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MRI EVALUATION OF PERINATAL HYPOXIC ISCHEMIC INJURY

Radiology

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ABSTRACT

Introduction: Hypoxic-ischemic injury (HII) is one of the most devastating of all neonatal brain insults. The acute clinical manifestation of HII in the newborn is termed hypoxic ischemic encephalopathy. It occurs in 1-6/1000 live full-term births and carries a high risk for subsequent neurodevelopmental disabilities.

Aims: To the study the role of MRI in evaluating spectrum of Hypoxic Ischemic Injury.

Material and methods: MRI brain (Plain) was done in 60 patients with clinical history suspicious of perinatal insult. MRI is the investigation of choice for detection of neonatal HII. It has higher sensitivity and specificity detection rate as compared to CT or ultrasound. Sedation is often needed to avoid motion artifacts.

Results: MRI is useful in determining different spectrum of findings of perinatal hypoxic ischemic injury.

KEYWORDS

MRI, hypoxic ischemic injury, HII, hypoxic ischemic encephalopathy, HIE

INTRODUCTION

Birth asphyxia is an important cause of static development & neurological handicap in both term and preterm infants. In simple words it is the most important cause of morbidity and mortality in newborns in developing countries like India. The incidence of perinatal asphyxia is 1-1.5 % in most centers. Estimates of the incidence of perinatal asphyxia vary from 1 to 8 per 1,000 live births. Neonatal encephalopathy (NE) occurs in 1-6/1000 live full-term births and carries a high risk for subsequent neurodevelopmental disabilities. This is usually related inversely to gestational age and birth weight.[1]

Moderate to severe forms of HII results in long term sequelae such as cerebral palsy. However in milder forms, it can and does cause cognitive impairments in children and young adults without having any motor difficulties (and therefore have not been diagnosed with cerebral palsy). These cognitive impairments are subtle and do not appear until children are school aged, manifesting as difficulties with reading, spelling, and mathematics. They also could be at risk of developing behavioral problems.[2]

Although ultrasonography (USG), computed tomography (CT) and magnetic resonance imaging (MRI) comprise the imaging modalities, MRI is the most sensitive and specific modality. The role of MRI is in excluding structural anomalies and mainly in assessing the extent and nature of injury. Thereby, it helps in prognosticating the outcome and planning neurodevelopmental therapy.[3]

AIMS AND OBJECTIVES

- To emphasize on the role of MRI in detection and characterization of Perinatal Hypoxic Ischemic Injury.
- To provide pictoral review of the different neuroimaging patterns of Perinatal Hypoxic Ischemic Injury using MRI.

LITERATURE REVIEW

Hypoxic Ischemic Injury (HII) to the brain during perinatal period primarily results from diminished cerebral blood flow (ischemia) and reduced blood oxygenation (hypoxemia). Infants and children are more likely to suffer asphyxia events which results in ischemia and hypoxia. Global hypoxic ischemic insults do not affect all brain structures uniformly.[4]

In any given patient, the sites in the brain that tend to be most vulnerable to hypoxic injury will be determined largely by the maturity of the brain, which, in turn, is a function of patient age and, in infants, gestational age at birth. One must be aware of the degree of brain maturity at the time of the insult when interpreting studies for suspected HII.[4]

The severity of a hypoxic-ischemic insult also plays an important role in determining the distribution of injuries in the brain. Episodes of severe hypoxia-ischemia result in a different injury pattern compared with less severe insults. Duration of insult also seems to be a key determinant of the pattern of injury in HII, since insults of short duration usually do not result in brain injury. It has been suggested that, in the pediatric population, an arrest must typically last at least 15 minutes for brain injury to occur.[4]

MATERIAL AND METHODS

This is a cross sectional prospective case series study involving 60 patients with signs and symptoms varying from neonatal seizures and loss of consciousness to subtle cognitive decline in young adults.

MRI performed using 1.5 T Siemen's Magnetom Essenza Scanner. MR imaging protocol used was sagittal T1 weighted images, axial T1 & T2 weighted images and coronal, axial FLAIR sequence. Axial diffusion weighted, ADC and GRE images were also used.

SPECTRUM OF BRAIN INJURY WITH ROLE OF MR IMAGING

Hypoxic ischemic injury (HII) is more common in preterm neonates than in term neonates. The prevalence of injury shows an inverse relationship to gestational age at birth.[6]

Although some overlapping features exist, four major patterns of brain

injury are observed. These patterns are influenced by the combinations of the level of brain maturity at the time of the insult and the severity and duration of the hypoperfusion event.[6]

1. Mild-to-moderate hypoxic–ischemic injury in preterm neonates.
2. Profound hypoxic–ischemic injury in preterm neonates.
3. Mild-to-moderate hypoxic–ischemic injury in full-term neonates.
4. Profound hypoxic–ischemic injury in full-term neonates.

