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Spectrum of etiological factors on MRI brain in childhood epilepsies in Kashmiri population: A tertiary care institute based analysis with brief description of causative factors

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Abstract

Introduction: Epilepsies are common causes of childhood morbidity and are characterized by multiple seizure episodes Many etiological factors have been found to be responsible, broadly classified as genetic, structural, metabolic and unknown. Proper diagnosis has significant influence on management strategy. MRI is the investigation of choice for evaluation of such cases because of its high spatial resolution, multiplanar imaging, advanced sequences and no risk of radiation exposure. Our study was primarily aimed at diagnosing the causative factors in childhood epilepsy with their relative distributions and imaging features.

Materials and Methods: We conducted our study in Skims Soura and Skims Medical College Bemina over a period of 16 months from November 2019 to April 2021. MRI was performed in children upto 16 years of age presenting with childhood epilepsy. A total of 200 patients were taken for the study.

Results: Out of 200 patients, MRI abnormalities were observed in 78 cases (39%). HIE was the most common cause (22 cases), followed by structural malformations (20 cases), Inborn errors of metabolism (20 cases), CNS infections (8 cases), MTLS (7 cases), CNS neoplasms (5 cases), Demyelinating disorders (4 cases) and Phakomatosis (2 cases).

Conclusion: MRI with its advanced sequences and better resolution due to advancement of technology has assumed significant importance in the evaluation of childhood epilepsies. HIE was the most important causative factor in our study group emphasizing the proper management of high-risk patients thereby reducing the incidence of antenatal/perinatal ischemic insults.

Keywords: MRI (Magnetic Resonance imaging), HIE (Hypoxic Ischaemic Encephalopathy), MTLS (Mesial Temporal Lobe Sclerosis), GTCS (Generalised Tonic Clonic Seizures), CT (Computerised Tomography)

Introduction

A seizure is defined as a sudden paroxysmal electrical discharge from the CNS resulting in involuntary movements, sensory or autonomic disturbances with or without alteration in sensorium[1,2]. Epilepsy is a condition in which seizures are triggered recurrently. International league against epilepsy defines epilepsy as; at least two unprovoked or reflex seizures more than 24 hours apart[2].

Prevalence of epilepsy is more in neonatal period; almost 10% in term and 20% in preterm[1]. Epilepsies are broadly divided into following categories: genetic, structural, metabolic and unknown[3,4]. MRI abnormalities are more often seen in epilepsies with localising signs. 50% of individual studies in children with localization related new onset seizures were reported to be abnormal[5]. Imaging studies in childhood absence epilepsy, juvenile myoclonic epilepsy and benign childhood epilepsy did not identify significant structural abnormality.

Neuroimaging is a core investigation in the evaluation of early childhood epilepsy which occurs in 1-2/1000 children[6,7,8]. Computed Tomography scanning can be used as an initial imaging modality in emergency situations; However, is inferior to MRI owing to its inferior resolution and risk of radiation exposure. CT is better at detecting calcifications and haemorrhage and has rapid acquisition time. However, its use is limited due to its reduced sensitivity which is not higher than 30% in unselected populations[9].

Magnetic Resonance Imaging (MRI) is considered as the imaging of choice because of its superior anatomic resolution and characterization of pathologic processes[5]. With the increased availability of high-quality MRI, lesions like heterotopias and MTLS are easily diagnosed which was not previously possible on CT scans [2,10,11]. Lack of radiation is another advantage of MRI over CT scan. Children younger than 2 years require special sequences as immature myelination affects the ability to identify common causes of epilepsy. Lesions may appear or disappear as myelination patterns change indicating possibility of cortical malformations[5].

MRI has significant impact on management of new onset seizures. In perinatal onset seizures, treatment can be initiated if MRI diagnoses a lesion likely being the causative factor because the risk of repeated seizures approaches 80%. Similarly, patients with temporal lobe epilepsy are strong candidates for intervention if MRI diagnosis of MTS is established[14,15]. Structural brain abnormalities are best diagnosed on MRI, and MRI dictates appropriate treatment. Patients with Malformations of Cortical Development (MCD) require lifelong antiepileptic drugs. Those with focal MCDs are amenable to surgical intervention. As a rule, the more widespread the MCD, the worse developmental outcome is expected[16].

