"ROLE OF MAGNETIC RESONANCE IMAGING IN EVALUATION OF HYPOXIC ISCHEMIC ENCEPHALOPATHY IN CHILDREN UNDER FIVE YEARS OF AGE: A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"

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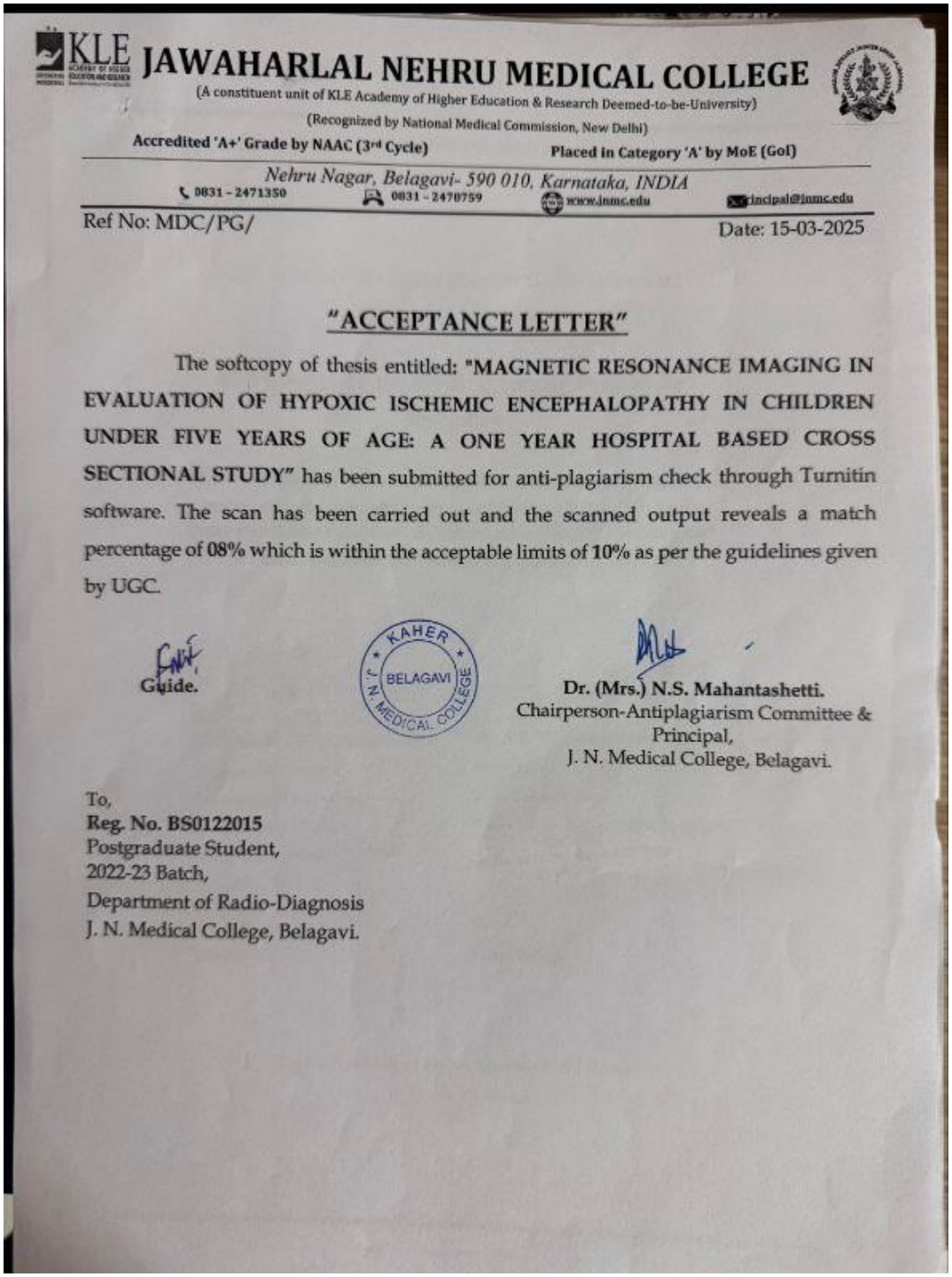

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ABSTRACT

Background

Hypoxic-Ischemic Encephalopathy (HIE) is a serious condition affecting both preterm and term neonates due to inadequate oxygen and blood supply to the brain. It occurs before, during, or after birth and leads to significant neurological impairments such as cerebral palsy, epilepsy, cognitive deficits, and sensory disorders. The incidence of HIE is higher in developing countries, and its long-term consequences pose a major burden on patients, families, and healthcare systems.

Introduction

Early diagnosis of HIE is crucial for timely intervention and better outcomes. Imaging modalities like ultrasound, CT, and MRI play a key role in detecting brain injuries. While transcranial ultrasound is the first-line investigation due to its accessibility, MRI is the gold standard for diagnosing HIE due to its superior sensitivity and specificity. MRI helps assess brain damage, monitor disease progression, and guide treatment strategies such as therapeutic hypothermia in neonates. This study evaluates the role of MRI in diagnosing HIE and its impact on clinical management.

Objective

This study aims to evaluate the role of the MRI in the evaluation of hypoxic ischemic encephalopathy in children under five years of age.

Methods

Following ethical clearance and informed consent, 51 participants were selected based on inclusion criteria. Brain MRI was performed after stabilizing respiratory and circulatory conditions, using Siemens Magnetom Spectra 3.0 Tesla MR equipment. Various MRI sequences, including T1, T2, FLAIR, DWI, and spectroscopy, were analyzed for abnormalities.

Results

The study included 51 children with a mean age of 18.6 months, predominantly male (72.5%). Abnormal muscle tone was observed in 58.8%, seizures in 51%, and altered consciousness in 43.1%. MRI findings revealed hypo-intense signals in 39.2% on T1 and 54.9% on T2 sequences. FLAIR hyper-intensity was seen in 47.1%, while DWI restriction was noted in 15.7%. Spectroscopy abnormalities (decreased NAA and increased lactate) were present in 19.6%, and gliosis in 17.7%. MRI grading classified 54.9% as Grade II, indicating moderate brain abnormalities.

Conclusion

In patients under the age of 5 years with history of perinatal asphyxia and presenting with symptoms male predominance was observed and 27.4 % cases had normal findings and in most of patients with findings changes were seen mostly in T2 sequence. Cystic changes were seen only in 17.6 % patients that also showed abnormal spectroscopy findings. No patient showed any changes of hemorrhages.

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ABBREVIATION	MEANING
HIE	Hypoxic Ischemic Encephalopathy
MR	Magnetic resonance
MRI	Magnetic Resonance Imaging
CT	Commuted tomography
PVL	Peri-ventricular Leukomalacia
RNA	**Ribose Nucleic Acid**
ATP	**Adenosine triphosphate**
HII	**Hypoxic Ischemic Insult**
ROS	**Reactive Oxygen Species**
Ca+2	**Calcium**
NMDA	**N-methyl-D-aspartate**
NO	**nitric oxide**
AMPK	**Adenosine monophosphate-activated protein kinase**
BBB	**Blood Brain Barrier**
MMPs	**Matrix Metalloproteinases**
USG	**Ultrasonography**
POCUS	**Point-of-Care Ultrasound**
PLIC	**Posterior Limb of Internal Capsule**
CBF	**Cerebral Blood Flow**
pCO2	**Partial pressure of carbon dioxide**
RI	**Resistivity Index**
SNR	**Signal-to-Noise Ratio**
T1WI	**T1 Weighted Imaging**
T2WI	**T2 Weighted Imaging**
DWI	**Diffusion Weighted Imaging**
FLAIR	**Fluid Attenuation Inversion recovery**

	Sequence
SWI	**Susceptibility Weighted Imaging**
MRS	**Magnetic Resonance Spectroscopy**
TR	**Repetition Time**
TE	**Echo Time**
NAA	**N-acetylaspartate**
CSF	**Cerebrospinal fluid**
EEG	**Electroencephalography**
NIRS	**Near-Infrared Spectroscopy**
SD	**Standard deviation**

INTRODUCTION

Hypoxic ischemic encephalopathy mainly affects preterm and term babies. It is brought on by insufficient oxygen and blood flow to brain, which causes localised or diffuse brain damage. The hypoxic ischemic event can occur before or during or after birth. HIE affects 26 / 1000 live births in developing & 1.5 / 1000 live births in developed countries. In affected newborns, 15 to 20% die in postnatal period and 20 to 25% will develop severe neurological impairment like cerebral palsy, epilepsy, visual or hearing impairment, cognitive impairment, intellectual disorders, behavioral disorders and social disorders.

The outcomes of HIE are often devastating and permanent, making it a burden for the patient, the family and society.

Imaging of brain with Ultrasound, Computed Tomography and Magnetic resonance imaging are important imaging techniques in the workup of patients suspecting HIE

Currently transcranial ultrasound is the first choice in cases suspected for HIE.

It is sensitive in detection of intracranial hemorrhage, cystic periventricular leucomalacia, hydrocephalous. Also, it is inexpensive, portable, inexpensive.

Radiation exposure is not seen with ultrasound. But it has marked interobserver variability, operator dependant and has low sensitivity in detecting cortical lesions.

Role of Computed Tomography:

As compared to MRI Brain, CT Brain is less sensitive & specific to diagnose HIE.

It can be used to screen very sick patients as it does not require sedation but main drawback is that it has a major radiation exposure.

Role of MRI:

MRI is more sensitive & specific in suspected cases of HIE. It is considered gold standard for diagnosing HIE. It doesn't have any radiation exposure. But neonate needs to be sedated before imaging.

Imaging of neonatal brain as compared to adult brain imaging, higher time for both T1 and T2 sequences is to be used to optimize signal to noise ratio and grey white matter differentiation.

Depending on the length of imaging and the clinical situation (e.g., haemorrhage, encephalomalacia, or gliosis), hypoxic injury to grey matter (especially the cerebral cortex and deep grey matter) exhibits typical T1 hyperintensity.

White matter injury causes cystic encephalomalacia or ischemia-induced oedema, which causes T1 hypointensity and T2 hyperintensity. On the other hand, astrogliosis is indicated by whitematter damage with aberrant

hyperintensity on T1 sequences and no discernible hypointensity in T2 sequences.

To show cystic leukomalacia and gliosis, Fluid attenuation inversion recover (FLAIR) sequence is quite helpful.

The conventional MRI sequences are less sensitive as compared to newer imaging modalities like DWI sequences or spectroscopy in diagnosis of acute injury.

Treatment and Management Based on Radiological Findings

The early detection of HIE through radiological imaging has significant implications for treatment.

In neonates, the primary treatment is therapeutic hypothermia, which showed to reduce extent of injury when initiated within six hours of birth.

Radiological imaging helps monitor the progression of the injury and guide the timing of interventions.

For adults, treatment strategies are based on stabilizing the patient, managing intracranial pressure and addressing the underlying cause of the ischemic event (e.g., cardiac arrest, trauma). MRI findings may influence decisions regarding the potential for recovery and long-term rehabilitation.

Need for study

20 -25 % of the diagnosed cases of HIE are having severe complications and early diagnosis of HIE can reduce the incidence of complications among HIE and currently MRI is considered gold diagnosis in diagnosis of HIE patients.

In this study, role of MRI Brain imaging in diagnosis of HIE is assessed.

Aim & Objective of the study

To study role of Magentic resonance imaging in the diagnosis of the Hypoxic Ischemic Encephalopathy in children under five years age.

REVIEW OF LITERATURE

A severe loss of oxygen to the brain during neonatal period causes hypoxic-ischemic encephalopathy (HIE), a disease that causes major physiological, biochemical, and molecular abnormalities. Lifelong morbidities such seizures, difficulty in breathing, altered awareness, reduced muscle tone, epilepsy, metabolic disturbance, cerebral palsy, intellectual incapacity, and behavioural issues as well as early death, may occur from it.[1]

Hypoxic ischaemic encephalopathy (HIE) is one of major causes of death in term and late preterm newborns, and survivors experience neurologic morbidity.[2]

Incidence:

In rich countries, HIE affects roughly 1.5 out of every 1,000 of live births, while in low- and middle income countries, it affects 10 - 20 out of every 1,000. [1]

Risk Factors and Prevention Strategies for Hypoxic-Ischemic Encephalopathy (HIE)

Multiple maternal, foetal and perinatal factors contribute to its development, and identifying these risk factors is essential for prevention and early intervention.

