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Varied Spectrum of Sequelae to Hypoxic Ischemic Encephalopathy on Magnetic Resonance Imaging

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ABSTRACT:
Background*: Hypoxic-ischemic encephalopathy (HIE), is one of the most frequent and serious neurological insult found in both neonates as well as adults. This is a neuro-vascular and neuro-metabolic syndrome, caused by a shortage of supply of oxygen and glucose or their metabolism in the brain. It is caused by a global hypoperfusion or oxygenation deficiency rather than from infarction in a specific vascular cerebral territory. Magnetic resonance imaging (MRI) is the most sensitive and safe modality for evaluating these patterns of brain injury.*
Materials and Methods*: In this case report, we discuss 3 uncommon cases of complicated hypoxic ischemic brain injury in patients who came to DR. D. Y. Patil Medical Hospital, Navi Mumbai. All scans were done on 1.5 Tesla MRI machine.*
Conclusion*: This report describes the clinical presentation and radiological features of these 3 rare cases along with a brief review of literature of these complications.*

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I. INTRODUCTION

Perinatal asphyxia still remains one of the most significant causes of mortality and long-term morbidity. Almost 20-22% of neonatal deaths in India is caused by perinatal asphyxia. Basic pathophysiology being impairment of the cerebral blood flow which in turn leads to fetal vascular compromise either in utero or in immediate postnatal period. Intrauterine asphyxia is a result of interrupted placental blood flow and gas exchanges in concordance with various fetal factors like fetal hemorrhage, placental hypoperfusion, impaired maternal oxygenation or disrupted umbilical circulation.

Asphyxia in the perinatal period is the most important cause of HIE, causing hypoxemia and hypercapnia thus leading to reduction in blood pressure and blood flow. This then triggers the cascade of events, which include acidosis, release of inflammatory mediators and neurotransmitters, generation of free radicals, calcium accumulation and lipid peroxidation. These accumulated biochemical metabolites cause failure in vascular autoregulation and thereby decreased cerebral perfusion, leading to neuronal cell death.

Hypoxic-ischemic encephalopathy is one of the most common causes of cerebral palsy and other severe neurological deficits in children, occurring in 2-9 of every 1000 live births.

Neuroimaging particularly MR imaging with its increased sensitivity and specificity and relatively acceptable availability, plays a significant role in diagnosis and in turn influencing early intervention in cases of HIE. In addition, imaging studies performed in the subacute stages of injury provide valuable data on the severity and extent of insult and can also be helpful in predicting long-term prognosis.

There is broad spectrum of imaging findings in HIE, which are highly variable and depend on a number of factors, including brain maturity at the time of insult, severity and duration of insult and timing of imaging studies. As early imaging findings can be subtle and are often overlooked. Therefore, awareness of the many patterns of injury that may be seen while addressing special focus on areas that are most likely to be injured when reporting the cases suspected of HIE is a necessity.

In this article, we review some of the uncommon and lesser seen magnetic resonance features of complication patterns of HIE.

PROCEDURE AND METHODOLOGY

After written informed consent was obtained, detailed clinical history was collected. It included socio-demographic characteristics such as age, gender, nationality, consanguineous marriage, antenatal / immediate postnatal complications or any insults and parental / familial lifestyle habits like smoking and alcohol.

It was supplemented by relevant clinical and biochemistry laboratory investigations such as complete blood count, CSF examination studies and optional specialized liver and renal function tests (to rule out other causes).

Magnetic resonance imaging of subjects was performed on 1.5 Tesla MRI machine at our institute. Adequate information about the study in form of protocol, risks and complications was provided to the concerned subjects in their understandable language. Axial and coronal images of the brain were obtained with T1W, T2W, FLAIR with gradient and diffusion weighted images.