Mild-To-Moderate Hypoxic–Ischemic Injury In Preterm Neonates

The spectrum of brain injury in this group is broad and include white matter injury of prematurity (WMIP), germinal matrix–intraventricular hemorrhage, or a combination of both.[6]

White matter injury of prematurity or periventricular leukomalacia: Histological evolution of PVL follows a characteristic pattern-initially necrosis, often progressing to cavitation; then cysts collapse and result in gliosis and marked loss of the periventricular white matter.[6]

Earliest findings are seen on DWI showing restriction of diffusion in periventricular region which pseudonormalize within 5–7 days (**Fig.9**). Usually by 3–4 days, early white matter injury causes reactive astrogliosis and manifests as periventricular foci of T1 hyperintensity (without corresponding T2 hypointensity). Subsequently, by 2–6 weeks of age, some cases show periventricular cysts (cystic variant) and while end-stage PVL occurs by 6 months of life (**Fig.8**).[6]

End-stage PVL shows a characteristic appearance due to gliosis and loss of volume of the periventricular white matter and centrum semiovale. This results in ventriculomegaly with dilatation of the trigones and occipital horns, as well as wavy ventricular contour (**Fig.6, 7**). Thinning of the corpus callosum (**Fig.5, 7**) is a characteristic late feature and is particularly noted posteriorly (involving the posterior body and splenium).[6]

PVL is most commonly seen as white matter hyperintensity adjacent to lateral ventricle at peritrigonal (**Fig. 1-7**) and foramen of Monro region.[6]

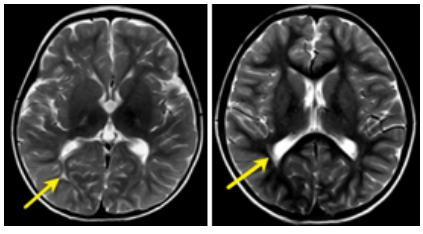

Figure 1. Axial T2WI in a 8yr old male with past history of delayed cry after birth shows mild periventricular hyperintense signal in the peritrigonal region.

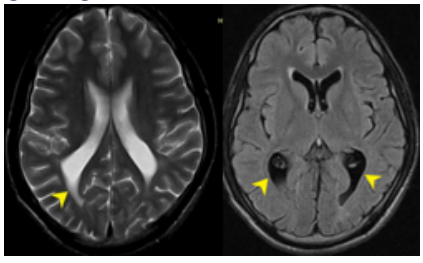

Figure 2. Axial T2 and FLAIR images in 21yr old male presenting with mild cognitive disability shows periventricular T2 hyperintense signal and prominence of bilateral occipital horns.

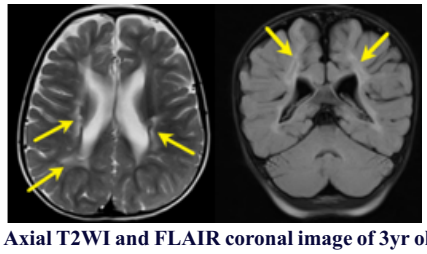

Figure 3. Axial T2WI and FLAIR coronal image of 3yr old female with history of preterm delivery shows periventricular hyperintense signal with prominence of bilateral lateral ventricles.

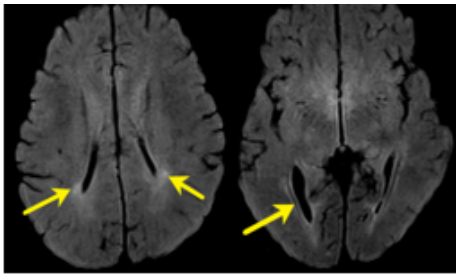

Figure 4. Axial FLAIR images of a 12yr old female with complaints cognitive disabilities shows prominence of occipital horns of bilateral lateral ventricles with periventricular hyperintense signal.

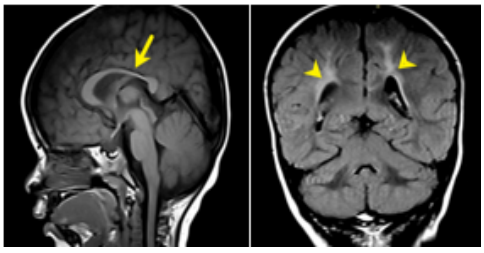

Figure 5. Sagittal T1 WI and coronal FLAIR images of a 1yr old female with low birth weight history shows periventricular hyperintense signal and mild thinning of the posterior part of body of corpus callosum.