Inborn errors of metabolism are important causes of childhood epilepsies and most common disorders associated with seizures are: Non ketotic hyperglycaemia, organic aciduria, urea cycle defects, Zellweger syndrome, Molybdenum cofactor deficiency and hypoglycemia[17]. MRI is particularly helpful in disorders with some specific imaging characteristics. Additionally, MRI can be helpful in molecular diagnosis of chemicals present in diseased brain aiding in further diagnosis[17,18].

HIE is a major cause of acute mortality, cerebral palsy, seizures and mental retardation. Ulegyria lesions are significantly associated with epilepsy mainly involving parasagittal watershed areas[19]. Seizures are one of the most common symptoms in paediatric brain tumours. Supratentorial tumours involving grey matter are more epileptogenic especially dysembryoplastic neuroepithelial tumours and gangliogliomas. Besides, viral, bacterial and tubercular meningoencephalitis can present with seizures.

Our study is aimed at presenting the distribution of these diseases on MRI imaging in childhood epilepsies in native Kashmiri population and relative prevalence of these diseases.

Materials and Methods

The study was conducted in SKIMS Soura and its affiliated SKIMS Medical College, Bemina over a period of 16 months, from November 2019 to April 2021. Children up to the age of 16 years were selected for the study with previous documentation of epilepsy where MRI was indicated and done in our setup. Written clinical history and consent were taken for the study.

MRI was done using 1.5Tesla Philips MRI located in SKIMS Medical College Bemina and 1.5Tesla Siemens, Erlangen, Germany MRI located in SKIMS Soura. MRI protocols include T1WI sequence in axial and sagittal planes, T2WI sequence in axial sagittal and coronal planes, FLAIR WI sequence in axial plane. DWI and SWI sequences were also done. Dedicated epilepsy protocol using special thin section coronal sequences including phase corrected and magnitude corrected sequences were done in all patients. Additionally, gadolinium enhanced contrast studies and MRS were done in selected patients as indicated.

Results

We conducted our study on 200 cases of childhood epilepsy. MRI abnormalities were noted in 78 out of 200 cases (39%) with normal MRI scans in rest of the 122 cases (61%). Gender wise, males were the predominant group making 65.5% (131 cases) with females comprising about 34.5% (69 cases) of the total cases; 57 cases were in the age group of upto one year (28.5%), 59 cases were in the age group of 1-5 years (29.5%) with the remaining 84 cases in the age group of 5-16 years (42%). GTCS was the predominant type of convulsion noted in 97 cases (48.5%).

Relative percentage of etiologic agents identified on MRI Brain

Disease type		Number of cases	Percentage (%)
Hypoxic ischemic encephalopathy (HIE) (22 cases)	Periventricular HIE	8	10.3
	Cystic HIE	7	9.0
	Subcortical term HIE	5	6.4
	Basal ganglia predominant	3	3.8
Structural malformations (20 cases)	Lissencephaly with Pachygyria/ Agyria complex	7	9.0
	Dysgenetic corpus callosum	4	5.1
	Schizencephaly	3	3.8
	Dandy Walker Malformations	3	3.8
	Arachnoid cysts	2	2.6
Inborn errors of metabolism (10 cases)		10	12.8
CNS Infections (8 cases)	Viral meningoencephalitis	3	3.8
	Tubercular meningoencephalitis	3	3.8
	Bacterial meningitis	2	2.6

MTLS		7	9.0
CNS neoplasms		5	6.5
Demyelinating disorders		4	5.1
Phakomatosis		2	2.6

Discussion

We conducted our study with primary motive of understanding the relative occurrence of diseases affecting CNS as causative factors of childhood epilepsies in Kashmiri population with brief description of MRI findings of these diseases. MRI abnormalities were noted in 39% of all cases in our study group (72 out of 200 cases). There is wide variation in MRI abnormality percentage in literature; Likewise, K.R Swepson noted 56.4% positivity percentage, 70.4% by **Rachna Chaurasia et al**[3], 41.7% by **Wang et al**[22] and 48.9% **by Chang et al**[23]. Our findings were comparable to study **by Wang et al**[22]. This difference is likely attributed to relative ease of access to MRI, expertise of paediatrician and strictness of criteria for MRI evaluation of childhood epilepsies. Males were more affected than females with male to female ratio of 1.9:1. Most of the studies we came across showed male predominance of disease occurrence [1,21,24]. Our findings were concordant with the study conducted by **R.A Umap et al**[1] where male to female ratio was 1.8:1[1]. Children younger than one years of age contributed significantly to total number of cases making 28.5% of the total. GTCS was the most common type of seizure disorder (49.5%) in our scenario.