- Birth Weight and Gestational Age
  - ❖ Low birth weight (< 2.5 kg) significantly increases the risk of HIE.
  - ❖ Birth weight below 3.0 kg or above 4.0 kg is associated with moderate-to-severe HIE.[3]
  - ❖ Some studies indicate gestational age ≥41 weeks may be a risk factor, though findings remain inconsistent.[4]
- Contamination of amniotic Fluid
  - ❖ One of the main risk factors for newborn asphyxia and HIE is meconium-stained amniotic fluid.
  - ❖ The danger of moderate-to-severe contamination of meconium is considerably increased by moderate-to-heavy meconium contamination.[5]

- Apgar Scores
  - ❖ HIE risk is strongly indicated by low 1 minute Apgar scores (≤3) and 5 minute Apgar scores (≤7) [6]
  - ❖ Apgar score is widely used as a predictor of neonatal asphyxia and postnatal complications.
- Mode of Delivery
  - ❖ Some studies suggest cesarean section increases the risk of HIE, while others do not find an association.[7]
  - ❖ Instrumental deliveries (forceps, vacuum) and emergency C-sections have been linked to higher HIE risk.
- Maternal and Placental Factors
  - ❖ Gestational hypertension increases the likelihood of neonatal HIE.[8]
  - ❖ Advanced maternal age (≥35 years) may be a risk factor.[4]
  - ❖ Certain genetic polymorphisms (e.g., in AGT, TACR3, CARD8, NOS3, OLIG2 genes) and non-coding RNAs (microRNA-210, microRNA-30b, etc.) have been associated with HIE.

- Other Factors
  - ❖ Some studies suggest male fetuses are more prone to HIE, possibly due to hormonal differences.[9]
  - ❖ Nuchal cord and placenta abnormalities have been linked to HIE in some studies but not consistently.

Pathophysiology:

ATP produced from ketone bodies, lactate and glucose is required for foetal brain metabolism. The ability of foetal brains to conserve energy allows it to withstand hypoxia-ischemia (HI) better than the adult brain. However, prolonged or severe HI can lead to critical ATP depletion, making the fetal brain vulnerable to injury. HI conditions arise from complications such as maternal hypoxia, pre-eclampsia, umbilical cord issues, and placental abruption, leading to oxygen deprivation and triggering a cascade of damaging cellular events.[10]

Phases of HI Brain Injury

1. Primary Energy Failure – ATP depletion leads to excitotoxicity, oxidative stress, inflammation, and blood-brain barrier damage.
2. Latent Phase (6) hours post-HI – Temporary stabilization occurs, but mitochondrial dysfunction persists.
3. Secondary Energy Failure (6–15) hours post-HI – Seizures, excitotoxin release, renewed cytotoxic edema, and neuronal cell death occur.

Pathophysiological Events in HIE

1. Oxidative Stress: Because of the high concentration of fatty acids (unsaturated) and metals that catalyse reactions of free radical and the low quantities of antioxidants, the foetal brain is especially vulnerable to oxidative stress [11]. Because to decreased antioxidant activity, ROS build up and harm proteins, lipids, and DNA, resulting in mitochondrial malfunction and metabolic failure.

2. Intracellular Ca^{2+} Overload – While intracellular Ca2+ is typically relatively low (approximately 100 nM), extracellular Ca2+ concentrations can reach up to 1 to 2 Mm[12]. Excessive Calcium influx, mainly via NMDA receptors, activates NO synthase, further exacerbating oxidative stress, mitochondrial permeability changes, and apoptosis.

3. Mitochondrial Dysfunction – Mitochondria fail to produce ATP, causing ion imbalance, ROS overproduction, and activation of apoptotic pathways. A kind of vicious cycle is established when severe energy failure leads to cell membrane depolarisation and, in turn, Ca2+ infusion [13]. The activation of AMPK as an energy sensor attempts to restore energy balance but ultimately contributes to neurodegeneration.

4. Excitotoxicity – Cellular death is caused by activation of excitatory amino acid receptors (extracellular) is known as excitotoxicity [14]. Excessive glutamate release leads to persistent NMDA receptor activation, Ca^{2+} overload, and subsequent neuronal death.

5. Inflammation –

- Microglia Activation – The brain receives immuno-surveillance from microglia. The blood brain barrier (BBB) breaks down when HI occurs because microglia activate and develop macrophage-like functions include antigen presentation, phagocytosis, synthesis of inflammatory and anti-inflammatory cytokines and release of matrix metalloproteinases (MMPs) [15]. Acts as both protective and damaging; M(1) phenotype exacerbates injury, while M(2) phenotype aids recovery.

- Astrocytes – Release protective antioxidants but also pro-inflammatory cytokines, worsening brain injury. Cytokines worsen HI injury by directly causing neuronal cell death, preventing neurogenesis, and raising toxic NO levels.[16]

- Neutrophils – Contribute to brain injury through ROS production and vascular obstruction, though their response in neonates is weaker than in adults.

Clinical presentation:

Newborns with mild to moderate HIE symptoms include:

• Floppy, weak muscles (hypotonia) or stiff muscles (hypertonia)
• Having trouble eating
• Weak cry;
• Fatigue
• Irritability
• Pale, blue, or grey skin, fingers, and lips (Cyanosis)

Newborns with severe HIE symptoms include:

- Minimal or nonexistent reaction to sound or touch
- Inadequate reflexes
- Unusual breathing habits
- A sluggish or erratic heartbeat
- Convulsions
- Unconsciousness [17]

Imaging in hypoxic ischemic encephalopathy:

Neuroimaging techniques, such as Magnetic Resonance Imaging brain and cranial ultrasonography (USG) are crucial for determining the degree of the injury, confirming the diagnosis, directing clinical judgements, and forecasting the possibility of unfavourable outcomes.[18]

Ultrasound Brain:

A useful screening method for the identification and treatment of asphyxiated infants is cranial ultrasonography (USG). The degree, timing, pattern, and progression of an injury can all be determined by serial USG. Increased neurological morbidity and mortality are linked to near-total suffocation with widespread involvement of the grey and white matter. Prior to the start of therapeutic hypothermia, resistant index values on a cerebral Doppler scan can be a significant indicator of the prognosis in the long run. [18]

The value of point of care ultrasonography (POCUS) in the treatment of HIE is becoming more widely acknowledged. The device can be utilised for a sequential assessment of the encephalopathy's evolution and is generally accessible and reasonably priced [19]

Brain injury due to asphyxia evolves over different phases, with distinct patterns of damage observed in acute, subacute, and chronic stages.

Changes seen in acute & subacute phases

1. Hyperechogenecity of white matter: The periventricular and subcortical areas may exhibit diffuse or patchy echogenicity, contingent on the extent of the lesion.

2. In severe cases, sub-cortical white matter hyperechogenicity appears like ‘tramlines’ due to the hypoechoic signals of the cortex between them. These changes are best observed in sagittal views. White matter injury occurs in both partial and sustained hypoxia [19][20].

3. **Loss of differentiation between grey and white matter:** In milder situations, grey-white matter differentiation is enhanced due to increased echogenicity in the white matter compared to the cortical grey matter. However, in severe cases, parenchymal edema causes the loss of anatomical landmarks such as the sulci, interhemispheric fissure, and Sylvian fissures, making it difficult to differentiate between grey and white matter on ultrasound[19].

4. **Mass effect** on adjacent brain structures is due to parenchymal edema: the edema in brain parenchyma is commonly observed within (24–48) hours after the hypoxic insult. It results in compressive changes such as slit-like ventricles, effacement of cerebral sulci, and narrowing of intrahemispheric fissures and basal cisterns. However, care must be taken when interpreting small ventricular size, as normal neonates may have small ventricles in the first (36) hours of life[19][21].

5. **Hyperechogenicity of Deep Grey Matter** – In severe cases, the grey matter structures, including the thalamus and putamen, show focal or diffuse hyperechogenicity. This appears as the 'four-column sign' in coronal views, where the echogenic thalamus and basal ganglia on both sides form four parallel pillars. This highlights the hypoechoic crescent-shaped posterior limb of the internal capsule (PLIC sign). This pattern suggests a severe hypoxic-ischemic event, often due to sentinel events such as placental abruption or umbilical cord prolapse[19][22].

6. **Involvement of cerebellum–** A hyperechoic cerebellum, which is best seen in axial views, represents a severe form of injury. This pattern is under-recognized in neonates and requires further research to better

understand its implications. Cerebellar ultrasound remains a specialized field requiring more systematic evaluation[19][23].

7. **Haemorrhages-** In term neonates, intraventricular hemorrhages usually originate in choroid plexus, whereas in preterm patients, hemorrhages commonly arise from the germinal matrix or parenchyma[19].

Hypoxic-Ischemic Injury patterns in brain:

Four different patterns of hypoxic-ischemic injury were identified by Myers, which depending on cause, severity, timing and site of insult. These patterns reflect the complex interactions between vascular supply and brain development in different clinical situations[19].

Changes seen in chronic phase

- The impacted brain areas exhibit gradual atrophy over the weeks after the hypoxic event, resulting in a loss of volume in white and grey matter.
- Ex-vacuo ventriculomegaly and subarachnoid space widening are ultrasound results that show a reduction in the volume of brain tissue.
- As brain damage worsens, the impacted areas may exhibit cortical necrosis and cystic alterations. Cysts typically develop ten or more days following the first hypoxic episode.

Normal Findings on Cranial USG

• Brain damage is not always ruled out by a normal cranial ultrasonography. To find small abnormalities that traditional ultrasound could overlook, advanced imaging methods like diffusion-weighted MRI or contrast-enhanced ultrasound may be required.

Duration of Hypoxic Alterations on Cranial USG:

- Because hypoxic-ischemic encephalopathy (HIE) is a dynamic injury of brain, its entire extent may not be captured by imaging at a single point in time.
- In early phase, edema may be mild, and cranial USG may appear unremarkable for the first (24–48) hours after the injury.
- The PLIC sign and thalamic and basal ganglial hyperechogenicity usually appear 48–72 hours after the event.
- The loss of differentiation of grey white matter and emergence of slit-like ventricles may take 48 to 72 hours to show up on cranial ultrasound in cases of severe injury.
- If the hypoxic insult happened prior to delivery, the oedema may have gone away by birth, making it challenging to identify sudden changes.
- Cranial USG Pattern in Preterm Hypoxic Insult
  Because of variations in cerebral vascular maturity, preterm newborns and term neonates have different anatomical distributions and patterns of ischaemic damage.
  Because preterm newborns have watershed zones and immature

premyelinating oligodendrocytes, the periventricular white matter is especially susceptible to ischaemia.[24].

- The most prevalent cause of periventricular leukomalacia (PVL) is preterm newborns weighing less than 1500 g at ≤33 weeks of gestation.[25].
- The periventricular white matter has strong bilateral diffuse echogenicity on cranial USG, with intensity nearly matching that of the choroid plexus.[26].
- Cystic alterations may appear in previously hyperechoic regions within three to four weeks following the first injury, accompanied by atrophic alterations in the surrounding brain parenchyma.
- In contrast to term neonates, preterm children born before gestational age of 32 weeks frequently exhibit diffuse echogenicity in thalami and basal ganglia (>90%), which makes interpretation difficult.[26].

Cerebral Doppler Alterations in Hypoxic Insults:

- Hypoxic insults cause alterations in cerebral blood flow (CBF), which could indicate an existing brain injury or act as a defensive mechanism.[27].
- Cerebral haemodynamics is influenced by a number of variables, such as changes in intracranial pressure, prostaglandins, nitric oxide, cardiac output and partial pressure of Carbon dioxide (pCO2).[28].