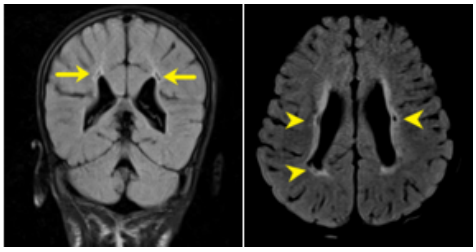

Figure 6. Coronal and axial FLAIR images of a 4yr old boy with complaints of seizures since 2 years and birth history of cord prolapse shows tiny periventricular cysts and prominent ventricles with irregular contour.

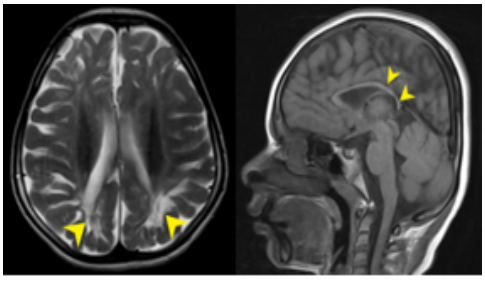

Figure 7. Axial T2WI of a 6yr old female who is a known case of neonatal seizures and hypoglycemia with preterm birth history shows periventricular T2 hyperintense signal in the posterior aspect with undulating ventricular lining. Sagittal T1WI of the same patient shows thinned out corpus callosum predominantly affecting the splenium.

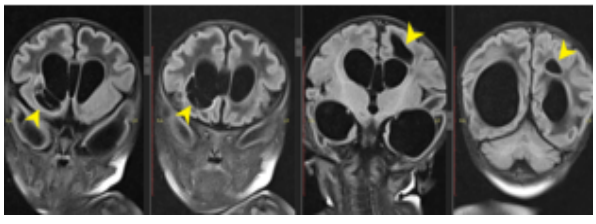

Figure 8. Serial coronal FLAIR images of a 2month old baby boy with maternal antenatal history of severe pre-eclampsia shows multiple focal areas of cystic encephalomalacia (Porencephaly) in bilateral fronto-parietal periventricular and subcortical white matter region with non-communicating hydrocephalus.

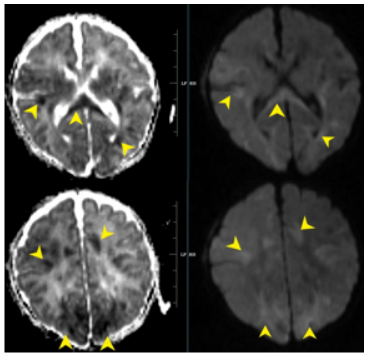

Figure 9. Axial DWI and ADC images of a 2 days old female with history of low birth weight and preterm delivery at 32 weeks shows areas of restricted diffusion involving splenium of corpus callosum and cortical-subcortical white matter of bilateral fronto-parietal peri-Sylvian region. Subtle restriction also seen in the periventricular region of the bilateral occipital horns.

Germinal matrix–intraventricular hemorrhage (GM-IVH):
It is unique to the immature brain and is never seen beyond the neonatal period. GM hemorrhages mostly originate from caudothalamic groove. Hypoxic ischemia causes damage to the capillaries of the GM and subsequent reperfusion results in GMH. Cranial ultrasound is generally adequate in this group, and MRI is used to detect concomitant deep grey matter injury and PVL.[6]

Profound Hypoxic–ischemic Injury in Preterm Neonates
Severe hypotension most frequently injures the early myelinating and metabolically active thalami, dorsal brainstem, and anterior vermis. There is less common involvement of the basal ganglia, hippocampus, perirolandic cortex, and corticospinal tract. This regional preference of injury is explained by the early myelination of thalamus and globus pallidus by 24–25 weeks of gestation and late myelination of corpus striatum (caudate nucleus and putamen) and perirolandic cortex beyond 35–36 weeks of gestation. There may be associated GMH or PVL.[6]

The earliest MRI finding is diffusion abnormality in thalami and basal ganglia (**Fig.10, 11**). There is T1 and T2 hyperintensity by approximately 7 days. When involved, the basal ganglia shows cavitation and volume loss without gliosis.[6]

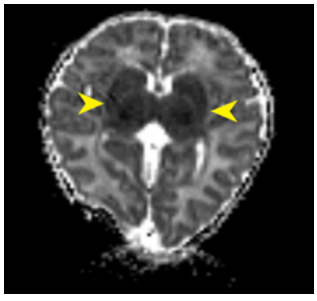

Figure 10. Diffusion abnormality in a 3 week old baby girl with history of NICU stay i/v/o preterm delivery at 32 weeks and maternal eclampsia shows low ADC values in bilateral basal ganglia and thalami.