Hypoxic ischemic encephalopathy (HIE) was the most common cause of childhood epilepsy in our study (28.2%). These findings were comparable to the study conducted by **Umap et al**[1] where HIE contributed to 31.5% of total cases. Similar results were noted in other studies as well[25]. However, CNS infections were found to be the major cause of epilepsy in other studies[3,21]. Imaging findings in HIE are variable depending on the age of ischemic insult, brain maturity and severity of the ischemic insult. During the maturation period of brain, different parts of brain have different vascularity with reduced vascularity of periventricular white matter in preterm brains (<36 weeks gestational age) which moves to the subcortical water shed zone as brain matures. Hence periventricular leukomalacia and germinal matrix haemorrhage is seen in preterm HIE. Periventricular leukomalacia (PVL) is most frequently noted adjacent to the trigone of lateral ventricles and adjacent to the foramina of monro[26]. Dilated ventricles due to reduced volume of periventricular white matter and irregular ventricular margins are important signs of PVL. End stage PVL can present with periventricular white matter cystic changes. Basal ganglia structures are highly vascular structures in term babies, however, are not affected in mild to moderate term HIE because of auto regulatory mechanisms diverting blood supply to vital structures like basal ganglia protecting them from ischemic changes. However, deep grey matter nuclei are affected in severe term HIE. Mild to moderate term HIE presents with subcortical watershed region ischemic changes and encephalomalacia predominantly involving parieto-occipital regions. Cystic HIE is a severe form of HIE presenting with cystic replacement of brain parenchyma and may show blood products of varying ages in cyst cavities in few cases. In our scenario, periventricular HIE was the most common type (8 cases), followed in order by cystic HIE (7 cases), Subcortical term HIE (5 cases) and basal ganglia predominant HIE (3 cases).

Structural malformations were the second leading causes of childhood epilepsies in our study group making 25.6% of total cases. Several studies have

proven structural malformations as predominant causes of childhood epilepsies especially in infants[26,27,28]. Lissencephaly with pachygyria-agyria Complex and heterotopias were the most common type (7 cases) followed by Dysgenetic Corpus callosum (4 cases), Schizencephaly (3 cases), Dandy Walker malformations (3 cases) and arachnoid cyst s (2 cases). Pachygyria-agyria Complex presents as smooth cortical surface with reduced gyration. Severe cases present with total loss of gyration with figure of 8 configuration. Heterotopias occur due to interruption of normal neuronal myelination from ventricles to the cerebral cortex and can be of nodular and diffuse type and are characterized by grey matter signal areas in abnormal locations. These are important causes of localising epilepsies.

Hemimegalencephaly is characterized by hamartomatous growth of the cerebrum or part of it with dilated ipsilateral lateral ventricles, cortical malformations and macrocrania. Focal cortical dysplasias present with focal altered signal of thick cortex and subcortical white matter appearing hyperintense onT2/FLAIR WI. Schizencephaly manifests as grey matter lined cleft extending from the ependyma to the pia mater; can be open lip and close lip type. Open lip Schizencephaly presents as grey matter lined clefts extending from ependyma to cortex whereas close lip type presents as nipple like clefts from ependyma not extending to cortical surface. Dandy Walker malformation presents as hypoplastic vermis with cystic dilatation of fourth ventricle extending posteriorly with enlarged posterior fossa. Dandy Walker variant present as less severe form with lesser degree of hypoplasia of cerebellar vermis. Arachnoid cysts are common benign cystic lesions in subarachnoid space compressing adjacent brain parenchyma with no ventricular communication and are less frequently associated with seizure disorders. Dysgenetic Corpus callosum presents as varying degree of hypoplasia of Corpus callosum which can be isolated or part of other disorders.