CASE 1

ULEGYRIA – MILD SEQUELAE TO PREVIOUS HYPOXIC ISCHEMIC INJURY

An 18-year-old male child, belonging to middle socioeconomic class, Indian by nationality and who is known case of seizure disorder since childhood, presented to Dr. DY Patil hospital with complaints of recent convulsion episode before 5 days. The seizures were episodic and tonic-clonic in nature with frequency of approximately 2 episodes / month. The patient had no other complaints.

No family history of seizure disorder was found. The patient was born from full term normal vaginal delivery, but didn't cry immediately after birth and was kept under observation for 24 hours.

The patient has had normal mental development and was currently studying in 11^{th} class with adequate academic performance and without any major deficit.

The patient was on Tablet Carbamazepine 200mg and Tablet Levipil 500mg.

The patient hadn't undergone any prior MRIexamination, and presented to our institute with purpose of further management of the condition.

So Magnetic Resonance Imaging (MRI) of brain was performed with epilepsy protocol, which showed focal areas of cortical atrophy and thinning with multiple small shallow sulci and gyri seen involving bilateral fronto-parietal regions, adjacent to the central sulcus with subcortical T2W and FLAIR hyperintense signal, suggestive of ulegyria.

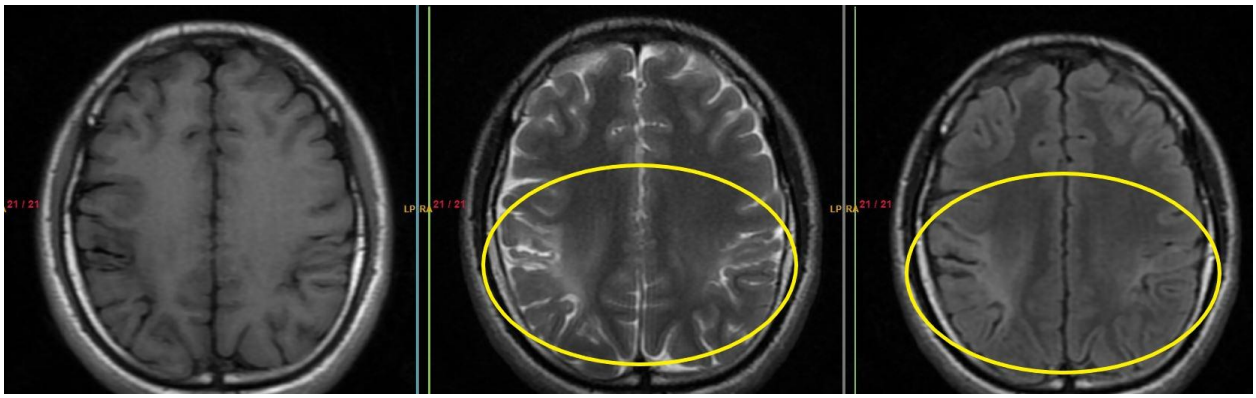


Figure 1: (A) Axial T1W image (left) (B) Axial T2W image (center) and (C) Axial FLAIR image (right) showing focal areas of cortical atrophy and thinning involving bilateral fronto-parietal regions with subcortical T2W and FLAIR hyperintense signal intensities.

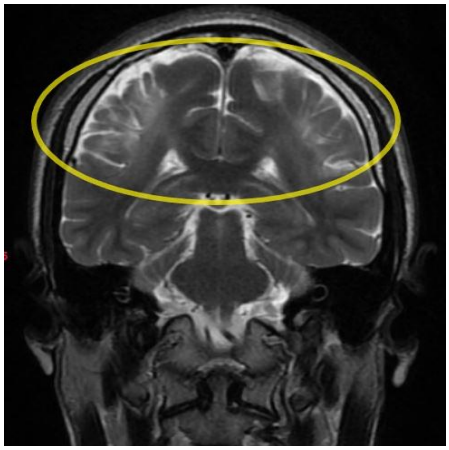


Figure 2: CORONAL T2W imagebetter reveals the T2W hyperintense signal intensity in subcortical white matter of bilateral fronto-parietal regions with cortical thinning, suggestive of ulegyria.