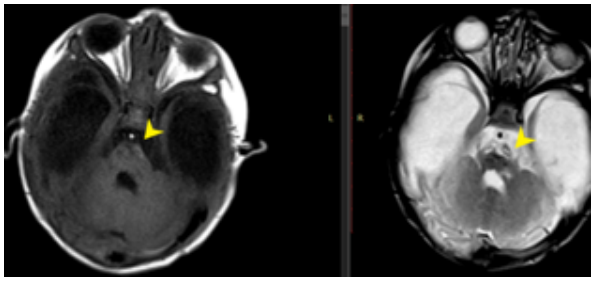

Figure 11. Axial T1 and T2WI sections of a 3 months old baby girl with history of NICU stay due to low birth weight and preterm delivery at 30 weeks shows Wallerian degeneration/gliosis seen in the left cerebral peduncle of the mid brain.

Profound Hypoxic–Ischemic Injury In Full-Term Neonates (Acute Severe Asphyxia, Basal Ganglia-Thalamus Pattern, Selective Neuronal Necrosis)
This pattern of injury is usually seen following an acute sentinel event such as ruptured uterus, placental abruption, or cord prolapse. Hence, this is also referred as a pattern following "acute near severe asphyxia." Because the injury primarily involves the bilateral ventrolateral thalami and posterior putamina, it is also known as basal ganglia–thalamus pattern (BGT).[6]

The injury primarily affects the deep gray matter–posterior putamina, ventrolateral thalami, hippocampi, perirolandic cortex, dorsal brainstem, and also involves the cerebellum in prolonged severe hypoxia (**Fig.12-14**). Usually minor cortical injuries may be seen, and more prolonged insults result in diffuse cortical involvement. As described, DWI is the first sensitive modality beginning from the first day of life. The involved regions show T2 hyperintensity, particularly in the ventrolateral thalami and posterior putamina. Extensive injury involving gray and white matter finally results in cystic encephalomalacia.[6]

Another finding is the absent posterior limb sign. It is the nonvisualization of the normal increased signal intensity of the posterior limb of the internal capsule (PLIC) on T1WI in a term neonate (after 37 weeks' of gestational age). T2WI shows corresponding decreased signal intensity in the PLIC and is more useful for determining the exact location of PLIC.[6]

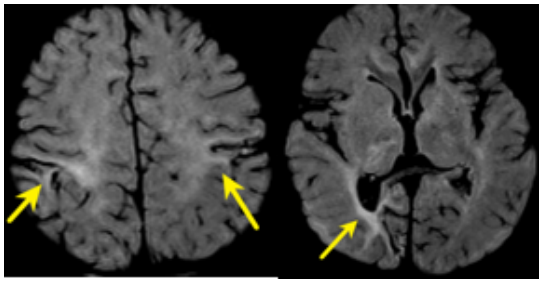

Figure 12. Axial FLAIR images of a 3yr old term baby boy with complaints of recurrent seizures and maternal history of preeclampsia shows FLAIR hyperintense signal in bilateral thalami, bilateral peri-Rolandic region and optic radiation on right side representing gliosis.

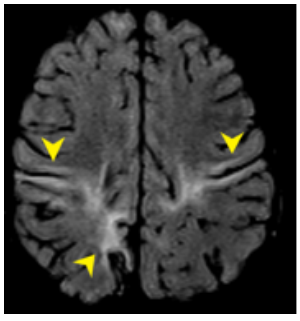

Figure 13. Axial FLAIR image of a 6yr old with signs of neurocognitive disability and birth history of caesarean section for obstructed labor shows hyperintense signal in bilateral cortical and subcortical parietal and peri-Rolandic region.

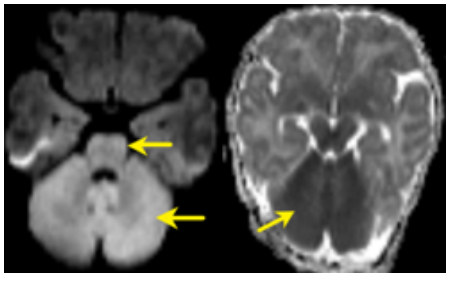

Figure 14. Axial DWI and ADC images of a 1month old full term male baby having history of delayed cry after birth and maternal antenatal history of Abruptio placenta shows diffusion restriction with low ADC values involving the brainstem and the cerebellum.