Seizure is a frequent symptom of metabolic diseases but epilepsy syndromes due to inborn errors of metabolism are rather rare[17,29]. we had 10 cases of inborn errors of metabolism making 10.2% of total cases. Inborn errors of metabolism are a diverse group with overlapping MRI brain findings yet can sometimes be diagnosed due to specific predilection of brain parts by some disorders which may show diffusion restriction. MRS may also be helpful by quantifying Chemicals present in abnormal areas like glycine in Nonketotic Hyperglycaemia, branched Chain amino acids in Maple Syrup Urine disease and N-acetyl aspartate in Canavan's disease. In Phenylketonuria, MRI shows periventricular hyperintensities showing diffusion restriction which can be reversed by Low phenylalanine diet. Leigh disease presents as high signal characteristically involving putamen with brain stem, periaqueductal and midbrain involvement. Glutaric aciduria typically presents as dilated extra-axial CSF spaces with bat wing configuration and T2/FLAIR hyperintensity of bilateral globi pallidii and deep white matter. Similarly, organic acid disorders like Propionic Aciduria, Methylmalonic Aciduria and Urea cycle disorders may show some specific MRI features aiding in proper diagnosis. Also, clinicolaboratory evaluation with MRI correlation further aids in diagnosis.

Infections were the fourth most common causes of seizure disorders in our study group with eight cases making 10.2% of total cases. This is in significant contrast to many studies which showed infections as the

leading causes of seizure disorders[1,3]. Study by **Rachna Chaurasia et al[3]** found tuberculosis and neurocysticercosis as leading causes of seizure disorder in Central India which may be explained by high burden of these diseases in the population studied. Relatively low percentage of CNS infections documented by MRI could be due to lesser referrals for MRI brain in children with febrile seizures already diagnosed by clinic laboratory methods. Hence the actual incidence of CNS infections leading to seizure disorders would be higher than documented by MRI studies in our study group. There were 3 cases of viral meningoencephalitis, 3 cases of tuberculous meningoencephalitis and 2 cases of bacterial meningitis in our study group. Herpes simplex virus encephalitis is the most common viral causative agent with bilateral limbic system, MTLs, and frontal lobe involvement which may show diffusion restriction and haemorrhages in some cases[31]. CNS tuberculosis can have myriad of presentations, like pachymeningitis, basal exudates, meningeal enhancement, cerebral tuberculomas showing variable stages of signal characteristics and enhancement patterns. Bacterial meningitis presents with meningeal and ependymal enhancement with mild intraventricular and Subarachnoid exudates.

Mesial temporal lobe sclerosis (MTLS) is an important curable cause of childhood epilepsy. We had 7 cases of MTLS comprising 9% of the total cases. MTLS is now being increasingly diagnosed because of improved MRI resolution and use of advanced sequences. Features of MTLS include reduced hippocampal volume, altered hippocampal signal, dilated temporal horns, atrophy of ipsilateral hippocampus, collateral white matter and temporal lobes. Loss of interdigitations of hippocampus, altered signal of mammillary bodies and fornixes are additional findings[32]. There were 4 cases of demyelinating disorders presenting with seizures in our study group (5.1 % of the total). This is comparable to the study by **Aarti Anand et al[33]** with MTLS making 3.6 % of total cases. Multiple sclerosis and ADEM are the two most important causes of childhood demyelinating disorders. Clinically isolated syndrome (C.I.S) presents with few periventricular white matter hyperintensities as compared to ADEM where deep grey nuclei and cortical grey matter is involved along with large periventricular white matter lesions.

We had 5 epilepsy causing childhood tumours in our study (6.5%). Seizures are a common presentation of paediatric brain tumours especially in supratentorial tumours with grey matter involvement. Features associated with increased risk include supratentorial location, grey matter involvement, low grade tumours and certain histological features like Oligodendroglioma, D-NET and Ganglioglioma[34]. We had two cases with Phakomatosis; one was tuberous sclerosis showing cortical and subcortical tubers (showing high signal on T2 and low signal on T1WI) and subependymal nodules. Subependymal giant Cell astrocytoma can also be noted which was not seen in our case. Another was a case of Sturge-Weber syndrome[35]. Our case presented with right sided hemi atrophy of brain on MRI with prominent leptomeningeal enhancement. Blooming on SWI was also noted,

Our study had few limitations. Firstly, it was based on a relatively small group of patients. Also, patients referred for MRI were included in the study with no first-hand role in patient selection. We have around four MRI studies with motion artifacts reducing image quality. Nevertheless, this study throws light on prevalence of causative factors of childhood epilepsies

in Kashmiri population and need of interventions at appropriate level to prevent/cure childhood epilepsies.

