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Research Article

Clinico-Radiological Correlation of Hypoxic-Ischemic Brain Injury on MRI: An Observational Study during Newborn Period in a Tertiary Care Centre in Bihar

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ABSTRACT

Objective: Birth asphyxia is hypoxic and ischemic insult to the newborn brain during the process of birth. This can lead to different changes in brain through multiple mechanism, known as hypoxic ischemic encephalopathy (HIE). There are multiple antenatal risk factors responsible birth asphyxia leading to multisystem involvement in the background of HIE. This is one of the significant causes of neonatal morbidity and mortality as well as poor neurodevelopmental outcome. Hence, we conducted our study with the objective of evaluating MRI brain findings at admission with clinical features of birth asphyxia .

Methods: This was hospital based cross-sectional study among 50 newborn babies with features of stage-II and stage -III HIE as per THOMPSON scoring. All included HIE babies were undergone MRI brain after stabilization. MRI findings were analyzed in different setting with clinical stages to identify patterns of injury of brain parenchyma and prognostic significance. Data were subjected to statistical analysis. Outcome was determined as discharged or death among HIE babies.

Results: Out of 50 HIE babies, 39 babies were in stage-II and 11 babies were in stage-III. Risk factors for birth asphyxia found as 18% oligohydramnios, 12% pregnancy induced hypertension (PIH), 24% leaking per vaginum, 18% eclampsia, 14% abruptio placenta and 8% some others. MRI brain finding was normal in 32% HIE babies, while 28% showed involvement in fronto-parietal involvement , 14% basal ganglia involvement, 4% with intraventricular hemorrhage, 2% cortical laminar necrosis, 4% cortical atrophy, 6% loss of differentiation, 2% brain edema, 6% cystic white matter, and 2 % with gliosis. Besides, multiple HIE babies presented with ulegyria and a few with multicystic encephalopathy.

Conclusions: Severe stages of HIE were found to be associated with MRI brain involvement in different white matter and grey matter area involvement.

Keywords*: Birth Asphyxia, HIE, MRI Brain, Ulegyria, Basal Ganglia, Fronto-parietal white matter.*

INTRODUCTION

Hypoxic Ischemic Encephalopathy (HIE) is an acute, non-static encephalopathy resulting from reduced oxygen and blood flowto the brain during the perinatal period. It typically manifests within the first few days of life and is a major cause of neonatal mortality and long-term neurodevelopmental disabilities such as cerebral palsy, epilepsy, and cognitive impairment[1].

The basic underlying mechanism is birth asphyxia, a combination of hypoxemia (decreased oxygen content in blood) and ischemia (reduced cerebral perfusion) [2,3]. Common perinatal risk factors include umbilical cord prolapse, uterine rupture, abruptio placentae, placenta previa, maternal hypotension, and shoulder dystocia. Clinical indicators of HIE include

abnormal fetal heart rate patterns, poor Apgar scores, metabolic acidosis in cord blood, meconium-stained amniotic fluid, and the need for resuscitation at birth[4,5,6].

The pathophysiology of HIE progresses through two main phases: primary energy failure and secondary energy failure[7]. The initial insult causes reduced cerebral blood flow, depletion of oxygen and glucose, and impaired ATP production, leading to neuronal depolarization, calcium influx, and release of excitatory neurotransmitters like glutamate[8,9,10,11]. This results in neuronal necrosis and apoptosis. Once blood flow is restored, there is a brief period of recovery known as the latent period, which is characterized by normal cerebral metabolism. This latent period is considered the optimal timing of therapeutic interventions[8,12,13]. The subsequent secondary phase, occurring 6–48 hours later, involves oxidative stress, excitotoxicity, and inflammation, which further exacerbate brain injury. Oxidative stress is harmful to the neonatal brain due to low concentrations of antioxidants and high consumption of oxygen when transitioning from fetal to neonatal life[14,15].