- The brain first suffers decreased CBF after hypoxia, which is indicated by an increase in the cerebral vessels' resistive index (RI). A period of hyperaemia that lasts for hours to days could follow if treatment is not received.[28],[29].
- • RI < (0.55) during 72 hours of delivery is highly linked to unfavourable outcomes including death or severe impairment.[19],[30].
- However, during therapeutic hypothermia, the predictive value of low RI decreases [19], likely due to vasoconstriction or altered metabolism. After rewarming, the predictive value returns [31].
- RI > (1) may be indicative of brain death [32].

HIE Scores Based on Cranial USG:

- In situations with limited resources, when MRI may not be accessible, composite cranial USG scores are useful instruments for forecasting outcomes in newborns with HIE.
- A number of rating schemes have been put forth, such as a (6)-pattern injury score based on profound grey and white matter involvement and a (8)-grade categorisation. [18], [33].
- A composite score of injury of deep grey matter and white matter is part of a new grading system developed by Annink et al. A score of ≥(3) is regarded as a cutoff point for suggesting additional neuroprotective measures or care redirection. ([18]).

- Although these scoring systems show potential, inter-rater variability remains moderate, and structured training may improve consistency in interpretation.

CT Brain in HIE:

Computed tomography (CT) is crucial for diagnosis and immediate treatment of hypoxic-ischemic brain injuries, which are catastrophic and cause high death and morbidity rates in both adults and children. However, minor CT alterations can resemble other disorders such dural sinus thrombosis and subarachnoid haemorrhage. Understanding the distinctive characteristics is essential for timely patient care.

The reversal sign is characterised by either reversal of the grey or white matter densities and significantly higher density of thalami, brainstem, and cerebellum or a diffuse decreased density of cerebral grey and white matter with a decreased or absent gray - white matter Interface.

However CT brain is not routinely advised for evaluation of hypoxic ischemic changes.

MRI Brain in HIE:

The most sensitive & specific imaging modality is magnetic resonance imaging (MRI), even though CT, MRI, and ultrasound (USG) make up the imaging arsenal. USG continues to be the preferred initial investigation due to its affordability, mobility, and accessibility. Haemorrhage, hydrocephalus, and the cystic form of periventricular leukomalacia (PVL)

can all be detected with USG's sensitivity. For higher-quality imaging, a special neonatal head coil is advised. The signal-to-noise ratio (SNR) rises with decreasing coil diameter, enhancing visual quality.

Preparation:

- **Scheduling & Coordination:** MRI scans are carefully scheduled to align with infant feeding times, ensuring they are calm and likely to sleep during the procedure. For outpatient scans, caregivers should arrive at least an hour before to allow adequate preparation time[34].
- **Caregiver Support & Orientation:** Upon arrival, caregivers are provided with a detailed overview of the MRI procedure, including expected timelines, safety protocols, and what to anticipate during the scan.
- They can address any concerns about sedation, motion artifacts, or the impact of noise on their infant[35].
- **Infant Comfort & Preparation:** To minimize discomfort and movement, the infant is swaddled in a snug blanket or hospital linen, with additional layers provided if needed. The MiniMuffs Noise Attenuators (Natus, USA) protect against scanner noise, reducing the risk of auditory stress[36]
- **Immobilization for Stability:** The baby is kept firmly in place by a Medvac Vacuum Immobilisation Bag (CFI Medical Solutions, USA). The bag is carefully adjusted, ensuring a firm yet gentle hold without applying excessive pressure. Straps across the forehead and body help maintain stability while allowing normal breathing and circulation [37].

- **MRI Safety Screening:** Both the infant and any accompanying caregiver must undergo rigorous MR safety screening. A detailed MRI safety form is completed to rule out metal implants, pacemakers, or devices that may interfere with the magnetic field. The MRI technician carefully verifies this information before allowing entry into the scanner room [38].

- **Pre-Scan Feeding Strategy:** A full feeding just before scanning helps keep the infant calm and asleep. If the infant is still hungry or unsettled, caregivers may provide additional feeding before the procedure starts. The goal is to optimize sleep duration during the scan[39].

Positioning & Scanning Process

- **Infant Placement in the Scanner:** For the best imaging quality, the baby is placed gently on the MRI table in supine position with their head in the special neonatal head coil. For extra stability, try towels, foam cushioning, or baby-sized headphones.[40].

- **Monitoring During the Scan:** To ensure infant safety, an MRI compatible pulse oximeter is attached to hand or foot to continuously track oxygen saturation (O_2) and heart rate. The MRI team listens for any signs of distress, crying, or movement via a microphone inside the scanner[41].

- **Controlling Infant Motion:** Some babies may still wake up or move in spite of immobilisation and swaddling treatments. To help calm the

infant and let scanning to resume in such circumstances, carers may provide an MRI safe dummy, sucrose drops or more feeding.[42].

- **Effective Scan Timing:** By restricting the procedure to the necessary MR sequences, the scan time is reduced. When time and baby comfort allow, important scenes may be redone if motion artefacts degrade image quality. Higher-quality photos can be obtained by scheduling additional scanning time when feasible.[43].

- **Caregiver Presence in Scanner Room:** Depending on institutional policies, caregivers may stay in the scanner room during the procedure. If permitted, they must follow strict MR safety guidelines, wear ear protection, and avoid any metallic objects that could interfere with the scan [44].

Completion of MRI & Post-Scan Care

- Post-Scan Infant Handling: Once imaging is complete, the vacuum bag is deflated, and the swaddling layers and noise attenuators are removed. The infant is checked for comfort, re-dressed in metal-free clothing, and offered another feeding if needed [45].

- Reviewing MRI Findings: Depending on hospital and research protocols, caregivers are informed about when and how they will receive results. Immediate clinical findings may be shared by the radiologist or neonatal specialist, while research-related scans may require additional review before results are communicated[46].

MRI Safety Considerations

- Radiation-Free Imaging: There is no ionising radiation in MRI like CT scans or X-rays. It uses magnetic fields and radiofrequency pulses to create fine-grained images of the brain which makes it a safe, non-invasive method for newborns.[47].
- Strict Metal Safety Protocols: Any metallic objects, implants, or foreign bodies pose a serious risk inside the MRI scanner due to the strong magnetic attraction. As a result, all equipment, clothing, and accessories must be MR-compatible before entering the scanner room [47].
- Noise Protection Measures: MRI machines generate loud noises that could impact neonatal hearing. Infants are provided MiniMuffs ear covers and padded headphones for protection. Caregivers also receive earplugs or adult headphones when present in the scanner room [48].
- Certified MRI Personnel: All medical and research staff involved in neonatal MRI scans must complete MR safety training and certification. They must adhere to strict institutional safety guidelines to ensure a secure environment for infants and caregivers [49].

Standard sequences of MRI used for adults must be modified for neonates because of the lower protein / lipid levels and increased water content of the newborn brain.

Increasing repetition duration (TR) of both T1-weighted images (T1WI) and T2-weighted images (T2WI) accomplishes this optimisation. For T1WI (400 ms), standard TR is raised to 800 ms and for T2WI (4000 ms) it is raised to

6500 ms. These modifications improve gray-white matter distinction and SNR.[50].

Neonatal MRI images are usually taken in 1.5 or 3.0 Tesla MRI machine. The protocol includes following sequences:

- Sagittal and coronal T2WI, Axial T1WI, FLAIR and T2WI
- The DWI sequence and ADC mapping
- T2 WI using gradient echo (SWI)
- Spectroscopy and FLAIR sequences

Key Features of MRI Sequences

- Axial T1WI is very helpful in identifying subacute haemorrhage, ischaemia, and myelination. Assessing myelination is essential for assessing neonatal brain development.
- Axial T2WI is useful for identifying anomalies in white matter signals because it offers superior gray-to-white matter contrast.
- In particular, gradient-echo T2 WI (SWI) is helpful in detecting haemorrhages. This sequence can distinguish between hemorrhagic and non-hemorrhagic astrogliosis, as well as between haemorrhage and ischaemic lesions.
- For detecting cytotoxic oedema, the most sensitive imaging method is DWI with ADC maps (24 hours–7 days). It is much better at detecting early ischaemic alterations than traditional T1 or T2WI.

- The ischaemic regions have limited diffusion, showing up as diminished signals on ADC maps and strong signals on DWI maps. Even if tissue damage continues, DWI anomalies typically peak three to five days after injury and then progressively return to normal. This normalisation, which necessitates association with other sequences, might be deceptive.

- Because of prenatal injury or delayed cell death, 15% of thalamic lesions and basal ganglia may exhibit normal DWI.[51].

- Abnormal T2 hyperintensity, which correlates to the growth of glial tissue and cystic lesions, can be detected by FLAIR imaging. However, because to their high water content, neonates are less responsive to it. After eight months, when myelination is more developed, its usefulness improves.[52].

MR Spectroscopy (MRS) for Hypoxic-Ischemic Injury (HII)

MRS is conducted at short TE (echo time) of 35 ms and intermediate TE of 135–144 ms. When assessing HII in full-term newborns, the preferred region of focus is the basal ganglia. Compared to other imaging methods, MRS is thought to be more sensitive over the first 24 hours.

- One important indicator of HII is the existence of a lactate peak. Lactate is distinguished from the nearby lipid peak at (0.9–1.3) ppm by appearing as a doublet peak at 1.3 ppm on short TE spectra and inverting below the baseline in intermediate TE spectra.

Preterm newborns typically have lower amounts of N-acetylaspartate (NAA) and higher levels of lactate. As the brain develops, these levels return to normal, hence gestational age affects how MRS is interpreted.

- Poorer neurological outcomes in full-term newborns with asphyxia are indicated by higher lactate-to-creatinine and lactate-to-choline ratios as well as lower absolute concentrations of NAA and choline in the basal ganglia. [53].
- Cerebrospinal fluid (CSF) normally contains lactate, so choosing the right voxel is essential to preventing CSF contamination.
- To increase diagnostic precision, MRS data should always be examined in combination with traditional MRI and DWI results.

MRI Findings in Hypoxic-Ischemic Injury (HII)

- T1 hyperintensity and varied T2 signal intensity are caused by HII's effects on grey matter, specifically the deep grey matter and cortex. Acute and chronic damage can be distinguished using the T2 signal pattern. Astrogliosis is suggested by white matter damage, which manifests as aberrant T1 hyperintensity without appreciable T2 hypointensity.
- Low T1 signal intensity, on the other hand, denotes oedema or cavitation.During the first week of life, DWI is still the most helpful imaging method; however, starting in the second week, T1 and T2WI offer the most diagnostic value.

Similar studies

In a study done by **Gp Capt A Alam et al.** 45 children with a history of preterm delivery and perinatal hypoxia, ages 4 weeks to 8 years, were investigated in 2010 at the Command Hospital in Banglore. These kids suffered from neurological impairments that ranged from severe mental retardation to cortical blindness, spastic diplegia, and spastic quadriplegia. A 1.5 T Siemens Magnetom Avanto MR equipment was used for the procedure. According to the study, periventricular white matter ischaemic infarction regions could be reliably detected by MR imaging in both its early and late stages. As a result, MRI could precisely show the parts of the brain damaged by this ischaemic process.[54]

Mirjam steiner, et al examined all perinatal asphyxia admissions made between June 2008 and February 2019 at the Medical University of Vienna, Austria's Department of Neonatology, Paediatric Intensive Care, and Neuropediatrics. This cohort research only included participants with prospectively available neurophysiology and/or neuroimaging data. In asphyxiated neonates, they examined the prediction capacity of neurophysiology and neuroimaging methods. According to the study, a combination of MRI and EEG parameter scores was more predictive than either one alone.When aEEG/MRI and NIRS data were combined, no additional improvement in outcome prediction was seen.[55]

Yi Li, et al examined infants receiving treatment at nine hospitals in San Francisco, California, who had mild HIE. Two reviewers used an established classification approach to assess the newborn brain MRIs, and disagreements were settled by consensus. The subgroup of MRIs that were done at or before 8 days of age was used to score the severity and timing of MRI brain injury (i.e., acute, subacute, or chronic). At median age 5, 87 of the 142 infants with mild HIE showed MRI damage. More frequent were punctate white matter injuries (18%), deep grey injuries (20%), and watershed injuries (23%). Mild damage was more prevalent (44%) than moderate (11%) or severe (4%), among the 125 (88%) newborns who had a bone femur MRI at or before 8 days. The prevalence of subacute (37%) vs acute (32%) or chronic (1%), lesions was higher.[56]

Anna Tuiskula, et al examined 50 newborns following neonatal asphyxia. The majority of these newborns, whether or not they had HIE, had successive abnormal neurological symptoms. Children with perinatal asphyxia without HIE have a neurological finding profile similar to that of children with HIE. White matter T2 hyperintensity was a common MRI finding in prenatal asphyxia without HIE and was linked to abnormal spontaneous movements in the HINE.[57]

Timing of MRI in Neonates

An MRI should be performed five to fourteen days following delivery. Because certain abnormalities may not yet be apparent, imaging done before five days may understate the severity of brain damage. Nonetheless, in ventilated children, early neonatal MRI is essential for clinical decision-making.