CASE 2

CYSTIC ENCEPHALOMALACIA – COMPLICATION OF HYPOXIC ISCHEMIC INJURY

A 7-month-old female infant, belonging to low socioeconomic class, Indian by nationality, born from non-consanguineous marriage, appropriately vaccinated for age and a case of spastic diplegia cerebral palsy was referred to our institutional hospital from a primary health care center.

No familial history of cerebral palsy was found. The patient was born from full term normal vaginal delivery with delayed cry. Patient was kept in NICU for a week, during which the patient was intubated for 3 days in view of falling oxygen saturation levels (perinatal hypoxia). After which, the patient was discharged as the conditions improved.

On inspecting developmental history, the patient had yet not achieved neck holding or rolling movement, suggestive of developmental delay.

On examination, eye-gazing and tracking were found to be absent. However, tone and reflexes of all four limbs were within normal limits.

MRIexamination of the brain was advised and performed to get relevant information about the severity of the condition.

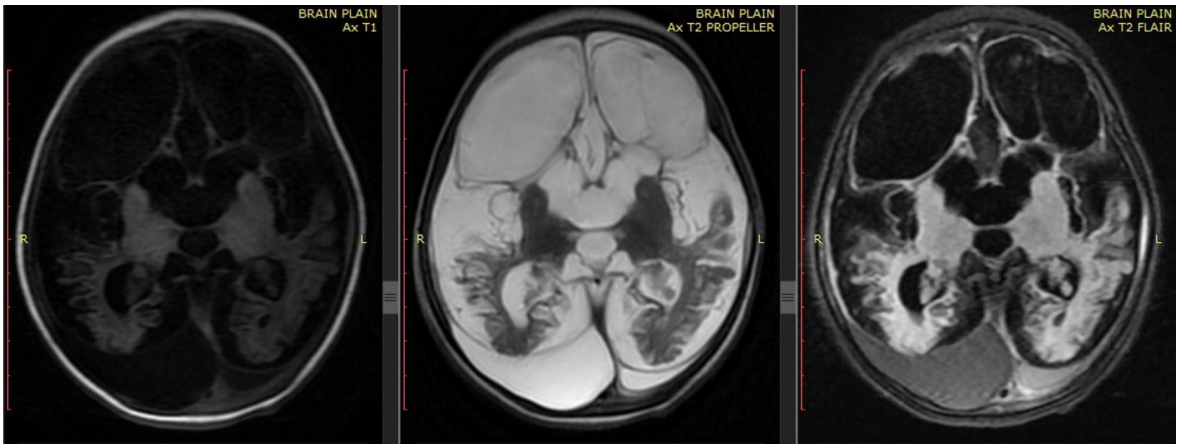


Figure 3:(A) Axial T1W image (left) (B) Axial T2W image (center) and (C) Axial FLAIR image (right) showing diffuse well-defined areas of CSF intensity with adjacent FLAIR hyperintense rim involving both grey and white matter of bilateral cerebral hemispheres, with relative sparing occipital and posterior parietal regions. There is also mild generalized prominence of the cortical sulci----suggestive of benign enlargement of the subarachnoid space in infancy.Well defined extra-axial crescentic areas of altered signal intensity (right > left) which appears hyperintense on T2W and hypointense on T1W images are noted in dependent posterior aspect of bilateral parietal region, representing bilateral subdural hygromas.