Despite advances in perinatal care, HIE continues to pose a significant challenge, particularly in low- and middle-income countries where incidence rates are higher[16]. Early recognition and timely intervention during the latent phase, such as therapeutic hypothermia, are crucial in improving neurological outcomes and minimizing permanent brain damage.

Neuroimaging plays a vital role in evaluating infants with hypoxic-ischemic encephalopathy (HIE) by identifying brain lesions and predicting outcomes. MRI is the preferred and safest modality, revealing the extent, nature, and pattern of injury, which depend on brain maturity, severity, and duration of hypoxia. It helps in prognosticating the outcome and planning neurodevelopmental therapy[17]. Hypoxic-ischemic injury to gray matter (deep gray matter, cortex) demonstrates characteristic T1 hyperintensity and variable T2 intensity, depending on the time at imaging and the dominant underlying pathologic condition, such as hemorrhage or gliosis. Injury to white matter generally results in T1 hypointensity and T2 hyperintensity due to ischemia induced edema[18] . Diffusion-weighted imaging (DWI) is sensitive for detection of injury in the first 24 hours, during which time the conventional T1 – and T2- weighted images may appear normal[17]. MRI findings in the second week can diagnose 75–100% of lesions, especially in the basal ganglia, thalami, and white matter. Abnormal signals in the posterior limb of the internal capsule and delayed myelination indicate poor neurodevelopmental prognosis.

In this background, we conducted this study to evaluate MRI findings in neonates with birth asphyxia for identifying injury patterns, comparing term and preterm differences, and predicting clinical outcomes.

METHODS

This hospital-based cross-sectional observational study was conducted in the departments of Pediatrics and Radio-diagnosis of our institute from April 2023 to August 2024. Fifty neonates (term and preterm) with clinically diagnosis of birth asphyxia (HIE Stage II/ MODERATE and Stage III/ SEVERE with Thompson Scoring) were included. All babies underwent MRI brain study under appropriate sedation. Neonates aged 1–14 days and hemodynamically stable were included, while those with Stage I HIE, major congenital anomalies, severe anemia, or cardiorespiratory disease were excluded. MRI findings were analyzed and correlated with clinical stages to identify patterns of injury and prognostic significance. Data were subjected to statistical analysis, and informed consent was obtained from parents before the procedure.

STATISTICAL ANALYSIS

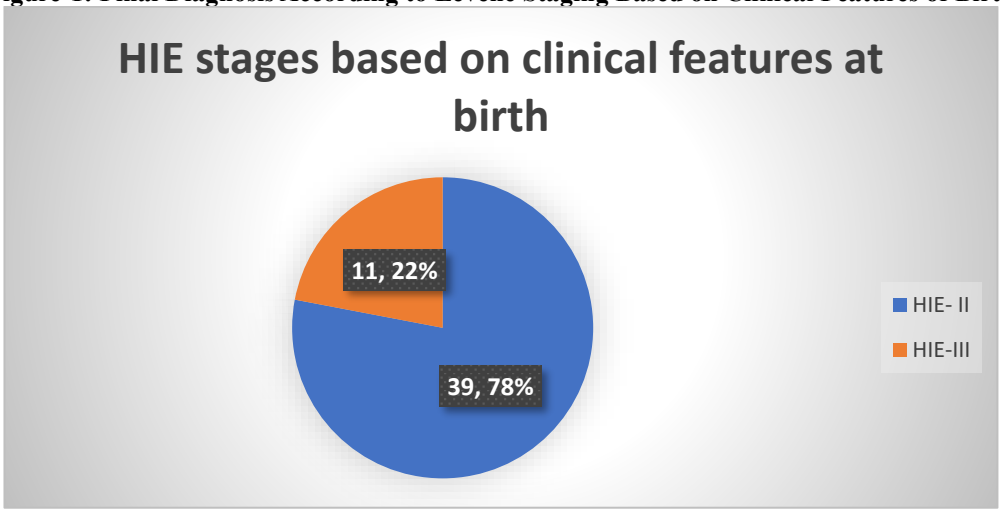
The following statistical methods has been employed in the present study.