MATERIALS AND METHODS

Source of Data:

Children under five who have experienced prenatal asphyxia in the past, preterm birth, multiple complications who are advised for MR brain imaging in Dr. Prabhakar Kore hospital and medical research center, Belagavi

Study Design:

Cross sectional study

Study Period:

A one year-based hospital study from June 2023 to June 2024.

Sample Size:

Formula used for sample size calculation is,

$$n = \frac{Z^2 - \frac{\alpha}{2}\, p\, q}{(15\%\ of\ P)^2} \text{ x } 1.10$$

where Z is the number that corresponds to the necessary degree of confidence (Z value = 1.96), n is the necessary sample size, and p is the percentage occurrence of a state or condition (proportion or prevalence).

Neurological complications were observed in 82% cases using MRI. The sample size is determined by taking into account similar results at a 95% confidence level and a 10% maximum error.

$$n = \frac{1.96^2 \text{x } 82\ (100 - 82)}{(0.15 \text{ x } 82)^2} \text{ x } 1.10$$

$$n = 41.2 \approx 42$$

Therefore, 42 is the bare minimum sample size needed. The precision of the result rises in tandem with the sample size.

Sampling Technique:

Universal sampling method

Inclusion Criteria:

1. Children under the age of five with a history of perinatal asphyxia

2. Children under the age of five with a history of preterm birth

3. Children under the age of five who were advised for MRI brain due to multiple complications.

Exclusion Criteria:

1. Children who are not in a stable respiratory and circulatory condition.

Study protocol:

Following ethical clearance, patients were chosen in accordance with the inclusion criteria listed above. After informing the patient's parents or guardians about the protocol in a language they can comprehend, written informed consent will be obtained. In-person interviews were used to gather a thorough history of the complaints that were presented.

As soon as the children's respiratory and circulatory conditions were stabilised, brain MRI was conducted. The type of anaesthesia was determined by consulting anaesthetist and the referring physician. A head coil was used to put the patients head first in a supine position, and Siemens Magnetom Spectra 3.0 Tesla MR equipment was used for imaging. Oxygen saturation and heart rate was continuously tracked.

A 210 x 448 matrix, an 18–23 cm field of view, and a 4 mm section thickness were used to acquire the images.

MRI sequences obtained were:

1. T1 Sequence
2. T2 Sequence
3. Diffusion Weighted Image
4. FLAIR sequence
5. Susceptibility Weighted Image
6. Magnetic resonance spectroscopy

Data collection procedure: The data was analysed and evaluated for the following: periventricular white matter loss or paucity; ventricular enlargement with irregularity of wall; corpus callosum morphology and signal characteristics; and any associated abnormalities. The data was also evaluated for the presence of areas of altered signal intensities within the periventricular white matter relative to the remainder of the cerebral hemisphere white matter.

STATISTICAL ANALYSIS

Microsoft Excel was used to enter the data, and SPSS software, version 17.0, was used to do statistical analysis. Age was transformed into age groups. Frequencies and percentages were used to display qualitative characteristics such as age groups, gender, muscular tone, respiratory function, consciousness, T1, T2, Flair, DWI sequences, spectroscopic findings, presence

of gliosis, and MRI grading. Data was represented graphically using pie charts and bar diagrams.

RESULTS

We have included 51 children below the age of 5 years for the purpose of the study. The mean (SD) age was 18.6 (18.4) months with the minimum of 3 days and maximum of 5 years. The median (IQR) was 12 (4-36) months

Table-1: Age distribution

Age groups	**Number**	**Percentage**
<1 month	10	19.6
1-5 months	3	5.9
12 months -1 year	18	35.3
1-5 years	20	39.2
Total	51	100.0

The study included 51 participants categorized into different age groups. The majority of participants, 20 (39.2%), were aged between 1 to 5 years, followed by 18 (35.3%) who were between 12 months to 1 year. Additionally, 10 (19.6%) participants were less than 1 month old, while 3 (5.9%) were between 1 to 5 months old.

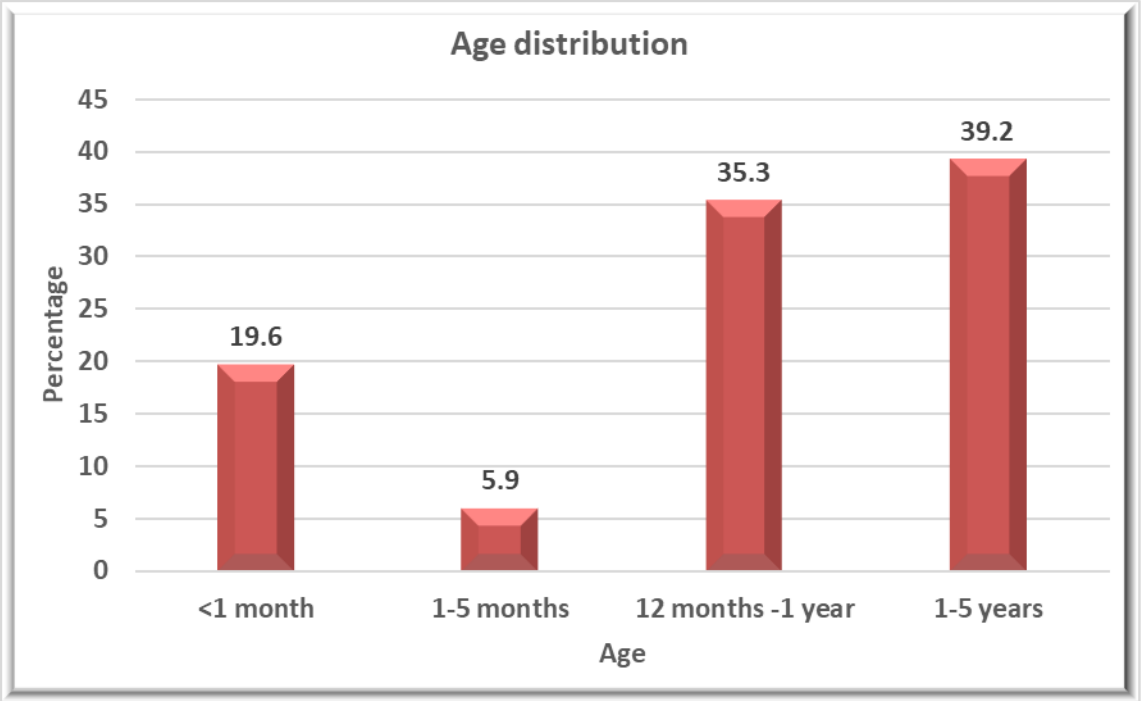


Table-2: Gender distribution

Gender	Number	Percentage
Male	37	72.5
Female	14	27.5
Total	51	100.0

Of all the participants, 37 (72.5%) were male and 14 (27.5%) were female. This indicates a higher proportion of male participants compared to female participants in the study

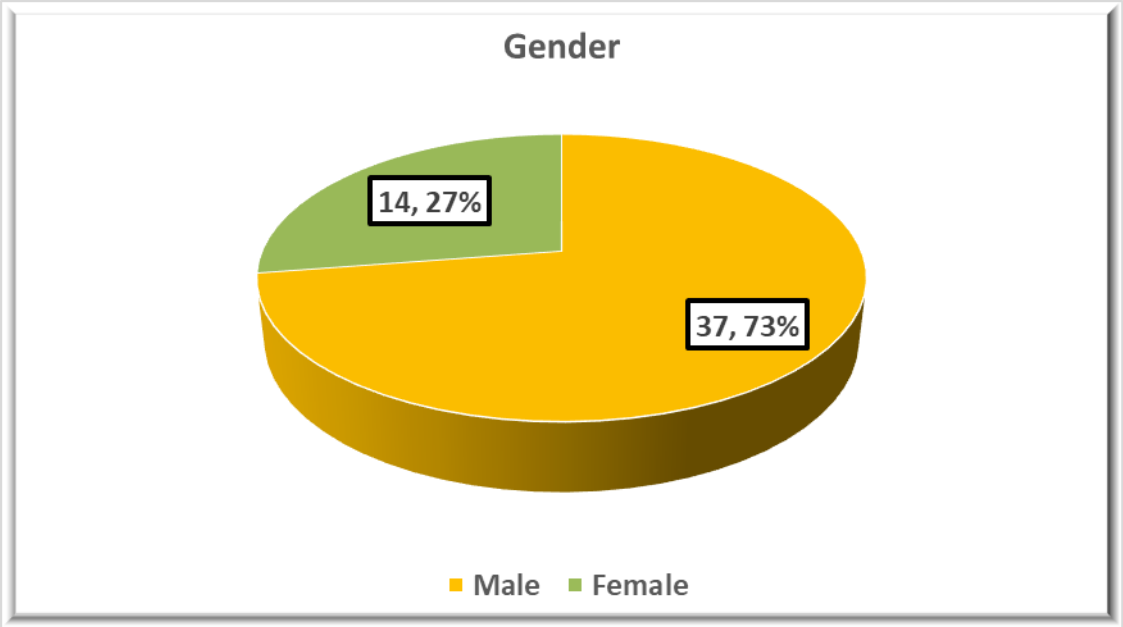


Table-3: Description of clinical symptoms - muscle tone. Seizures, respiratory pattern and consciousness

Parameters		Number	Percentage
Muscle tone	Normal	21	41.8
	Abnormal	30	58.8
Seizure	Present	26	51.0
	Absent	25	49.0
Respiratory pattern	Normal	28	54.9
	Abnormal	23	45.1
Conscious	Normal	29	56.9
	Abnormal	22	43.1

Muscle tone was abnormal in 30 (58.8%) participants, while 21 (41.8%) had normal muscle tone. Seizures were present in 26 (51.0%) participants, whereas 25 (49.0%) did not experience seizures. Respiratory patterns were normal in 28 (54.9%) participants, while 23 (45.1%) exhibited abnormalities. Consciousness levels were normal in 29 (56.9%) participants, whereas 22 (43.1%) had altered consciousness.

