis is an important and a common cause of epilepsy in pediatric age group.

References

References:

- 1- Fisher RS, van Emde Boas W, Blume W, et al. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). *Epilepsia* 2005;46:470–472.
- 2- Fisher, R. S., Acevedo, C., Arzimanoglou, A., Bogacz, A., Cross, J. H., Elger, C. E., ... Wiebe, S. (2014). ILAE Official Report: A practical clinical definition of epilepsy. *Epilepsia*, 55(4), 475-482.
- 3- Management of provoked seizure Usha Kant Misra and Jayantee Kalita
- 4- David Y Ko, MD; Chief Editor: Selim R Benbadis, MD. *Epilepsy and Seizures Clinical Presentation*. Medscape, 2018.
- 5- Robert S. Fisher, J. Helen Cross. Operational classification of seizure types by the International League Against Epilepsy: Position Paper of the ILAE Commission for Classification and Terminology. *Epilepsia*, 58(4):522–530, 2017
- 6- Carl E. Stafstrom. *Back to Basics: The Pathophysiology of Epileptic Seizures: A Primer For Pediatricians*
- 7- T. Dura´-Trave´, S. Aguilera Albesa, M. E. Yoldi-Petri, J. Esparza-Estau´n, F. Gallinas-Victoriano and A. Sagastibelza-Zabaleta; Magnetic resonance imaging abnormalities in children with Epilepsy. *European Journal of Neurology* 2012, doi:10.1111/j.1468-1331.2011.03640.x
- 8- Gaillard WD, Chiron C, Cross JH, Harvey AS, Kuzniecky R, Hertz-Pannier L, Vezina LG; Guidelines for imaging infants and children with recent-onset epilepsy. *Epilepsia*, 50(9):2147–2153, 2009.
- 9- Summary of populations who do and do not warrant imaging. *Epilepsia*, 50(9):2147–2153, 2009 doi: 10.1111/j.1528-1167.2009.02075.x
- 10- Kuzniecky RI, Knowlton RC. Neuroimaging of epilepsy. *Semin Neurol*. 2002 Sep;22(3):279-88.
- 11- Roy T, Pandit A. Neuroimaging in epilepsy. *Ann Indian Acad Neurol*. 2011;14.
- 12- Cepeda C, Andre VM, Levine MS, et al. Epileptogenesis in pediatric cortical dysplasia: the dysmature cerebral developmental hypothesis. *Epilepsy Behav* 2006;9:219–235.
- 13- Kabat J, Król P. Focal cortical dysplasia - review. *Pol J Radiol*. 2012;77 (2): 35-43. Free text at pubmed - Pubmed citation
- 14- Blumcke I, Thom M, Aronica E, et al. The clinicopathologic spectrum of focal cortical dysplasias: A consensus classification proposed by an ad hoc Task Force of the ILAE. *Diagnostic Methods Commission Epilepsia*. 2011;52(1):158–74.
- 15- Guerreiro MM, Andermann F, Andermann E, et al. Surgical treatment of epilepsy in tuberous sclerosis: strategies and results in 18 patients. *Neurology* 1998;51:1263–1269.