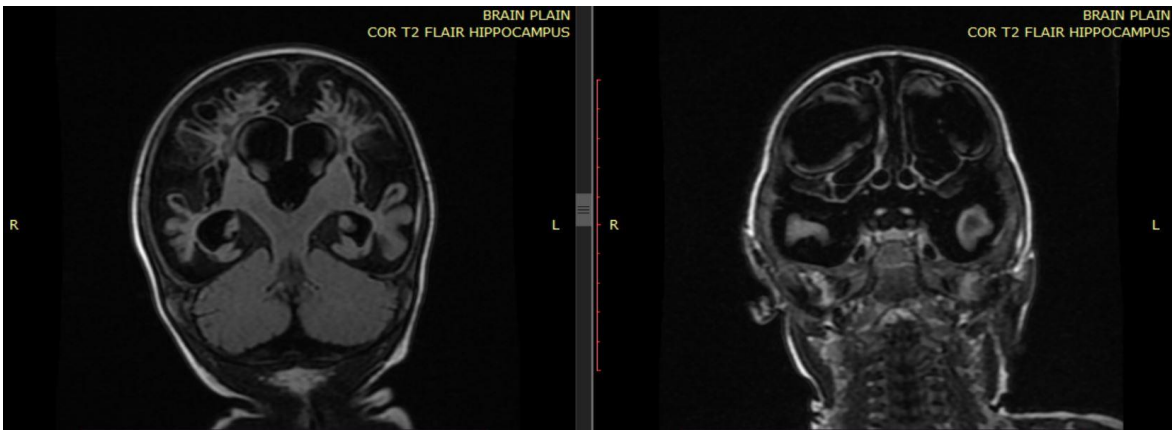


Figure 4:CORONALFLAIR images of the same patient demonstrating diffuse well-defined areas of CSF intensity with adjacent FLAIR hyperintense rim involving both grey and white matter of bilateral cerebral hemispheressuggestive of cystic encephalomalacia.It is seen causing significant thinning of the cortex. However, both cerebellar hemispheres appear normal.

CASE 3

ACUTE NECROTIZING ENCEPHALOPATHY– LIFE THREATENING AND RARE COMPLICATION OF HYPOXIC ISCHEMIC INJURY

A 7-year-oldfemale child, belonging to middle socioeconomic class, presented to Dr. DY Patil hospital with complaints fever, loose stools and convulsions.There was no significant medical history and no recent history of vaccination. Systemic examination showed mild hepatomegaly with the absence of meningeal signs or neck stiffness. She required fluid resuscitation-crystalloid and colloid boluses followed by inotropic support. She was started on high-flow nasal oxygen and other supportive care. Initial investigations showed leukocytosis with neutrophilic predominance (75%) and thrombocytopenia.On day 4 of pediatric ICU stay, the child developed another episode of seizure (generalized tonic clonic) lasting around 10 min. Seizure subsided with levetiracetam, and she was continued on supportive care. Hersensorium continued to fluctuate with intermittent episodes of irritability and drowsiness.

MRIexamination of the brain was advised and showed multiple well-defined rounded lesions in right frontal, left parafalcine parietal, left periventricular, and bilateral cerebellar lobes with minimal postcontrast enhancement, suggestive of ADEM. She was continued on pulse steroids and other supportive care.

In view of worsening sensorium, the child was intubated, mechanically ventilated and neuroprotective measures were initiated. Electroencephalography showed generalized slowing, suggestive of encephalopathy. MRI brain was repeated which showed increase in size of lesions in the bilateral frontoparietal region and cerebellar lobes with blooming on SWI sequence, with evolving obstructive hydrocephalus – suggestive of hemorrhagic ADEM. Neurosurgeon opinion was taken, and she was advised decompression. Parents were also counseled regarding further immunomodulation (plasma exchange) and high risk involved in the same. However, her general condition worsened and parents wanted to transfer to another facility, where the child succumbed to her illness.