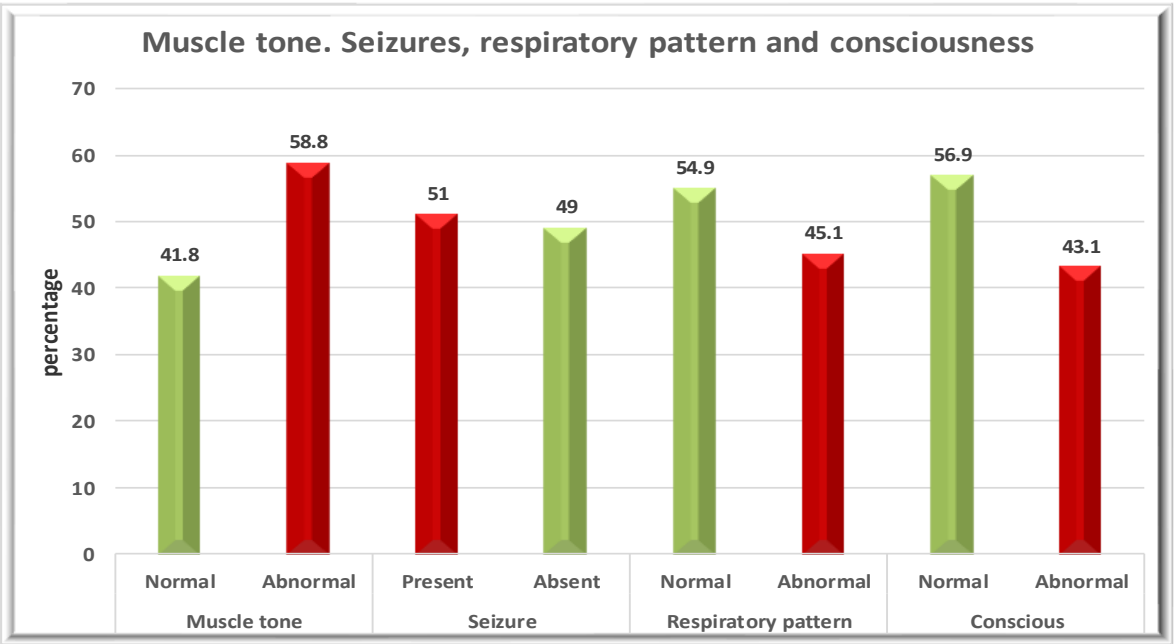


Table-4: T1 sequence

T1 sequence	Number	Percentage
Normal	26	51.8
Hypo-intense	20	39.2
Hyper-intense	5	9.0
Total	51	100.0

The T1 sequence findings revealed that 26 (51.8%) had normal results, while 20 (39.2%) exhibited hypo-intense signals. Additionally, 5 (9.0%) participants showed hyper-intense signals. These findings indicate that while over half of the participants had normal T1 sequence results, a considerable proportion exhibited hypo-intense signals, suggesting potential pathological changes.

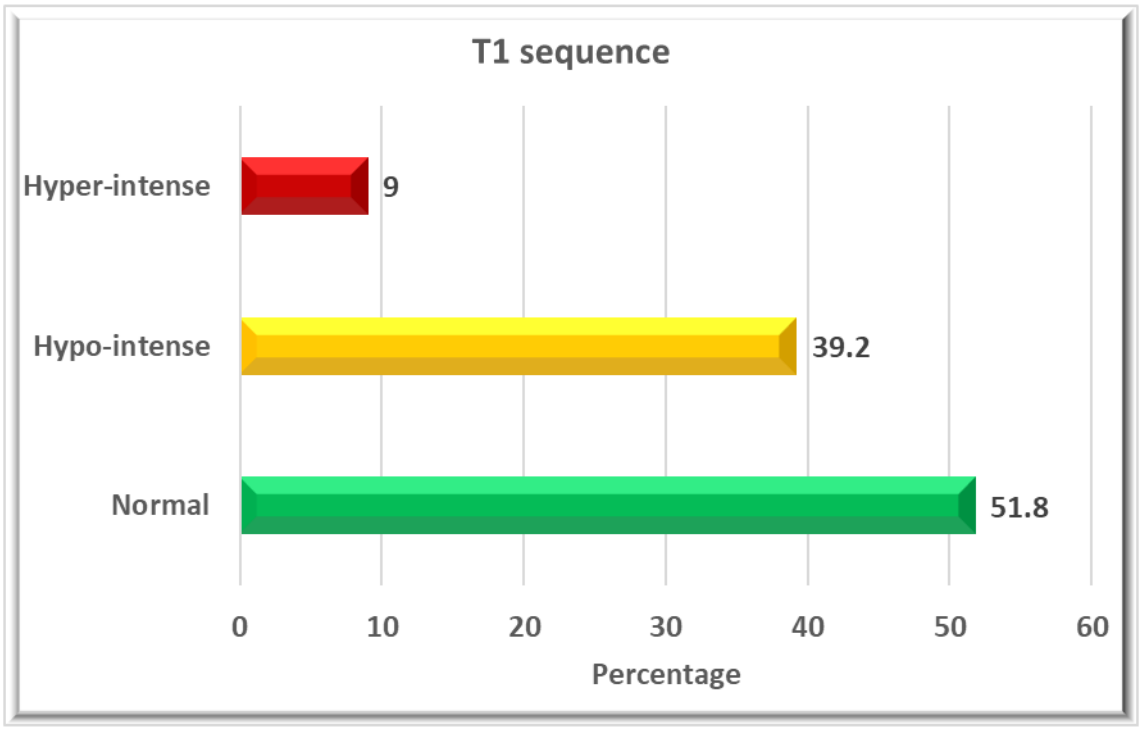


Table-5: **T2 sequence**

T2 sequence	**Number**	**Percentage**
Normal	23	45.1
Hypo-intense	28	54.9
Total	51	100.0

The T2 sequence findings showed that 23 (45.1%) had normal results, while 28 (54.9%) exhibited hypo-intense signals. These findings indicate that more than half of the participants had hypo-intense signals on the T2 sequence, suggesting potential underlying pathological changes.

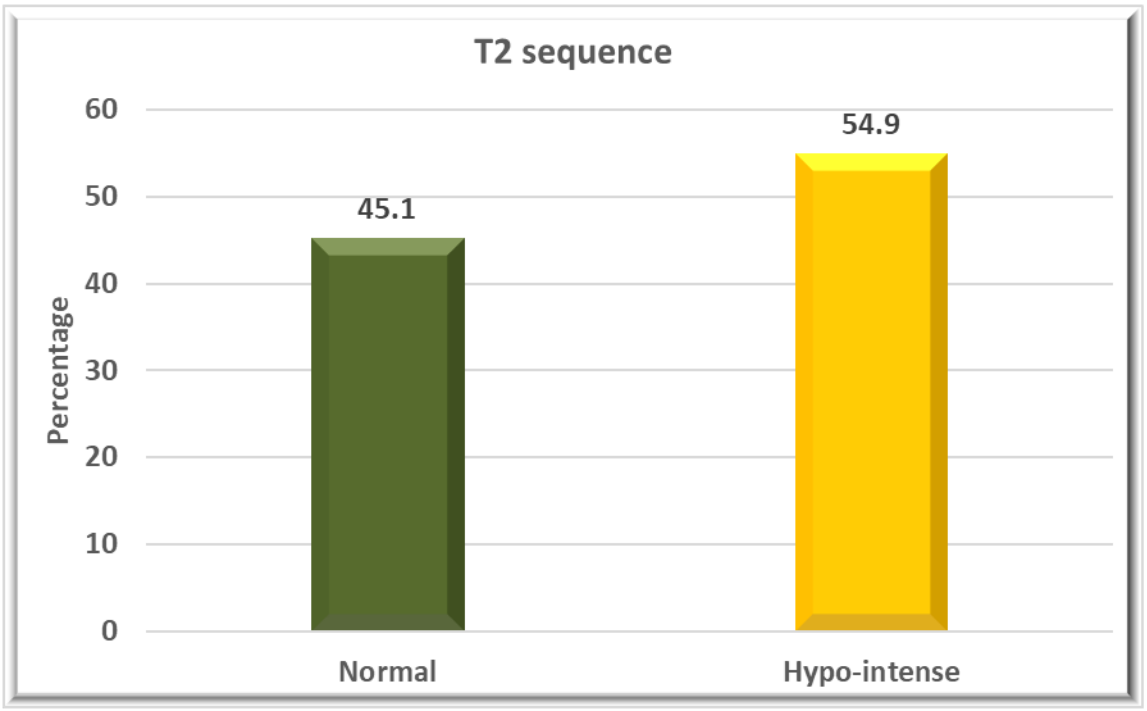


Table-6: Flair sequence

Flair sequence	Number	Percentage
Normal	23	45.1
Hypo-intense	4	7.8
Hyper-intense	24	7.1
Total	51	100.0

The FLAIR sequence findings showed that 23 (45.1%) had normal results, while 4 (7.8%) exhibited hypo-intense signals and 24 (47.1%) had hyper-intense signals. These findings indicate that nearly half of the participants

had hyper-intense signals, suggesting potential abnormalities, while a smaller proportion exhibited hypo-intense changes.

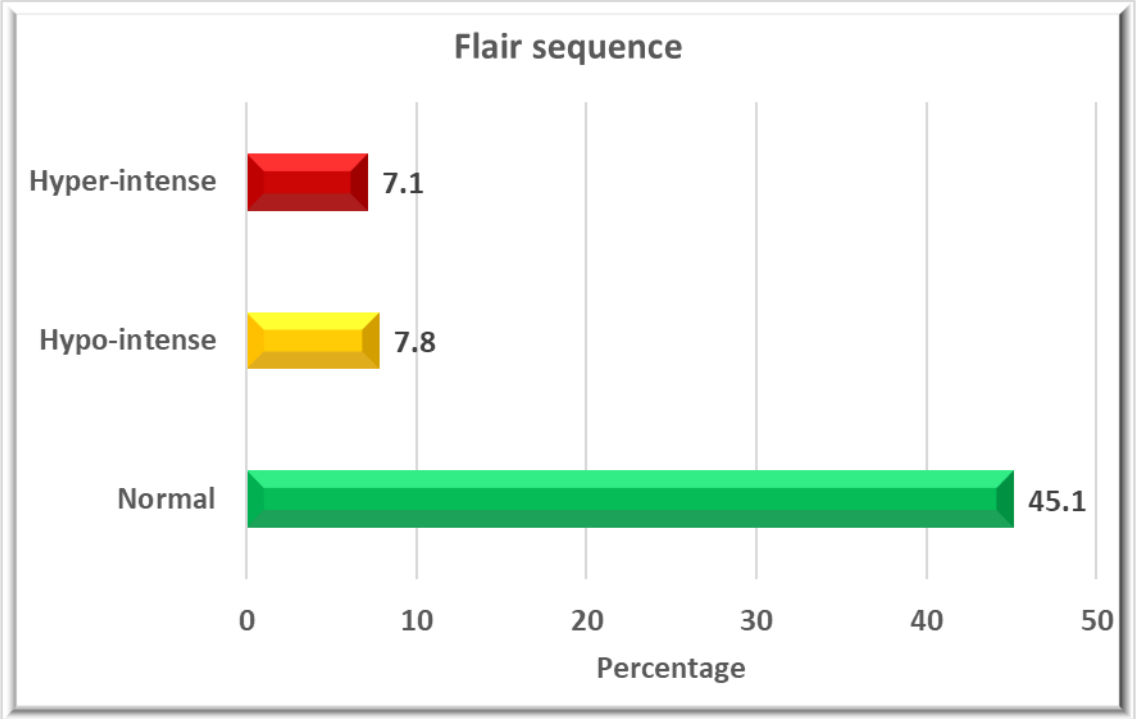


Table-7: DWI sequence

DWI sequence	Number	Percentage
Normal	43	84.3
Restriction	25	15.7
Total	51	100.0

The DWI sequence findings revealed that 43 (84.3%) had normal results, while 8 (15.7%) exhibited diffusion restriction. These findings indicate that the majority of participants had normal DWI sequences, with a small proportion showing restricted diffusion.