1. Statistical Analysis has been performed with help of Epi Info (TM) 7.2.3.1 of the Centers for Disease Control and Prevention (CDC) & SPSS Statistics (IBM Corp.).
2. Test of proportion (Z-test) has been used to test the significant difference between the two proportions. A t-test has been performed to compare the means. Odds ratio (OR) with a 95% Confidence Interval (CI) has been calculated to measure the different risk factors.

RESULTS

Among the included 50 HIE neonates, 39 newborn babies (78%) had moderate HIE (Stage-II) while 11 babies (22%) had severe HIE (Stage-III). [Figure-1]

Figure-1: Final Diagnosis According to Levene Staging Based on Clinical Features of Birth



Out of 50 newborns, 18 with HIE II&3 with HIE III were admitted within 6 hours of life; 13 with HIE II, and 8 with HIE III were admitted within 7-12 hours of life; 8 with HIE II& no baby with HIE IIIwere admitted in 13-18 hours of life.14 (70%)newborn in HIE II and 6 (30%) in HIE III , were born through LSCS. Similarly 25 (83.3%) in HIE II and 5 (16.7%) in HIE III were born through NVD. Out of 50 newborns, 7 (77.8%) patients in HIE II and 2 (22.2%) in HIE-III had risk factor as oligohydramnio, 4 (66.7%)patients in HIE IIand 2 (33.3%)in HIE III had risk factor as pregnancy-induced hypertension (PIH) as a risk factor. 11 (91.7%) patients in HIE II, 1 (8.3%) in HIE III had leaking per vaginum as a risk factor. 3 (50%) patients in HIE II ,3 (50%) in HIE III presented eclampsia as risk factor. 6 (85.7%) patients in HIE II and 1(14.3%) in HIE III had abruption placentae as a risk factor. 2 (50%) patients in HIE II and 2 (50%) in HIE III had others (CPD, Obstructed labor, cord prolapse) risk factor. All "p" values were statistically insignificant (p>0.05; Not Significant).

Regarding resuscitation of HIE-IIbabies, 10 newborn needed suction stimulation, 13 babies received bag & mask ventilation, 9 babies required intubation and 2 babies needed adrenaline infusion. For HIE-III babies, 9 babies received suction-stimulation, 7 babies received bag and mask ventilation, 4 babies with intubation and 2 babies were given adrenaline infusion .[Table-1]

Table-1: Perinatal factors associated with both stages of HIE

		HIE-II (n=39)	HIE-III (n= 11)	TOTAL (n=50)
Age (Admission at hours)	**≤ 6**	18	3	21 (42%)
	7-12	13	8	21(42%)
	13-18	8	0	8 (16%)
Mode of Delivery	**LUCS**	14	6	20 (40%)
	NVD	25	5	30 (60%)
Risk factors	**Oligohydramnio**	7	2	9 (18%)
	PIH	4	2	6 (12%)
	Leaking per vaginum	11	1	12 (24%)
	Ecclampsia	3	3	9 (18%)
	Abruptio placenta	6	1	7 (14%)
	Others	2	2	4 (8%)
Resuscitation	**Suction & stimulation**	10	9	19 (38%)
	Bag & Mask	13	7	20 (40%)
	Intubation	9	4	13 (26%)
	Adrenalin	0	2	2 (4%)
p Value >0.05				