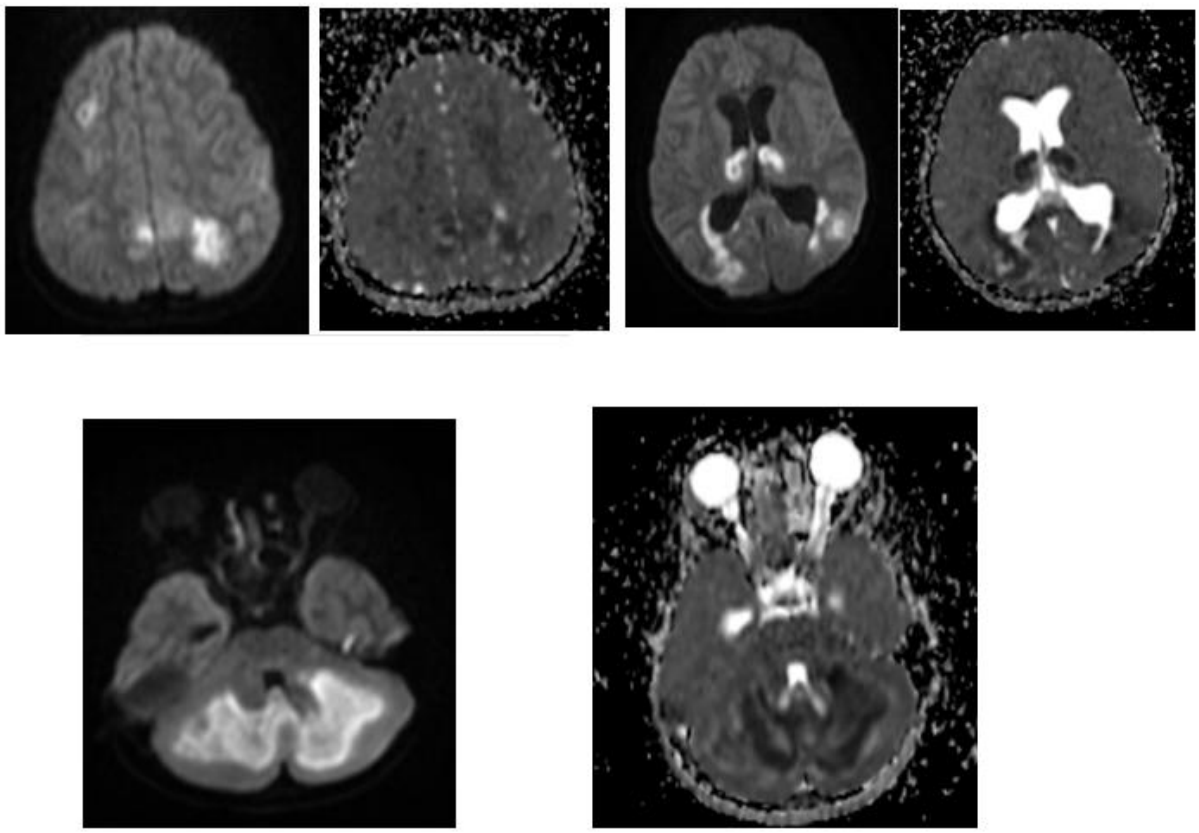


Figure 5:Axial DWI imagesat the level of fronto-parietal lobes, at the level of lateral ventricles and at the level of cerebellar hemispheres showingscattered areas of restricted diffusion (with low ADC values) involving white matter of bilateral fronto-parietal lobes, bilateral peri-ventricular white matter and bilateral cerebellar hemispheres.

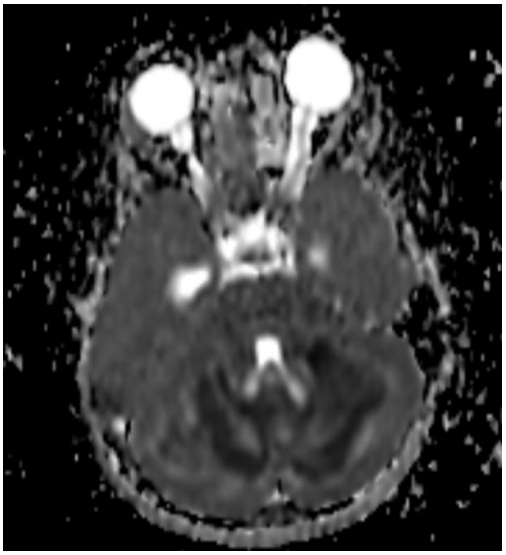


Figure 6: AXIAL T2W image reveals the T2W hyperintense signal intensity in peri-ventricular white matter of bilateral fronto-parietal regions.