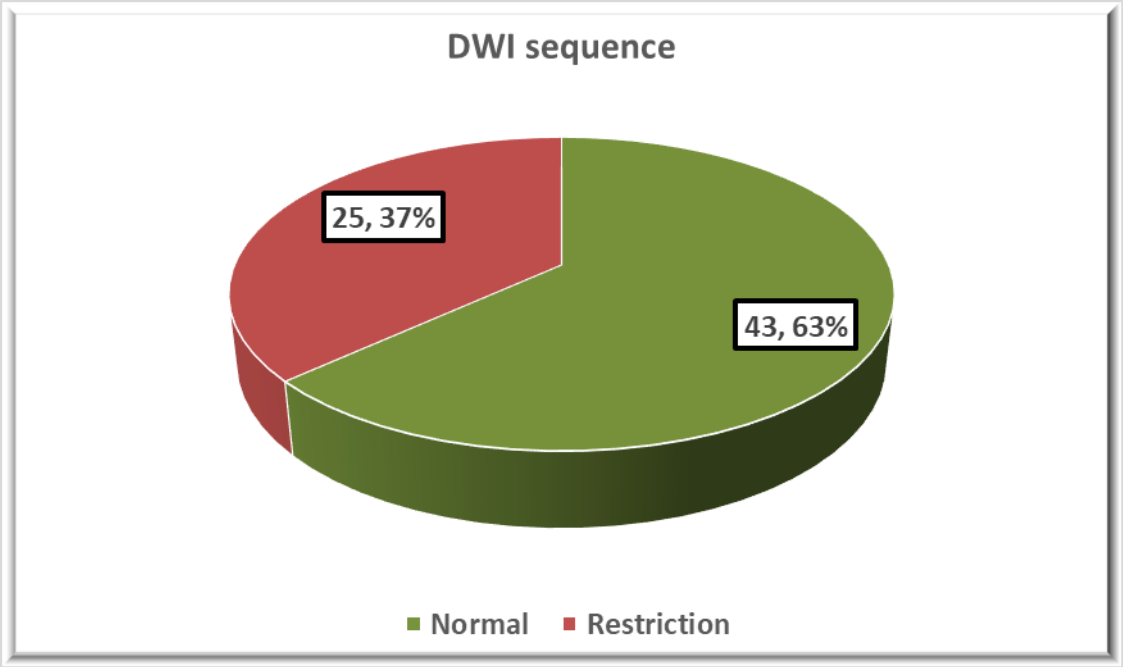


Table-8: Spectroscopy findings

Spectroscopy	Number	Percentage
Normal	41	80.4
Decreased NAA and increased Lactate	10	19.6
Total	51	100.0

The spectroscopy showed that 41 (80.4%) had normal results, while 10 (19.6%) exhibited decreased N-acetylaspartate (NAA) and increased lactate levels. These findings indicate that the majority of participants had normal metabolic profiles, whereas nearly one-fifth showed metabolic abnormalities.

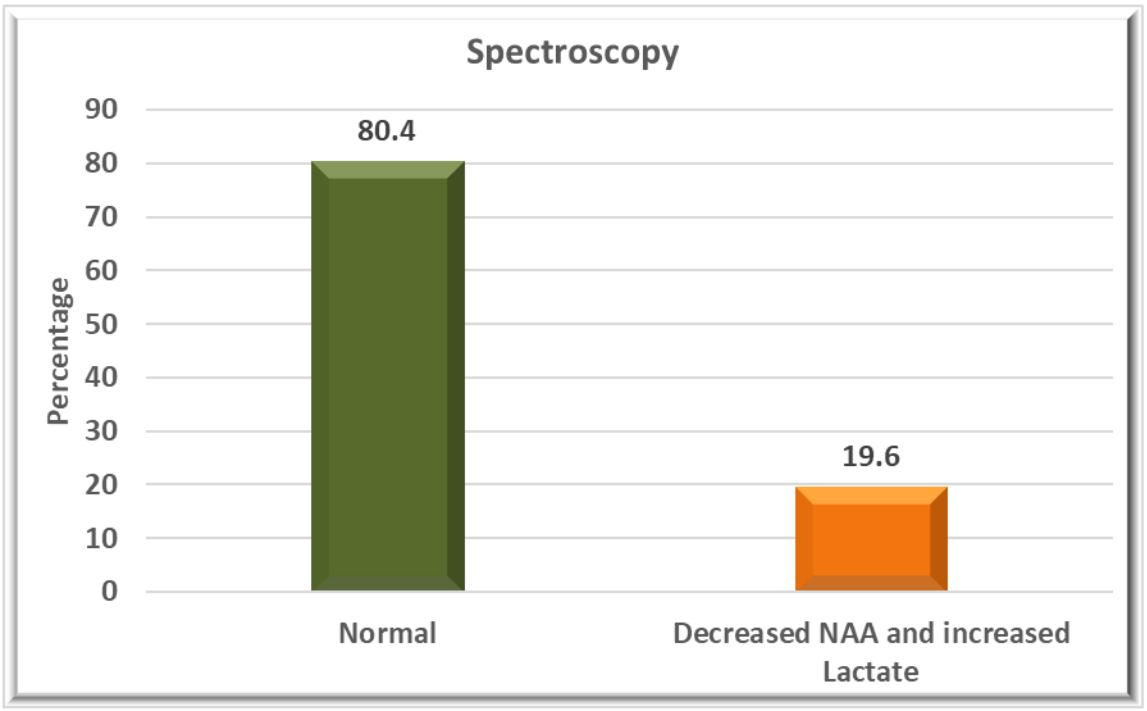


Table-9: Gliosis

Gliosis	Number	Percentage
Absent	42	82.4
Present	9	17.7
Total	51	100.0

Of all the children, 42 (82.4%) had no signs of gliosis, while 9 (17.7%) showed its presence. These results indicate that the majority of participants did not exhibit gliotic changes, whereas a small proportion demonstrated gliosis.

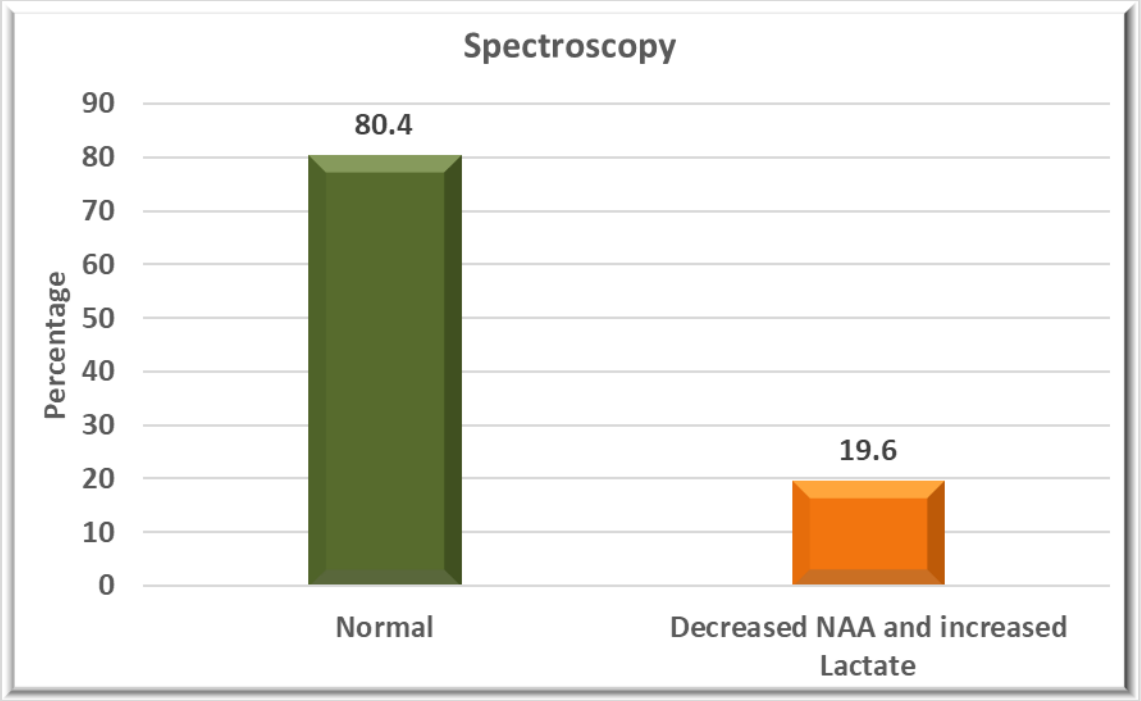
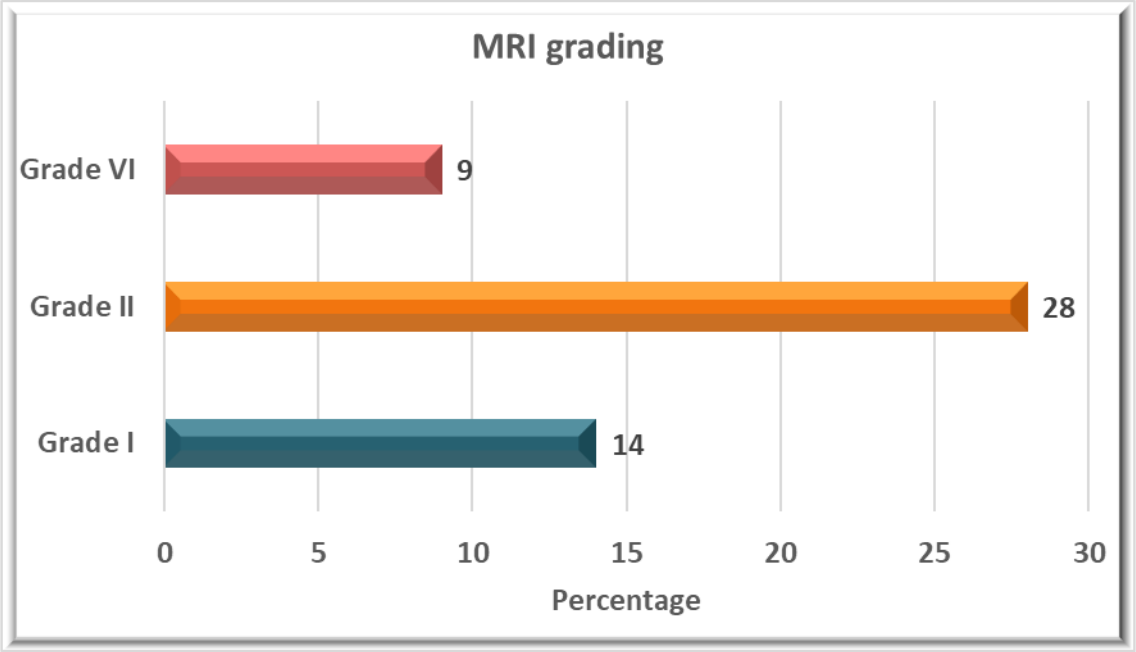


Table-10: MRI Grading

MRI Grading	Number	Percentage
Grade I	14	27.4
Grade II	28	54.9
Grade VI	9	17.7
Total	51	100.0

MRI showed that 14 (27.4%) were classified as Grade I, 28 (54.9%) as Grade II, and 9 (17.7%) as Grade VI. These results indicate that more than half of the participants had Grade II changes, suggesting moderate abnormalities,

while a smaller proportion had either mild (Grade I) or severe (Grade VI) findings



DISCUSSION

Out of 51 children below the age of 5 years for the purpose of the study. The mean (SD) age was 18.6 months (with the minimum age of 3 days and maximum age of 5) unlike in study by **Lokesh Rana et al.** [58] where median age was 7 months. The median (IQR) was 12 (4-36) months and 37 (72.5%) were male and 14 (27.5%) were female.

This study indicates a higher proportion of male participants compared to female participants in the study similar to the study done by Hayes er al. [9]

This study demonstrated that muscle tone was most common clinical feature in majority of the participants (30 (58.8%) participants), followed by seizures which were present in 26 (51.0%) participants, and 23 (45.1%) patients shows respiratory abnormalities and 22 (43.1%) patients had altered consciousness.

As compared to study done by Alam et al. [54], where only 8.9 % of patients had normal findings, this study had 27.4 % cases with normal findings. But as compared to study done by Kivi et al. [57] normal findings were seen in 27 % of cases similar to that of our study and study done by Elshal et al. [59] showed normal MRI in 53.3 % cases.

In MRI findings, abnormalities in signal intensity in T1 sequence were seen in 25 patients, where 20 (39.2%) patients exhibited hypo-intense signals and 5 (9.0%) participants showed hyper-intense signals.

In The T2 sequence, hyperintense signal intensities were seen in 28 (54.9%) patients, more than half of the participants had hyperintense signals on the T2 sequence, suggesting potential underlying pathological changes.