Conclusion:

Childhood epilepsies are an important cause of morbidity in children affecting socio developmental aspects of a child. Proper diagnosis helps in proper management of such cases. MRI plays a pivotal role in diagnosing childhood epilepsies because of its excellent soft tissue resolution, advanced sequences, multiplanar imaging with no exposure to ionizing radiations. MRI best diagnoses structural, developmental and metabolic causes of epilepsy and has proven useful in diagnosis of MTLS. HIE was the most common cause of epilepsy in our setup emphasising the need of proper management of late pregnancies and identification of pregnant women at risk of prolonged labour/ foetal distress which could be managed at tertiary care centres with advanced facilities available thereby reducing the chances and severity of HIE.

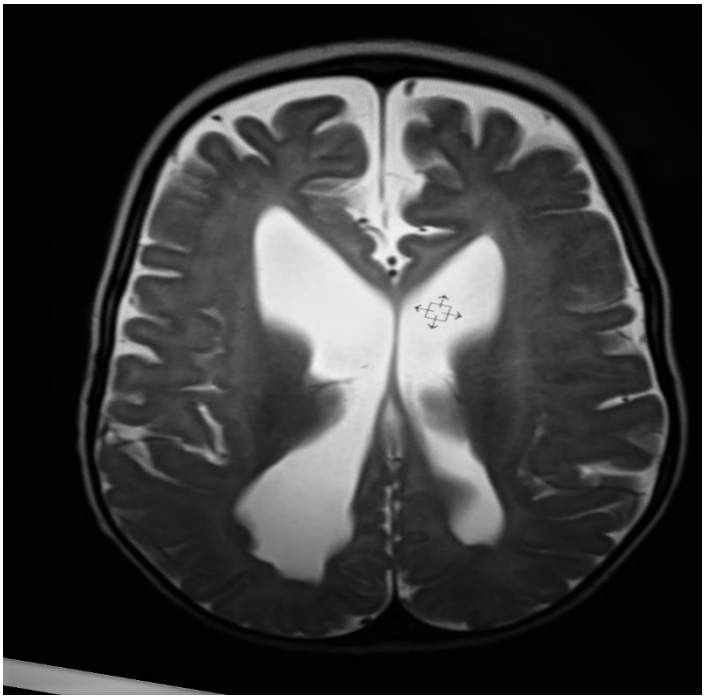

Figure 1: Term HIE. Dilated lateral ventricles with reduced Subcortical white in bilateral cerebral hemispheres Predominantly frontal lobe involvement.

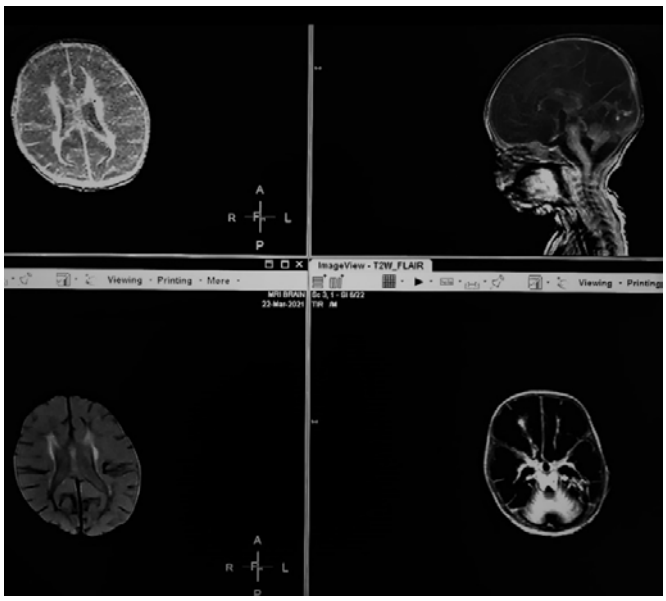

Figure 2: Cystic HIE showing severe replacement of brain Parenchyma with cystic spaces. Severe atrophy of bilateral deep grey matter nuclei with gliosis also noted.