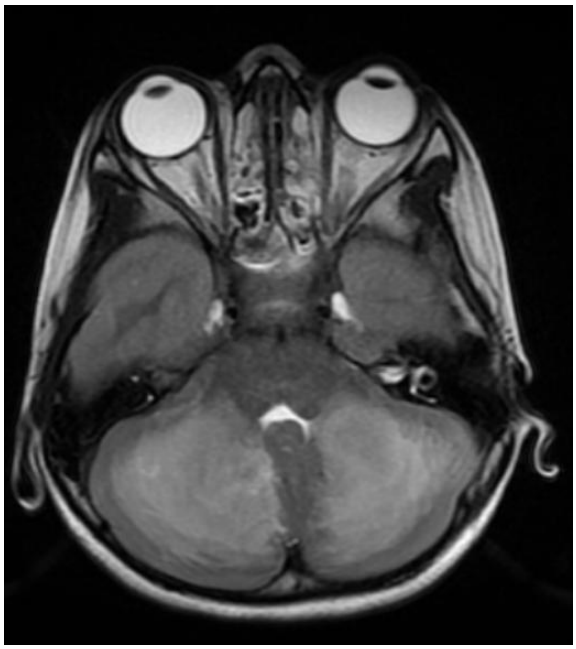
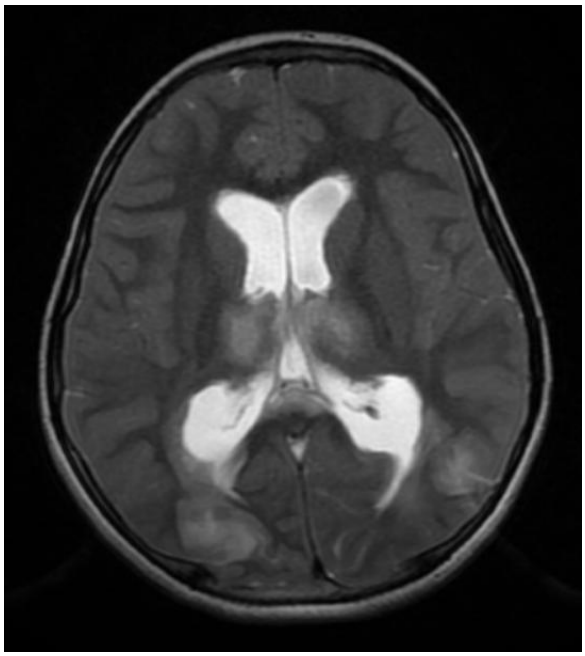


Figure 7: AXIAL T2W image reveals the significantT2W hyperintense signal intensity in white matter of bilateral cerebellar hemispheres.



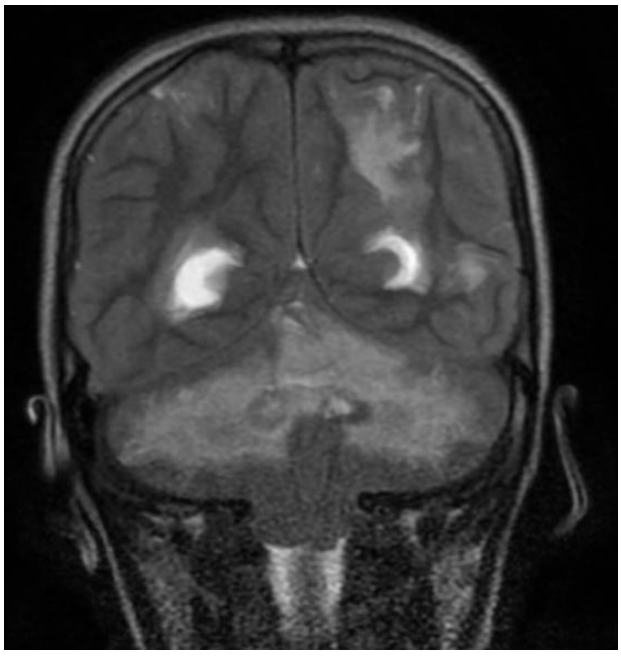


Figure8: CORONAL T2W image reveals the significant T2W hyperintense signal intensity in white matter of bilateral fronto-parietal lobes and cerebellar hemispheres.