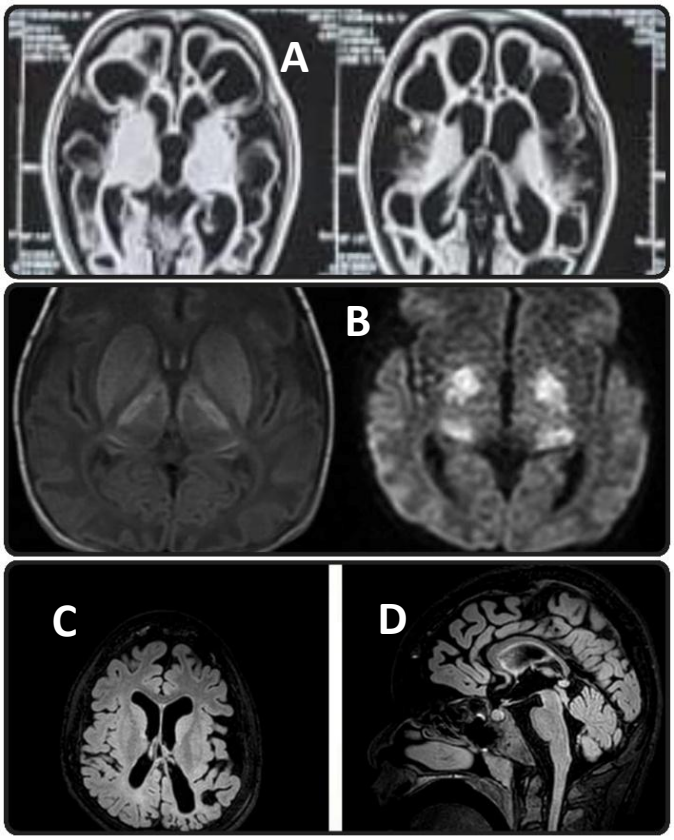
MRI brain finding is described in Table-2 and Figure-2. 16 out of 50 babies showed normal finding (32%). Predominant involvement wasin fronto-parietal white matter (28%) followed by basal ganglia and thalamus involvement (14%).

Table-2: MRI Brain finding of babies with HIE

MRI FINDING AT ADMISSION	HIE-II	HIE-III	TOTAL (n=50)
Normal	16	0	**16 (32%)**
Basal Ganglia and thalamus	5	2	**7 (14%)**
Frontoparietal white matter	12	2	**14 (28%)**
Intraventricular Hemorrhage	1	1	**2 (4%)**
Cortical laminar necrosis	1	0	**1 (2%)**
Cortical atrophy	0	2	**2 (4%)**
Loss of differentiation	2	1	**3 (6%)**
Brain edema	0	1	**1 (2%)**
Cystic white matter	2	1	**3 (6%)**
Gliosis	0	1	**1 (2%)**
Total	39	11	**50**

d.f.18; p= 0.000

Figure-2 : MRI brain imaging



A. MRI (FLAIR) image showing multiple encephalopathy
B. Injury of Basal Ganglia & Thalamus in term newborn (T1 image, DWI)
C &D . Ulegyria in Flair MRI image

Out of 50 babies. 31 babies were discharged (62%) and 19 babies with birth asphyxia expired (38%). However, among 39 HIE-II babies 9 babies expired (47.4%) and among

11 HIE-III babies 10 babies expired (90.90%). [TABLE-3]

Table-3:Correlation between outcome and diagnosis

Outcome	HIE II (n=39)	HIE III (n=11)	TOTAL (n=50)
Death	9 (23.07%)	10 (90.90%)	19 (38%)
Discharge	30 (76.92%)	1 (9.09%)	31 (62%)
Total	39	11	50