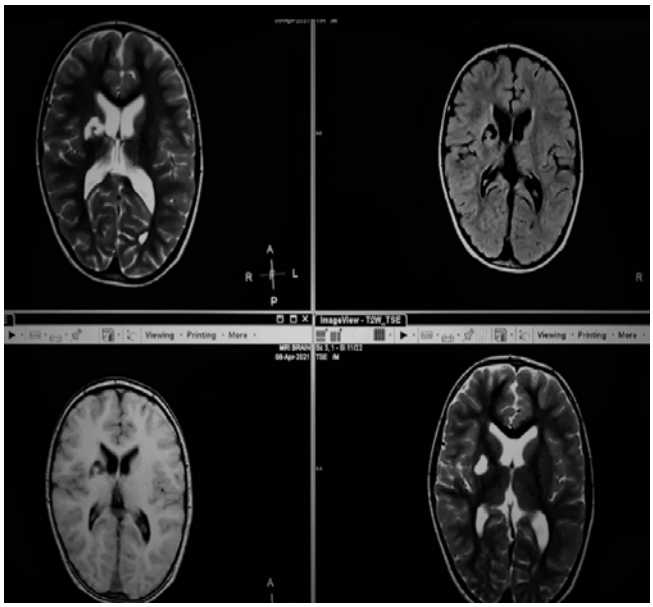

Figure 3: Periventricular cystic HIE with porencephalic cyst formation in preterm HIE involving right frontal region.

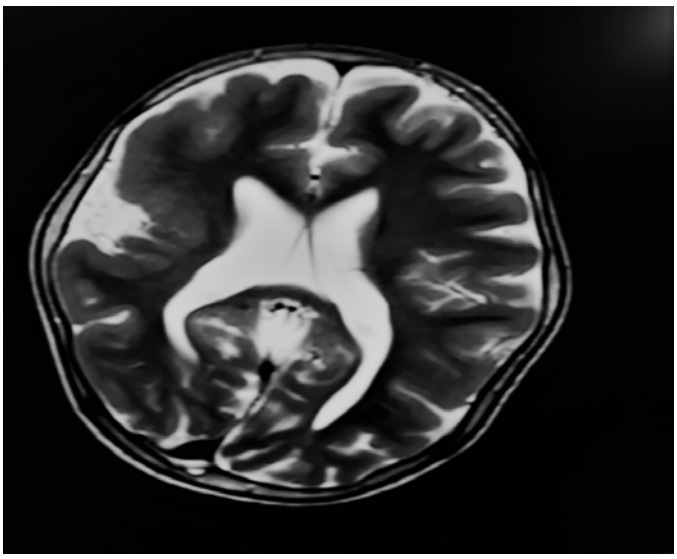

Figure 4: Pachygyria agyria complex showing reduced gyri And sulci leading to smoothened cortical surface involving right frontoparietal region with associated mild ventriculomegaly.

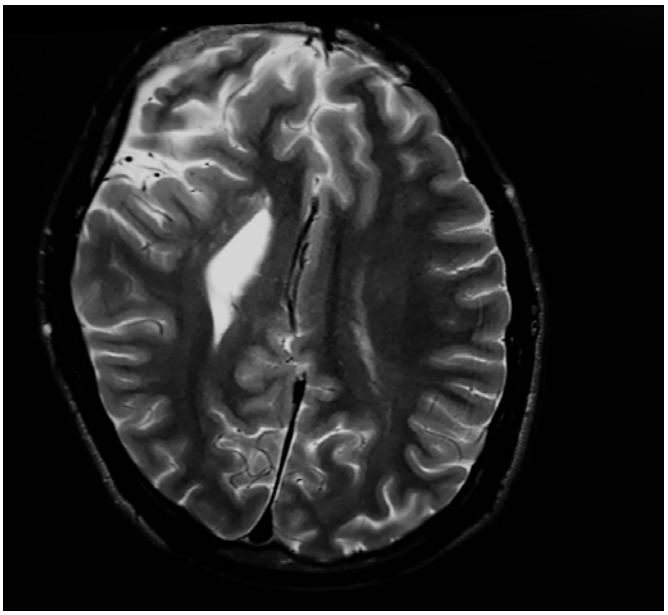

Figure 5: Hemimegalancephaly. Right frontoparietal polymicrogyria and subcortical heterotopia with dilated ipsilateral lateral ventricle and sylvian fissure