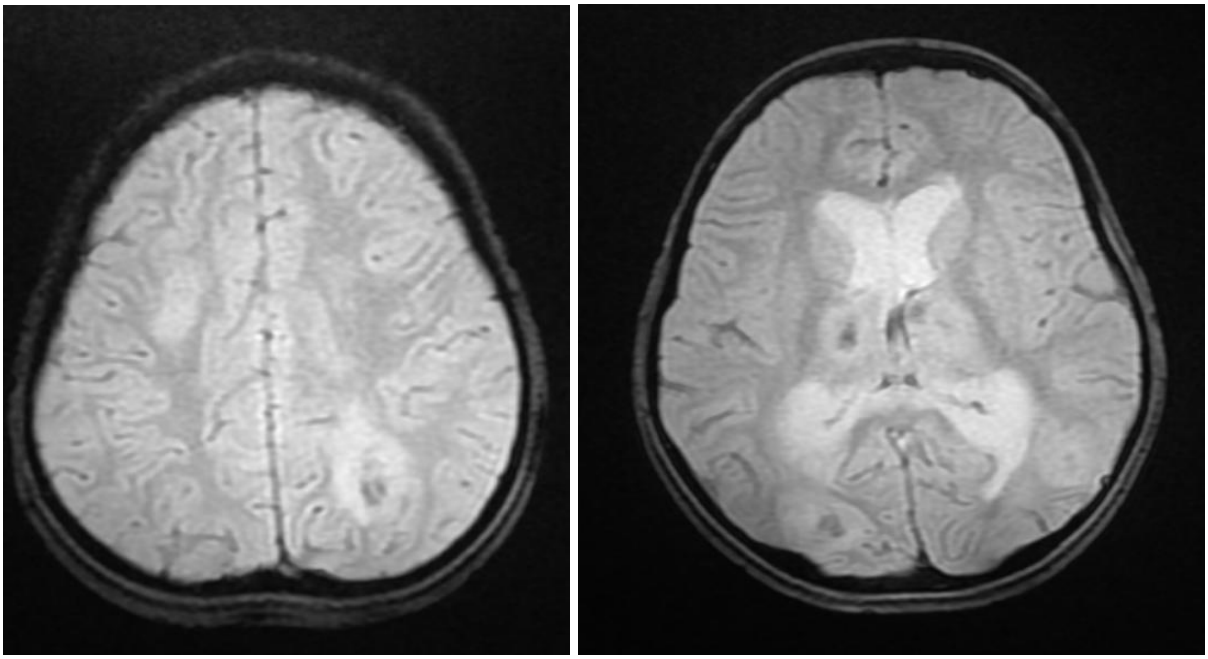


Figure 9:Axial GRADIENT imagesat the level of fronto-parietal lobes demonstrating blooming in areas of white matter hyperintensities in bilateral fronto-parietal lobes and bilateral peri-ventricular white matter.