The FLAIR sequence, 23 (45.1%) had normal results, while 4 (7.8%) exhibited hypo-intense signals and 24 (47.1%) had hyper-intense signals.

As compared to study done Rana et al. [58] where no significant changes were observed in both normal and aberrant grey matter ADC values, this study had 8 (15.7%) exhibited diffusion restriction, which could indicate acute ischemic injury.

The spectroscopy showed that 41 (80.4%) had normal results, while 10 (19.6%) with cystic changes exhibited reduced N-acetylaspartate (NAA) and raised lactate levels in the abnormal cystic areas.

These findings indicate that the majority of participants had normal metabolic profiles. Study by Kivi et al. [57]demonstrated presence of lactate in 12 (24%) of patients similar to that of this study where lactate peak was demonstrated in 19.6 % of patients.

Similar to the study done by Alam et al.[54], where 13.3 % of patients demonstrated cystic changes this study also demonstrated 9 (17.7%) patients had cystic changes.

Majority of patients, 42 (82.4%) had no signs of gliosis. These results indicate that majority of participants did not exhibit gliotic changes, whereas a small proportion demonstrated gliosis.

MRI showed that 14 (27.4%) were classified as Grade I, 28 (54.9%) as Grade II, and 9 (17.7%) as Grade VI. These results indicate that more than half of the participants had Grade II changes, suggesting moderate abnormalities, while a smaller proportion had either mild (Grade I) or severe (Grade VI) findings

No patients shows any signs of hemorrhage.

SUMMARY

Based on the available data, hypoxic ischemic encephalopathy in smaller age group is a concerning medical emergencies that present with wide range of medical symptoms and diplomatic outcomes are seen. Also, etiological spectrum is variable depending on studied population by various research scholars. The changes in MRI will help us to analyse associated abnormalities

In this prospective observational study for one year, we included 51 cases presented with multiple symptoms having a history of perinatal asphyxia and analysed the variantions that were seen in MRI brain study.

Out of 51 children below 5 years of age were taken for the purpose of the study. The mean age was 18.6 months and this study indicates a higher proportion of male participants compared to female participants.

This study also demonstrated that abnormal muscle tone was most common clinical feature in majority of the participants followed by seizures, respiratory abnormalities and altered consciousness.

In 8.9 % of patients no abnormalities were seen in MRI study and in patienst with abnormal MRI findings abnormal hyperintense signal intensities were seen in more than half of the participants in T2 sequences. The FLAIR sequence of MRI showed abnormal signals in 54.9 % cases.

Normal spectroscopy findings were seen in 41 (80.4%) while 10 (19.6%) with cystic changes exhibited decreased N-acetylaspartate (NAA) and increased lactate levels in the abnormal cystic areas.

Majority of patients, 42 (82.4%) had no signs of gliosis.

MRI showed that 14 (27.4%) were classified as Grade I, 28 (54.9%) as Grade II, and 9 (17.7%) as Grade VI. The results indicate that more than half of the participants had Grade II changes, suggesting moderate abnormalities, while a smaller proportion had either mild (Grade I) or severe (Grade VI) findings. No patients shows any signs of hemorrhage.

LIMITATIONS OF THE STUDY

1. **Challenges in Interpreting Neonatal MRI**

- Interpreting MRI results in neonates is inherently complex due to the ongoing developmental stage of their brains. Distinguishing between normal developmental changes and abnormal ischemic damage can be particularly challenging.
- Misinterpretation of neonatal brain scans may lead to incorrect diagnoses or inappropriate treatment strategies. This complexity underscores the need for highly specialized expertise, which may not always be readily available in all clinical settings.

2. **Patient Movement and Need for Anaesthesia**

- Neonates, especially those with hypoxic-ischemic encephalopathy (HIE), often require sedation or anaesthesia to remain still during MRI scans. However, this introduces potential risks, such as respiratory complications or adverse reactions to sedatives.
- The necessity for anaesthesia adds complexity to the imaging process. If anaesthesia is either unsafe or insufficient, it can result in lower-quality images or, in some cases, prevent the scan from being performed altogether.

3. **Challenges in Predicting Long-Term Outcomes**

- While MRI can provide valuable insights into the extent of brain injury, it is not always a reliable predictor of long-term developmental and neurological outcomes.
- MRI findings alone may not provide sufficient information to accurately predict a neonate's future development or recovery potential. This limitation can complicate family counselling and the formulation of prognostic decisions.

4. **Additional Challenges:**

- Cost of the Study: MRI scans can be expensive, which may limit accessibility for some patients.
- Portability Limitations: MRI equipment is not easily portable, restricting its use to specialized facilities.
- Lack of Control Group: The absence of a control group in the study limits the ability to draw definitive conclusions.

- Uncertainty of Injury Timing: In most cases, the exact timing of the brain injury was unknown, which complicates the interpretation of MRI findings and their correlation with clinical outcomes.

STRENGTH OF OUR STUDY

Of all the observed cases with changes in MRI, has been analysed for the commonest findings which would be one of the reliable pieces of evidence for the further clinical evaluations

CONCLUSION

In patients under the age of 5 years with history of perinatal asphyxia and presenting with symptoms male predominance was observed and 27.4 % cases had normal findings and in most of patients with findings changes were seen mostly in T2 sequence. Cystic changes were seen only in 17.6 % patients that also showed abnormal spectroscopy findings. No patient showed any changes of hemorrhages.

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ANNEXURE I

KAHERs JNMCBELAGAVI

INFORMED CONSENT FORM

"MAGNETIC RESONANCE IMAGING IN EVALUATION OF HYPOXIC ISCHEMIC ENCEPHALOPATHY IN CHILDREN UNDER FIVE YEARS OF AGE: A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"

Name of Student/Principal Investigator: BS0122015
RADIOLOGY RESIDENT
DEPT OF RADIO-DIAGNOSIS
JN MEDICAL COLLEGE
BELAGAVI

Introduction:

Hypoxic ischemic encephalopathy is a condition that mainly affects term and preterm babies. It is caused by inadequate blood flow and oxygen supply to brain resulting in focal or diffuse brain injury. The hypoxic- ischemic event can occur before, during or after birth. HIE affects 26 per 1000 live births in developing countries, 1.5 per 1000 live births in developed countries. Of the affected newborns, 15 to 20% will die in the postnatal period, and an additional 20 to 25% will develop severe neurological impairment, including cerebral palsy, epilepsy, visual and hearing impairment, cognitive impairment, intellectual, behavioral and social disorders.

Role of MRI:

MRI is most sensitive & specific for evaluating suspected cases of HIE. It is considered gold standard for diagnosing HIE. It doesn't have any radiation exposure. But neonate needs to be sedated before imaging.

In neonatal brain imaging as compared to adult brain imaging, a relatively higher repetition time for both T1 and T2 is used to optimize the signal to noise ratio and grey white matter differentiation.

Hypoxic injury to grey matter (Cortex and deep gray matter) demonstrates characteristic T1-hyperintensity depending on duration of imaging and pathological condition such as hemorrhage, encephalomalacia, or gliosis.

White matter injury results in T1 hypointensity and T2 hyperintensity due to ischemia induced edema or cystic encephalomalacia. Where as, white matter injury with abnormal T1 hyperintensity and without marked T2 hypointensity denotes astrogliosis.

The fluid attenuation inversion recover (FLAIR) sequence is particularly useful for demonstrating cystic leukomalacia and gliosis

However conventional MRI is less sensitive than newer Imaging modalities like DWI or MR spectroscopy in diagnosis of acute injury.

PARTICIPATION IN THE STUDY

I request you to kindly participate in the study titled "**MAGNETIC RESONANCE IMAGING IN EVALUATION OF HYPOXIC ISCHEMIC ENCEPHALOPATHY IN CHILDREN UNDER FIVE YEARS OF AGE:A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY**" at Dr. Prabhakar kore hospital and medical research center, Belagavi is being conducted by

BS0122015, Post graduate in radio diagnosis at J.N. Medical college, Belagavi, Karnataka.

We request you to participate in this study as your child is eligible to be included. During the study, you will be asked questions regarding your child's past and present medical history and you will be required to answer to the best of your knowledge. You will also be medically examined as per the protocol drawn.

Purpose of Study:

Participation of your child in this study will help us to analyze the role of MRI in evaluation of Hypoxic ischemic encephalopathy in children.

Withdrawal from participation in the study:

Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: you will not get any benefits by

participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating

in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only

processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: **"BS0122015"** If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**MAGNETIC RESONANCE IMAGING IN EVALUATION OF HYPOXIAC ISCHEMIC ENCEPHALOPATHY IN CHILDREN UNDER FIVE YEARS OF AGE:A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY**".

The content of the provided information sheet, has been carefully read by me in my understandable language and have understood its content. I confirm that I have had the opportunity to ask questions and have received satisfactory answers.

The nature and purpose of study and its potential benefits and expected duration of study, and relevant details of study have been explained to me in details. I understand that the participation of my child in the study is entirely voluntary and that I am free to withdrew my consent at any time without the need to provide an explanation, knowing that it will not affect my medical care or legal rights.

Name of the parent/guardian:
Signature or left thumb impression
of parent/guardian:
Name of the witness:
Signature or left thumb
impression of the witness:Name
of the investigator:
Contact Number:
Signature of the investigator:

BS0122015

ANNEXURE 2

PROFORMA:

Name of Child:-

Age/Sex:-

IP/OP NO:-

Name of Parent/Guardian:-

Age/Sex:-

Symptoms	Present/Absent	If present then duration	Episodes/Day (if applicable)
Extreme lethargy Comatose Extreme alertness Lack of reflexes Abnormal movements High/low muscle tone Seizures Breathing problems Loss of consciousness			

Headache Blurring of vision Hearing impairment Cognitive impairment Intellectual, behavioral and Social disorders Any other neurological abnormality			

Baseline Vitals

1. Blood pressure

2. Temperature

3. Heart Rate

4. Oxygen saturation

MRI Findings:-

Diagnosis:-

ANNEXURE III

IMAGES

Fig 1- 3.0 Tesla Siemens MRI Machine (Magnetom Spectra)

CASE I

2- year-old girl child presented with history of poor muscle tone and global developmental delay since birth

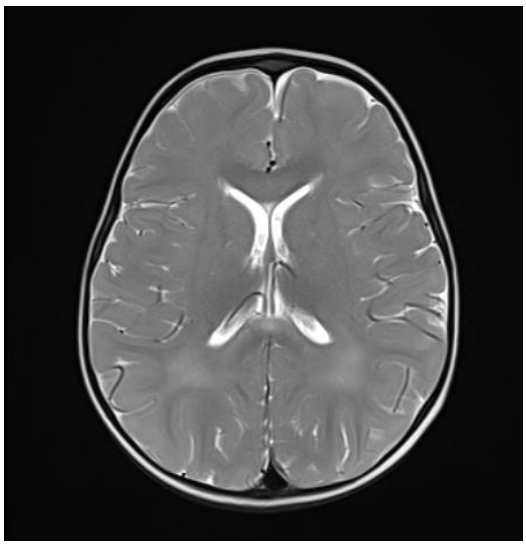

Fig. 2

T2 Axial WI shows bilateral symmetric T2 hyperintensities in periventricular regions

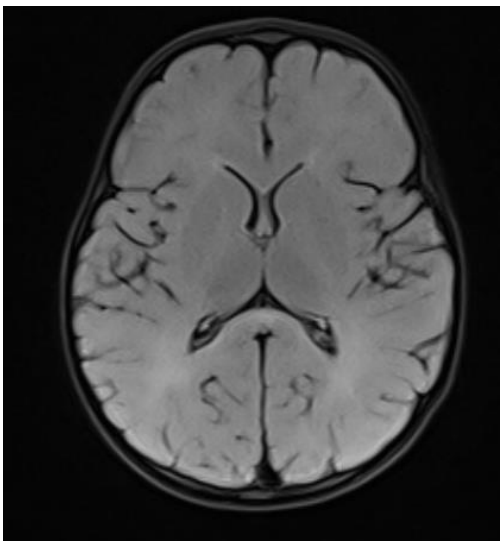

Fig. 3

FLAIR Axial WI shows bilateral symmetric T2 hyperintensities in periventricular regions