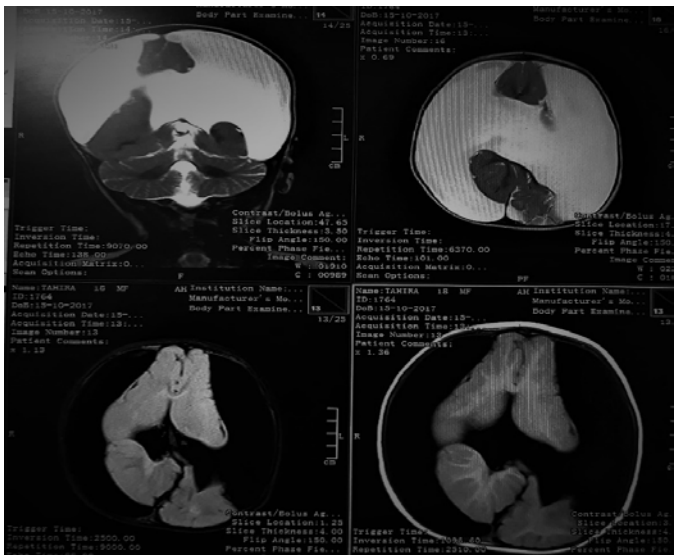

Figure 6: Bilateral open lip Schizencephaly showing cleft like CSF spaces extending from ependyma to cortical surface.

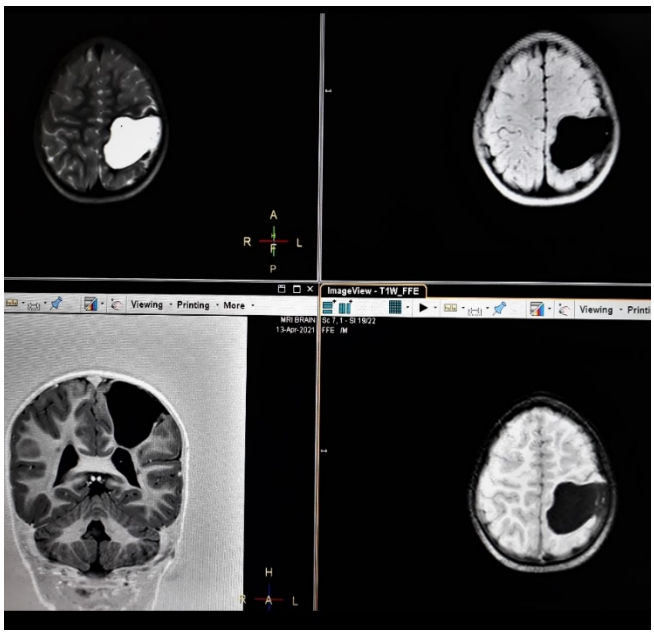

Figure 7: Arachnoid cyst. Well defined cystic lesion in left parietal region compressing adjacent brain with no ventricular communication.

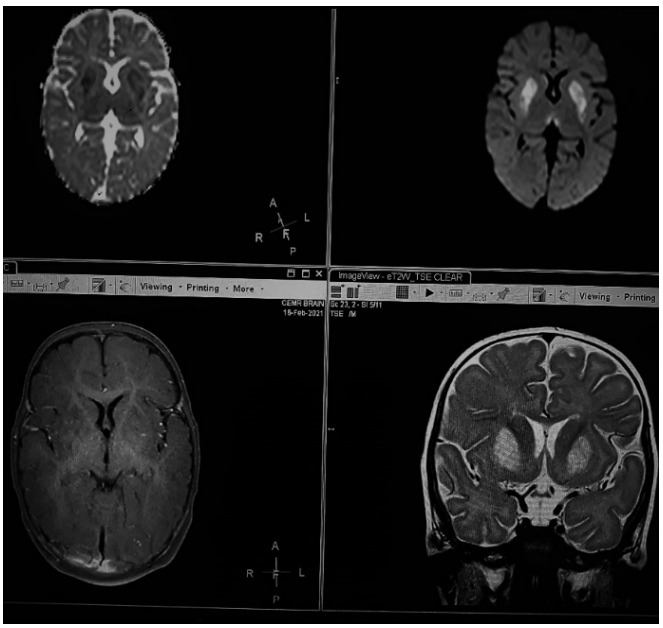

Figure 8: Leighs disease. Bilateral basal ganglia hyperintensity showing diffusion restriction and no significant postcontrast enhancement.