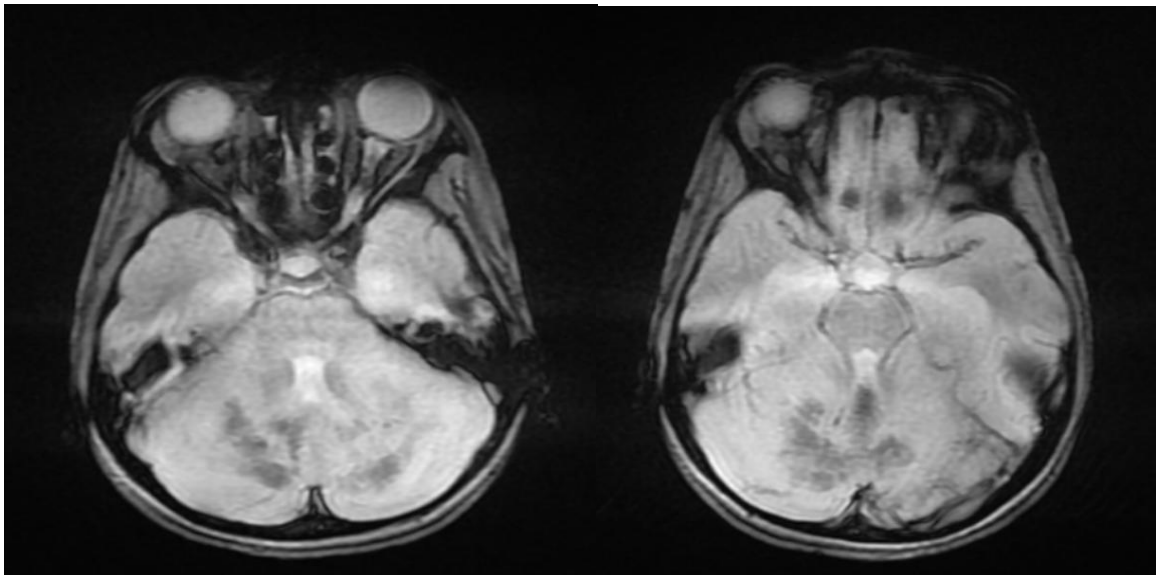


Figure10:Axial GRADIENT imagesat the level of cerebellum demonstrating blooming in areas of white matter hyperintensities in bilateral cerebellar hemispheres and the dentate nuclei.

II. DISCUSSION

- Early recognition and diagnosis of the varied presentations of hypoxic ischemic encephalopathy is of crucial importance of the long-term survival and better quality of life for the patient.

- Studies estimate a short therapeutic window of 2-6 hours during which interventions may be efficacious in reducing the severity of ultimate brain injury; thus, early identification of a neonate who has sustained a hypoxic-ischemic insult is a paramount objective for optimal management and treatment.
- Although term infants with mild encephalopathy generally make a full recovery, 20% of affected infants die in the neonatal period and another 25% develop significant neurologic sequelae. For preterm infants, compared with term infants, the overall prognosis is worse.

- Global ischemic insults depending on the severity and duration of insult and the age of the neonate which makes particular brain areas selectively vulnerable to damage. Areas of high metabolic demand containing a high concentration of excitatory amino acid receptors, such as the deep gray matter and peri-Rolandic cortex are commonly damaged in acute severe ischemia. In comparison to this, less severe but more prolonged ischemic insults affect the cortical and sub-cortical watershed regions. We also found in our study that peri-Rolandic involvement remains the most common area involved in the patients of cerebral palsy.

III. CONCLUSION

Acute hemorrhagic leukoencephalitis is a rare demyelinating disease characterized by an acute rapidly progressive fulminant inflammation of the white matter, first described by Hurst in 1941. It is regarded as the most severe sub-form of acute disseminated encephalomyelitis.

Early diagnosis of acute hemorrhagic leukoencephalitis is critical because it frequently causes severe morbidity within a few days or leads to death, but the patients can survive with early treatment with combinations of corticosteroids, immunoglobulin, cyclophosphamide, and/or plasma exchange.

Imaging findings in hypoxic ischemic injury (HII) are highly variable and depend on a number of factors, including brain maturity, severity and duration of insult, and type and timing of imaging studies. Early imaging findings can be subtle and are often overlooked. Therefore, it is essential to be familiar with the many patterns of injury that may be observed and to focus attention on areas that are most likely to be injured when interpreting studies performed for suspected HII.

If MR imaging performed in the first 24 hours is negative, a repeat examination performed at 2– 4 days may help rule out the possibility of delayed injury.

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