d.f. 4; p=0.000

DISCUSSION

Present study was conducted to find the clinical and MRI features in neonates with HIE at admission.50 newborns were included in the study. 39 (78%) patients were in HIE II and 11(22%) patients were in HIE III. Out of 50 newborns, 21 (42.0%) cases presented at ≤6 hours of life, 21 (42.0%) between 7-12 hours, and 08 (16.0%) cases at 13-18 hours. 20 (40.0%) cases were LSCS born and 30 (60.0%) cases were NVD born. Oligohydramnios was present in 9 (18.0%) pregnancies, history of PIH was present in 6 (12.0%) pregnancies. LPV was present in 12 (24.0%) pregnancies eclampsia was present in 6 (12.0%) pregnancy abruption placenta was present in 7 (14.0%) pregnancies. Other (CPD, Obstructed labor, cord prolapse) risk factors were present in 4(8%) pregnancies. Out of 50 newborns, suction and stimulation were required in 19 (38%) newborns, Bag and Mask were done in 20 (40%) babies, 13 (26%) babies required intubation, and 2 (4%) babies required drugs (Adrenalin). This was like the study conducted by Patil B et al(2015) in which 37 cases were included in the study, 22 were maleand 15 were female. Different Risk factors were found during pregnancy in 29 cases. 9 babies were delivered by LSCS, 25 were delivered by NVD, and 3 by assisted vaginal delivery[19].21 cases were of HIE stage-II and 16 cases were of stage-III. In our study, MRI was done to evaluate the changes of hypoxia in the brain.Out of 50 newborns, 16 (32%) had normal MRI, 7 (14%) had hyperintensity in basal ganglia and thalamus, 14(28%) patients had hyperintensity in frontoparietal white matter, 2(4%) patients had intraventricular hemorrhage, 1 (2%) patient had cortical laminar necrosis, 2(4%) patient had cortical atrophy, 3(6%) patient had a loss of differentiation between gray-white matter, 1(2%) patient had brain edema, 3(6%) patients had a cyst in white matter and 1(2%) patient had gliosis.Patil B et al(2015) described that among 37 cases, 12 (32.4%) had normal MRI, 10 (27%) showed abnormal signals in the basal ganglia/thalamus, and 20 (54%) showed abnormal signal in the cortex. 5 (13.5%) cases showed abnormal signals in the cortex and the basal ganglia, 63 newborns had the watershed pattern (52%), 26 had the basal ganglia/thalamus pattern (22%), and 32 had normal MRI results (26%)[19]. At LLUCH, 15 newborns had the watershed pattern (29%), 18 had the basal ganglia/thalamus pattern (35%) and 19 had normal MRI results (37%)[20]. According to the data reported by different studies, alterations on MRI performed at the end of the first week or the beginning of the second week of life are evident in 75%–100% of patients with stage I-III HIE. The main types of lesions include a watershed pattern of injury and an isolated or concomitant basal ganglia/thalamus pattern. The injuries of the cortical gray matter or internal capsule posterior limb are documented to be less frequent. The findings of the present study were like the study conducted by Jose A et al(2013) in which of the 31 cases, only 26 could undergo an MRI brain (four deaths, one lost to follow-up). Among the 26 cases, 16 (61.5%) had normal MRIs, two (7.7%) showed abnormal signals in the basal ganglia/thalamus, and two (7.7%) showed abnormal signals in the cortex. Six (23.1%) cases showed abnormal signals in the cortex and the basal ganglia.[21] Out of 50 newborns, 39 (78%) patients were in HIE II and 11(22%) patients were in HIE III. 7 (77.8%) patients in HIE II, 2 (22.2%) in HIE III of pregnancies with oligohydramnios., 4 (66.7%)patients in HIE II 2 (33.3%)in HIE III of pregnancies with pregnancy-induced hypertension 55 (PIH) as a risk factor. 11 (91.7%) patients in HIE II, 1 (8.3%) in HIE III of pregnancies with leaking per vaginum as a risk factor. 3 (50%) patients in HIE II ,3 (50%) in HIE III of pregnancies with eclampsia as risk factors. 6 (85.7%) patients in HIE II and 1(14.3%) in HIE III of pregnancies with abruption placentae as a risk factor. 2 (50%) patients in HIE II and 2 (50%) in HIE III of pregnancies with Others (CPD, Obstructed labor, cord prolapse) as a risk factor. In an earlier study of risk factors for neonatal encephalopathy conducted by Miller SP, et al (2005)[20], 69% of cases had antepartum risk factors, 24% had both antepartum and intrapartum risks, and 5% only had intrapartum risks. Although many risk factors are prenatal,6 recent evidence from prospective cohorts of neonatal encephalopathy using MRI demonstrates that the brain injury happens close to the time of birth. The MRI findings were also consistent with recent rather than chronic brain injury, and the antenatal conditions measured were remarkably similar in newborns with normal and abnormal MRI scan results.[20] Out of 50 newborns, 16 (100%) were HIE II and none in HIE III had normal MRI. 5 (71.4%) in HIE II and 2 (28.6%) in HIE III had basal ganglia, and thalamus hyperintensity. 12 (85.7%) in HIE II and 2 (14.3%) in HIE III had frontoparietal white matter involvement. 1 (50.0%) in HIE II and 1 (50.0%) in HIE III had an intraventricular hemorrhage. 1 (100.0%) in HIE II and none in HIE III had cortical laminar necrosis. none in HIE II and 2 (100.0%) in HIE III had cortical atrophy. 2 (66.7%) in HIE II and 1 (33.3%) in HIE III had a loss of differentiation. None in HIE II and 1 (100.0%) in HIE III had brain edema. 2 (66.7%) in HIE II and 1 (33.3%) in HIE III had cystic white matter. none in HIE II and 1 (100%) in HIE III had gliosis. All “p” values were statistically insignificant. Mary A. Rutherfordetal(2010) deduced in their study that TCUS had relatively higher sensitivities in lesions at the periventricular white matter (79.5%), subcortical white matter (71.9%), and deep gray matter (71.1%) rather than lesions at the cortex (58.8%), corpus callosum (50%) and the brainstem (26.7%)[22]. MRI is the reference standard for infant brain imaging. It is needed in most neonates with suspected parenchymal brain injury or neurological manifestations. MRI is an indispensable and conclusive evaluation modality on account of its superior sensitivity and pathology discriminating