CASE II

10-month-old girl child presented with history of poor muscle tone, seizure (3 episodes) and developmental delay since childhood. History of similar episodes in first born child

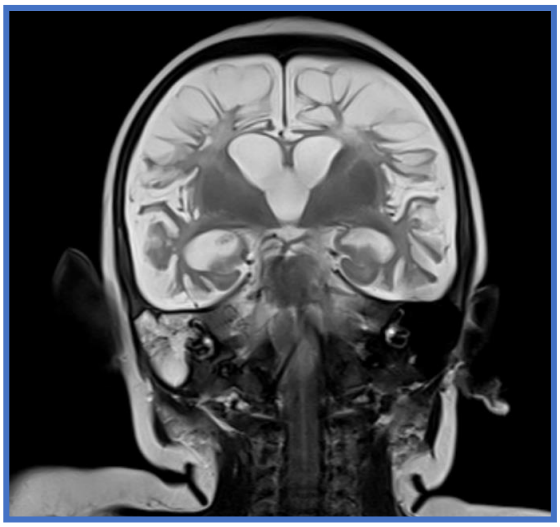

Fig 4

T2 Coronal image shows Gliosis in bilateral fronto-parieto-temporal regions

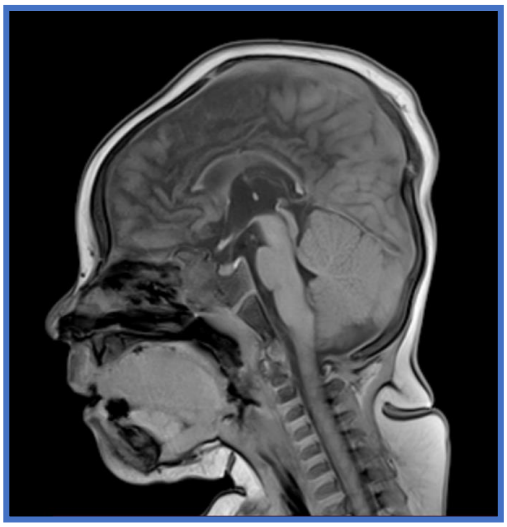

Fig 5

T1 Sagittal imaging shows thinning of corpus callosum with atrophy

CASE III

2 – year -old boy presented with history of poor muscle tone and seizures since birth with ICU admission.

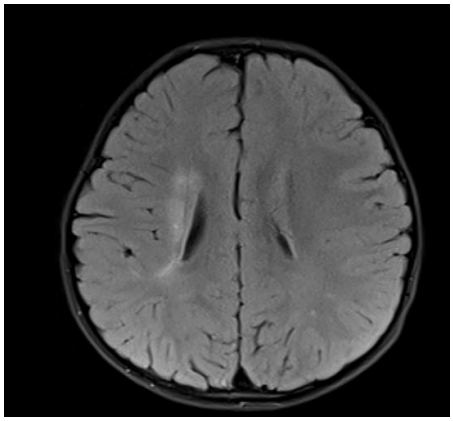

Fig 6

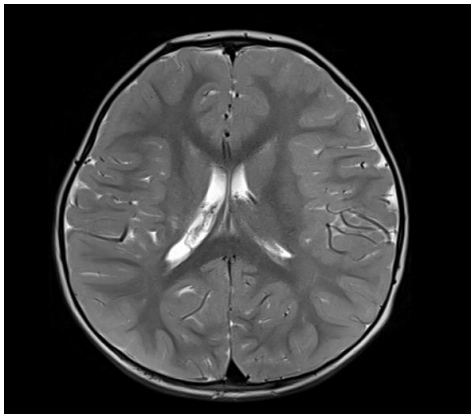

Fig 7

FLAIR and T2 Axial images shows hyperintensities in right periventricular regions

CASE IV

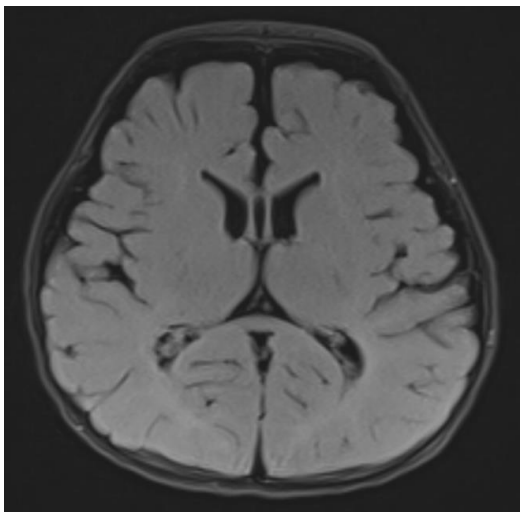

Fig 8

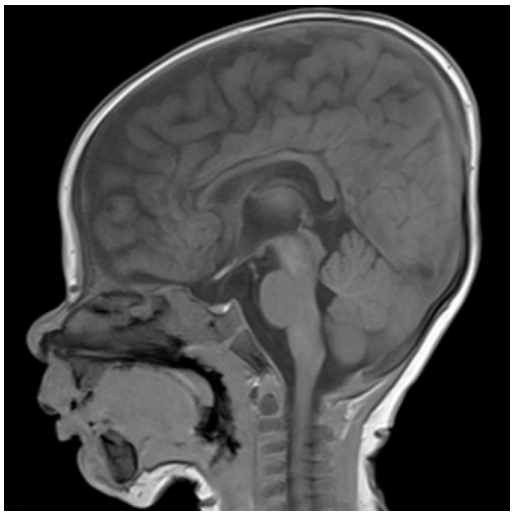

Fig 9

FLAIR Axial WI and T1 sagittal images reveal normal Brain MR anatomy with no significant pathology detected

ANNEXURE IV

KEY TO MASTERCHART

P	Present
	Absent

PMT – Poor mucle tone

S- Seizures

ARP- Abnormal Respiratory Pattern

AC- Abnormal Consciousness

DWI S – DWI Sequence

SWI S – SWI Sequence

28	12 MONTHS	F	P			P	Bilateral peritrigonal regions		Hyperintense	Hyperintense				Grade II
29	1YR	M	P		P	P	Bilateral fronto-parieto-temporo-occipital regions				Restriction			Grade II
30	15 DAYS	M		P			Bilaetral thalami, putamena and internal capsule	Hyperintense			Restriction			Grade II
31	10 MON	F	P	P	P	P		Normal	Normal	normal				Grade I
32	2 YRS	M	P				Bilateral periventricular and peritrigonal regions	Hypointense	Hyperintense	Hyperintense				Grade II
33	3 DAYS	M			P	P	Bilateral fronto-parieto-temporo-occipital regions				Restriction			Grade II
34	3 DAYS	M		P	P			Normal	Normal	Normal				Grade I
35	5 YRS	F	P				Left fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense				Grade II
36	11 MONTHS	M			P	P		Normal	Normal	Normal				Grade I
37	4 YRS	M	P			P	Bilateral frontal and peritrigonal regions	Hypointense	Hyperintense	Hyperintense				Grade II
38	6 MONTHS	M		P	P		Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense		Decreased NAA and in P		Grade VI
39	6 DAYS	F	P		P	P	Bilateral lateral thalami, putamen, corticospinal tracts	Hyperintense			Restriction			Grade II
40	7 MONTHS	M		P				Normal	Normal	Normal				Grade I
41	2 YRS	F	P				Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense				Grade II
42	2 YRS	M		P	P	P	Right periventricular region	Hypointense	Hyperintense	Hyperintense				Grade II
43	7 MON	M		P				Normal	Normal	Normal				Grade I
44	7 MONTHS	M	P				Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense		Decreased NAA and in P		Grade VI
45	3 YRS	M		P		P		Normal	Normal	Normal				Grade I
46	5 DAYS	F	P	P	P		Bilateral periventricular regions	Hypointense	Hyperintense	Hyperintense				Grade II
47	12 MONTHS	M	P			P	Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense		Decreased NAA and in P		Grade VI
48	10 MONTHS	M		P	P			Normal	Normal	Normal				Grade I
49	2 YEARS	F	P				Bilateral periventricular regions	Hypointense	Hyperintense	Hyperintense				Grade II
50	7 MONTHS	F		P		P	Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense		Decreased NAA and in P		Grade VI
51	3 MONTHS	M	P		P			normal	Normal	Normal				Grade I
52	5 DAYS	M	P	P			Bilateral fronto-parieto-temporo-occipital regions				Restriction			Grade II

1		SEX	PMT	S	ARP	AC	AREA AFFECTED	T1	T2	FLAIR	DWI S	SWI S	SPECTROSCOPY	GLISOSIS	MRI GRADING
2	16DAYS	M	P	P	P	P	Right periventricular area	Hyperintense		Hyperintense					Grade II
3	16 MONTHS	F	P	P	P	P	Right frontoparietooccipito-temporal regions	Hypointense	Hyperintense	Hypointense			Decreased NAA and in P		Grade VI
4	3 YRS	M	P	P			Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hypointense				P	Grade VI
5	1 MONTH	M	P			P	Corpus Callosum, bilateral cerebral peduncles & ventral brain stem		Hyperintense		Restriction				Grade II
6	3 YRS	M	P		P		Left lentiform nucleus, left insular cortex and left coror Hypointense		Hyperintense	Hypointense			Decreased NAA and in P		Grade VI
7	12 MONTHS	M	P	P			Bilateral caudate nucleus and basal ganglia	Hyperintense		Hyperintense					Grade II
8	5 DAYS	M			P	P	Right parasagittal frontal, right fronto-parieto-temporal regions, right c		Hyperintense	Hyperintense	Restriction				Grade II
9	2 YRS	F	P	P			Bilateral periventricular and peritrigonal regions	Hypointense	Hyperintense	Hyperintense					Grade II
10	5 YRS	M					Bilateral corona radiata		Hyperintense	Hyperintense					Grade II
11	1 YR	M		P	P	P		Normal	Normal	Normal			Decreased NAA and increased La		Grade I
12	5 MONTHS	M	P			P	Bilateral Thalamus and putamen	Hypointense	Hyperintense						Grade II
13	2 YRS	M		P	P			Normal	Normal	Normal					Grade I
14	3 YRS	M	P				Bilateral centrum semiovale, periventricuar, peritrigonal regions		Hyperintense	Hyperintense					Grade II
15	1 YR	M		P	P		Bilateral periventricular and peritrigonal regions		Hyperintense	Hyperintense					Grade II
16	5 MONTHS	F	P			P		Normal	Normal	Normal					Grade I
17	3 YRS	M		P	P		Left frontal periventricular region		Hyperintense	Hyperintense			Decreased NAA and increased La		Grade II
18	1 MONTH	F	P	P			Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense				P	Grade VI
19	11 MONTHS	M		P	P		Bilateral periventricular and peritrigonal regions		Hyperintense	Hyperintense					Grade II
20	4 YRS	M	P	P				Normal	Normal	Normal					Grade I
21	4 MONTHS	M			P		Bilateral Thalami	Hyperintense					Decreased NAA and increased La		Grade II
22	8 MONTHS	M	P	P	P			Normal	Normal	Normal					Grade I
23	9 MONTHS	F		P			Bilateral parieto-occipital regions	Hypointense	Hyperintense	Hypointense			Decreased NAA and in P		Grade VI
24	4 YRS	F	P			P	Bilateral periventricular and peritrigonal regions	Hypointense	Hyperintense	Hyperintense					Grade II
25	9 DAYS	M	P	P	P		Bilateral perventricular regions				Restriction				Grade II
26	3 YRS	M	P			P		Normal	Normal	Normal					Grade I
27	8 DAYS	M		P			Bilateral fronto-parieto-temporo-occipital regions	Hypointense	Hyperintense	Hyperintense					Grade II
28	12 MONTHS	F	P			P	Bilateral peritrigonal regions		Hyperintense	Hyperintense					Grade II