- 16- Baron Y, Barkovich AJ. MR imaging of tuberous sclerosis in neonates and young infants. *AJNR Am J Neuroradiol* 1999;20:907–916.
- 17- Barkovich AJ, Kjos BO. Gray matter heterotopias: MR characteristics and correlation with developmental and neurologic manifestations. *Radiology*. 1992;182 (2): 493-9. *Radiology* (abstract) - Pubmed citation
- 18- Abdel razek AA, Kandell AY, Elsorogy LG et-al. Disorders of cortical formation: MR imaging features. *AJNR Am J Neuroradiol*. 2009;30 (1): 4-11. doi:10.3174/ajnr.A1223 - Pubmed citation
- 19- Leite CC, Lucato LT, Sato JR et-al. Multivoxel proton MR spectroscopy in malformations of cortical development. *AJNR Am J Neuroradiol*. 28 (6): 1071-5. doi:10.3174/ajnr.A0511 - Pubmed citation
- 20- Di Rocco C, Battaglia D, Pietrini D, Piastra M, Massimi L. Hemimegalencephaly: clinical implications and surgical treatment. *Childs Nerv Syst* 2006;22:852–866.
- 21- Yagishita A, Arai N, Tamagawa K, Oda M. Hemimegalencephaly: signal changes suggesting abnormal myelination on MRI. *Neuroradiology* 1998;40: 734–738.
- 22- Bocti C, Robitaille Y, Diadori P, et al. The pathological basis of temporal lobe epilepsy in childhood. *Neurology* 2003;60:191–195.
- 23- Mathern GW, Babb TL, Vickrey BG, Melendez M, Pretorius JK. The clinical-pathogenic mechanisms of hippocampal neuron loss and surgical outcomes in temporal lobe ep
- 24- Cascino GD, Jack CR Jr, Parisi JE, et al. Magnetic resonance imaging-based volume studies in temporal lobe epilepsy: pathological correlations. *Ann Neurol* 1991;30:31–36
- 25- Raybaud C, Shroff M, Rutka JT, Chuang SH. Imaging surgical epilepsy in children. *Childs Nerv Syst* 2006;22:786–809.
- 26- Koeller KK, Henry JM. From the archives of the AFIP. Superficial gliomas: radiologic-pathologic correlation. *RadioGraphics* 2001;21:1533–1556
- 27- Di Rocco C, Tamburrini G. Sturge-Weber syndrome. *Childs Nerv Syst* 2006;22:909–921.
- 28- Elster AD, Chen MY. MR imaging of Sturge-Weber syndrome: role of gadopentetate dimeglumine and gradient-echo techniques. *AJNR Am J Neuroradiol* 1990;11:685–689.
- 29- Guermazi A, De Kerviler E, Zagdanski AM, Frijia J. Diagnostic imaging of choroid plexus disease. *Clin Radiol* 2000;55:503–516.
- 30- Griffiths PD. Sturge-Weber syndrome revisited: the role of neuroradiology. *Neuropediatrics* 1996; 27:284–294.
- 31- T M Salmenpera, J S Duncan. Imaging in Epilepsy. *J Neurol Neurosurg Psychiatry* 2005;76(Suppl III):iii2–iii10. doi: 10.1136/jnnp.2005.075135
- 32- Wadad K. Mohammed, Sabah A. Jafaar. Quality Of Life for Patients with Epilepsy in Baghdad City. 2013.

- 33- Armon K, Stephenson T, Gabriel V, MacFaul R, Eccleston P, Werneke U, Smith S. Determining the common medical presenting problems to an accident and emergency department. *Arch Dis Child* 2001;84(5):390-2.
- 34- Gaillard WD, Chiron C, Cross JH, Harvey AS, Kuzniecky R, Hertz-Pannier L, Vezina LG; ILAE, Committee for Neuroimaging, Subcommittee for Pediatric. Guidelines for imaging infants and children with recent-onset epilepsy. *Epilepsia* 2009;50(9):2147-53.
- 35- Susan Amirsalari, Amin Saburi, Reza Hadi, Mohammad Torkaman, Fatemeh Beiraghdar, Shahla Afsharpayman, and Yasaman Ghazavi. Magnetic Resonance Imaging Findings in Epileptic Children and its Relation to Clinical and Demographic Findings
- 36- T. Dura´ -Trave´ a, M. E. Yoldi-Petria, J. Esparza-Estau´ nb, F. Gallinas-Victorianoa, S. Aguilera- Albesaa and A. Sagastibelza-Zabaletaa. Magnetic resonance imaging abnormalities in children with epilepsy. *European Journal of Neurology*, doi:10.1111/j.1468-1331.2011.03640.x
- 37- Vandana V. Ahluwalia, Neelmani Sharma, Ankita Chauhan, Shamrendra Narayan, Perna Singh Saharan, Dipti Agarwal. MRI imaging in afebrile pediatric epilepsy: experience sharing. *International Journal of Contemporary Pediatrics*. 2017 Jan;4:300-305.
- 38- Shinnar S, O'Dell C, Mitnick R, Berg AT, Moshe SL. Neuroimaging abnormalities in children with an apparent first unprovoked seizure. *Epilepsy Res* 2001;43:261–269. [PubMed: 11248538]
- 39- Kasasbeh A, Hwang EC, Steger-May K et-al. Association of magnetic resonance imaging identification of mesial temporal sclerosis with pathological diagnosis and surgical outcomes in children following epilepsy surgery. *J Neurosurg Pediatr*. 2012;9 (5): 552-61.
- 40- Camacho DL, Castillo M. MR imaging of temporal lobe epilepsy. *Semin. Ultrasound CT MR*. 2007;28 (6): 424-36. - Pubmed citation
- 41- Hauser, W. Epidemiology of epilepsy in children. In: Pellock, J.; Dodson, W.; Bourgeois, B., editors.
- 42- Kolk A, Beilmann A, Tomberg T, Napa A, Talvik T. Neurocognitive development of children with congenital unilateral brain lesion and epilepsy. *Brain Dev* 2001;23:88–96. [PubMed: 11248457]