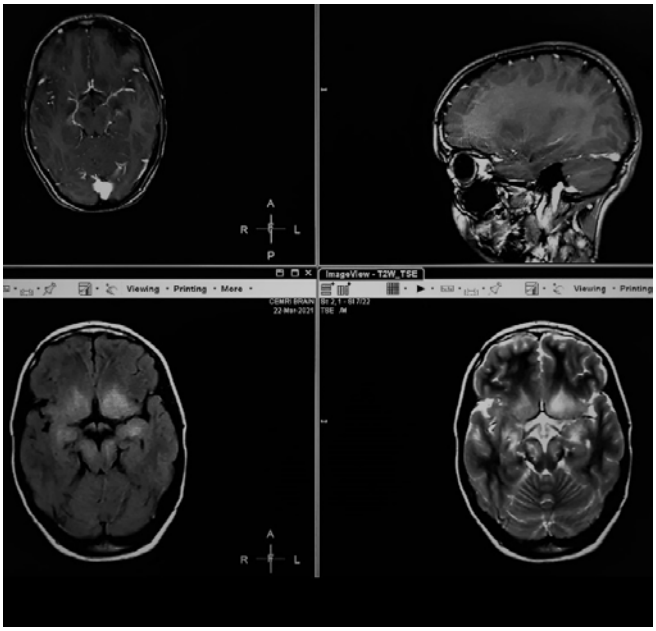

Figure 9: Herpes Simplex Encephalitis. T2, FLAIR hyperintensities In bilateral basifrontal regions, MTLs and midbrain regions.

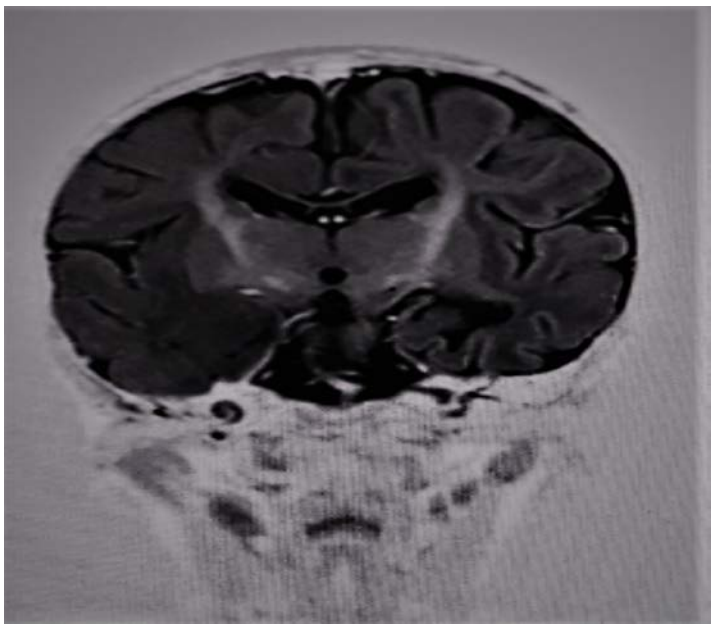

Figure 10: Hippocampal atrophy involving left hippocampus With dilated ipsilateral temporal horn and reduced digitations.

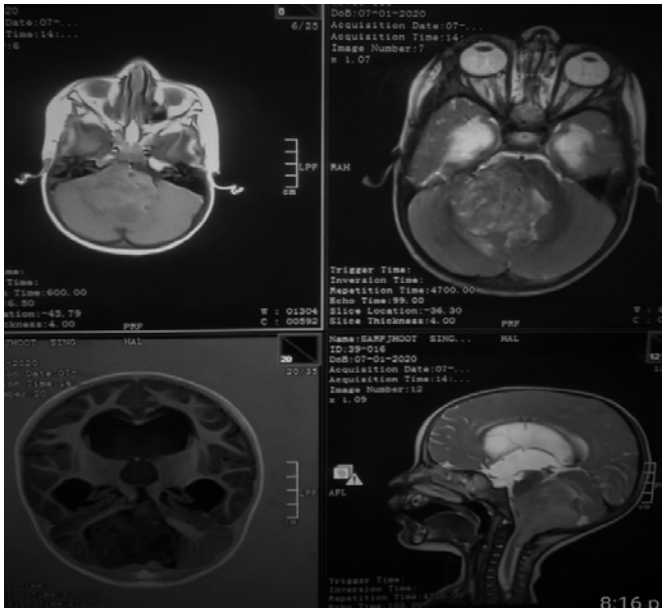

Figure 11: Ependymoma. A heterogenous signal intensity lesion occupying 4th ventricle and extending to lateralforamina with resultant obstructive hydrocephalus.

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