techniques. Diffusion-weighted imaging (DWI) often reveals ischemic brain injury at an earlier stage than conventional MR imaging. Cowan et al(2003) performed MRI within the first 2 weeks after birth, which was able to show that more than 90% of affected newborns had evidence of perinatally acquired lesions on their MRI, with a very low rate of established antenatal brain injury. The presence of ventricular dilatation, widening of the subarachnoid space and interhemispheric fissure, and presence of germinolytic cysts or cystic lesions in the white matter, seen at birth or during the first week, are all suggestive of an antenatal insult or an underlying problem, for instance, a metabolic disorder. A reduced apparent diffusion coefficient can be calculated, showing reduced values (restricted diffusion) during the first few days after the insult, with pseudo normalization by the end of the first week. Sequential imaging has shown that lesions in the basal ganglia may increase in size and site during the first week after birth[23]. Barkovich et al(1997) in their sequencial imaging in ten newborn infants noted that patterns of injury varied considerably during the first 2 weeks after injury. The appearance of new areas of reduced diffusion simultaneous with the pseudo-normalization of areas that had reduced diffusion at earlier times could result in an entirely different pattern of injury[24]. In neonates with HIE early MRI may show evidence of brain swelling. This clears during the first week of life as signs of permanent damage become more obvious. Abnormal signal intensities may be seen in the basal ganglia and thalami, the internal capsule, the periventricular and subcortical white matter, and the cortex. In term 57 infants with HIE, there may be late MRI abnormalities within the basal ganglia, the cortex, the white matter, the hippocampi, the insulae, and the brainstem.

LIMITATION

This was a single center time bound study with minimal sample. MRI brain was done only once only.

What this Study Adds:

The present study added data to the published literature about the importance of MRI to detect involvement of different zone of brain among the babies suffering from birth asphyxia. More severe the stages, more chances of involvement in grey matter , white matter as well as basal ganglia. We got unusually increased numbers of ulegyria in our study group. Early MRI brain following the diagnosis of HIE can detect the exact area of involvement in details to predict the prognosis of baby and neurodevelopment outcome.

Conflict of interest: Nil
Founding: Not Declared

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