Mild-To-Moderate Hypoxic–Ischemic Injury In Full-Term Neonates (Prolonged Partial Asphyxia, Watershed Pattern)

Prolonged partial asphyxia results in injury to the watershed zones of cerebrum, i.e, parasagittal white matter, and whenever severe, extending to the overlying cortex. This is due to the relative hypoperfusion of these areas as the result of autoregulation. The major etiologies for this type of injury are prolonged difficult delivery and long standing antenatal risk factors. Again, DWI is the earliest to change and show cortical and subcortical white matter restriction (**Fig.15**). Later, T2WI may often show cortical swelling, loss of gray-white differentiation, and hyperintensity in the cortical and subcortical white matter.[6]

Ulegyria (shrunken cortex with flattened mushroom shaped gyri) and diminished white matter volume, predominantly in the parieto-occipital region, is seen in the chronic stage (**Fig.16**).[6]

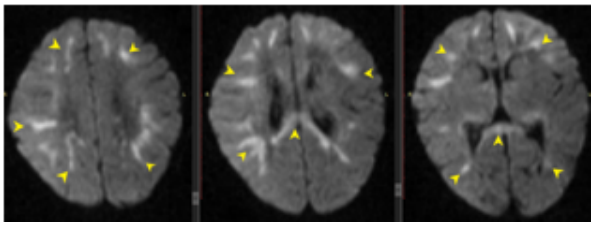

Figure 15. Serial axial DWI images of a 4 day old full term female baby with history delayed cry after birth and neonatal hypoglycemia shows multiple areas of restricted diffusion seen in bilateral watershed territories. Restricted diffusion also seen in corpus callosum and around the occipital trigones.

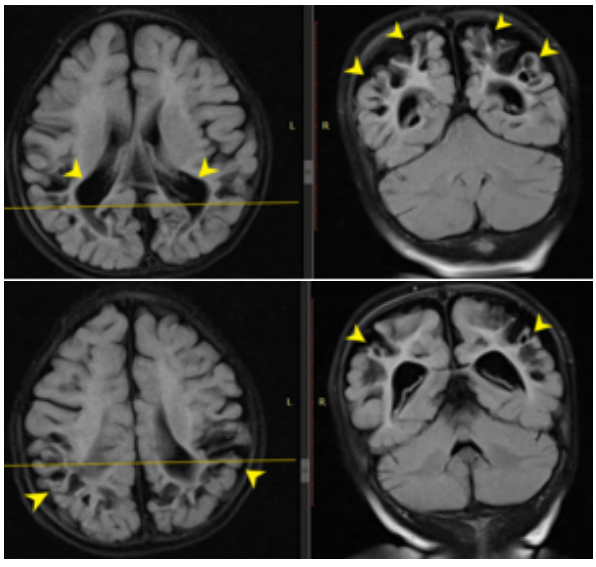

Figure 16. Axial and coronal FLAIR images of 1year old full term female baby with recurrent seizures, neonatal hypoglycemia and maternal history of pre-eclampsia shows bilateral poarieto-occipital cortical atrophy with subcortical gliosis, mushroom shaped gyral pattern and ventricular prominence.

DISCUSSION

In the present study, it was observed that the most common age group affected were the preterm neonates who later presented with complaints ranging from subtle neurocognitive disability to recurrent seizures. Most common pattern of hypoxic injury seen in this study was of periventricular white matter changes with prominence of bilateral lateral ventricle predominantly affecting the occipital horns and the trigones. Second most common finding was that of gliosis signal in cortical and subcortical region predominantly the parieto-occipital and peri-rolandic region. Involvement of brainstem, cerebellum and basal ganglia was seen in only severe cases of HIE thus justifying the phenomenon of preserving blood flow to these structures in cases of mild to moderate hypoxic injury.

CONCLUSION

MRI thus plays a crucial role in providing detail insight into the nature and spectrum of perinatal hypoxic ischemic injury.

Preterm neonates show less severe involvement of basal ganglia. Term neonates show frequent involvement of perirolandic cortex.

Common findings of HIE on MRI includes prominence of bilateral lateral ventricles predominantly the occipital horns, trigone and periventricular T2/FLAIR hyperintensities.

Mild to moderate perinatal HII involves periventricular region & germinal matrix in preterm and watershed zones in term neonates. Profound hypoxia involves mostly the deep gray matter irrespective of maturity.

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