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RESEARCH ARTICLE

EVALUATION OF ROLE OF MRI BRAIN IN HYPOXIC ISCHEMIC INJURY IN PRE TERM AND TERM INFANTS/CHILDREN

[1,] Dr. Rushabh Bhikhabhai Suthar, [2],*Dr. (Col) Sunita Dashottar and [3]Dr. RDS Ahluwalia

[1]Principal, Department of Radiodiagnosis and Imaging, Command Hospital, Lucknow, Uttar Pradesh, India
[2]Senior Advisor and HOD, Department of Radiodiagnosis and imaging, Command Hospital, Lucknow, Uttar Pradesh, India
[3]Professor and HOD, Department of Anesthesiology, Command Hospital , Lucknow, Uttar Pradesh, India

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ABSTRACT

Introduction: Birth asphyxia is one of the common causes of early neonatal mortality. Hypoxic-ischemic encephalopathy (HIE) leads to severe neurological deficits in neonates which can result in early neonatal deaths. MRI brain is the modality of the choice to look for various patterns of hypoxic ischemic injury. **Materials and methods:** Hospital based prospective study was done on 30 term and preterm infants/children with history of birth asphyxia and other neonatal, maternal and placental causative factors to look for MRI patterns of HIE. MRI Brain was used as screening modality to look for evidence of hypoxic ischemic injury (HIE). **Results:** The study demonstrated that term infants had significant involvement of subcortical and/or periventricular white matter and basal ganglia/thalamus region. While the preterm patients had predominant periventricular leucomalacia type of involvement. **Conclusion:** MRI of the brain is the ideal imaging modality of choice for the diagnosis and follow-up of infants/children suspected/diagnosed with hypoxic-ischemic encephalopathy (HIE). The pattern of brain injury depends on brain maturity at the time of hypoxic insult, duration and severity of insult.

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INTRODUCTION

Birth asphyxia is one of the common causes of early neonatal mortality. Hypoxic-ischemic encephalopathy (HIE) leads to severe neurological deficits in neonates with resultant early neonatal deaths (Kurinczuk *et al*., 2010). Among the institutional births, the incidence of asphyxia is 5% and which in turn responsible for 24.3% of neonatal deaths (Neonatal morbidity and mortality, 1999). Cerebral palsy is the most important long-term outcome of birth asphyxia which may be associated with seizure disorder, mental retardation or other motor/sensory disabilities (Rajeshwar Reddy, 2004). The pattern of hypoxic brain injury depends on the duration and severity of hypoxia and on status of brain maturation. The MRI imaging findings in full-term neonates (>36 weeks of gestation) may differ from those in preterm neonates (<36 weeks of gestation) (Barkovich, 2005). Imaging modalities like ultrasound, computed tomography (CT) scan and magnetic resonance imaging (MRI) are used in identification and characterization of the precise location, extent and severity of the brain injury.

Corresponding author: **Dr. (Col) Sunita Dashottar,**
Senior Advisor and HOD, Department of Radiodiagnosis and imaging, Command Hospital, Lucknow, Uttar Pradesh, India.

Advanced imaging techniques such as diffusion-weighted imaging (DWI) and magnetic resonance spectroscopy (MRS) are more sensitive to diagnose acute brain injury and therefore have significant role in early diagnosis and timely intervention (Barkovich, 2001; Zarifi *et al*.,20026). Improved antenatal are will definitely bring down the incidence of birth asphyxia. Once the cerebral injury occurs, the management is mainly supportive. Most of the severely affected babies do not survive and the patient without systemic involvement has excellent outcome (Vishnu Bhat, 2005). MRI of the brain is the ideal imaging modality of choice for the diagnosis and follow-up of infants/children suspected with hypoxic-ischemic encephalopathy (HIE) (Huang *et al*., 2008; Latchaw, 1995; Rutherford *et al*., 1996).

Inclusion criteria: MRI was performed as a screening test to diagnose hypoxic ischemic encephalopathy (HIE).

Following criteria were formulated for inclusion of the patient in the study:

- Pre term or term infants, children < 12 yrs of age
- History of birth asphyxia and/or evidence of fetal distress

- Patients with history of adverse neonatal, maternal or placental factors
- Patients presented with seizure disorder, speech deficit, gross developmental delay, cerebral palsy or other sensory/motor neurological deficits.

Equipment: MRI machine 1.5-T Magnetom Avanto, M/S Siemens AG, Germany.

RESULTS

The study is a hospital based prospective study conducted for a period of two years. Our study population consisted of 30 patients with history of birth asphyxia and/or fetal distress, adverse neonatal, maternal or placental factors and other clinical variables.

Table 1. Age distribution of patients

Age Group	No of patients	Percentage (%)
<1 yr	9	30
1-5 yrs	14	46.67
5-11 yrs	7	23.33
TOTAL	30	100%

Table 2. Sex distribution of the patients

Sex	No of Patients	Percentage (%)
MALE	18	60
FEMALE	12	40
TOTAL	30	100

Table 3. Distribution of risk factors in patients

Risk factors	No of patients	Percentage (%)
Neonatal	17	56.66
Maternal	10	33.33
Placental	3	10
Not known	5	16.66

Table 4. Distribution of patients according to gestational age

Preterm/term	No of Patients	Percentage (%)
Term	18	60
Preterm	12	40

Table 5. Apgar score at 5 minutes in the patients

Apgar Score	No of Patients	Percentage (%)
<= 3	5	16.66
4-7	12	40
>7	13	43.34

Table 6. Imaging findings in preterm neonates

Mri Findings	Number of Patients	Percentage (%)
Periventricular leucomalacia	9	75
Cerebral atrophy	1	8.33
Corpus callosum thinning	1	8.33
Encephalomalacia in bilateral cerebral hemispheres	2	16.66

The age of patients ranged from 0 years to 11 years. Maximum numbers of children were in the age group of 1-5 yrs constituting 14 (46.67%), followed by the age group of < 1 yr constituting 9 (30 %). In this study group of 30 patients, 18 were males and 12 were females. Thus, males were more commonly affected than females. In this study the risk factors were categorized according to neonatal, maternal, placental and not known. In this study, 12 patients had a history of preterm delivery and 18 patients had term births. In our study, 12 cases of preterm neonates who suffered perinatal hypoxia were evaluated by MRI Brain.

The most common finding in these cases were periventricular leucomalacia (75%). Other findings were cerebral atrophy (8.33%), corpus callosum thinning (8.33%) and encephalomalacia in bilateral cerebral hemispheres (16.66%). 18 cases of term neonates who had history of perinatal hypoxia underwent MRI Brain. Most common finding was T2/FLAIR hyperintensities in subcortical and/or periventricular white matter region. Other findings were T2/FLAIR hyperintensities in bilateral basal ganglia and/or thalami, acute infarcts in cerebral hemispheres, cerebral atrophy, encephalomalacia in bilateral cerebral hemispheres and periventricular leucomalacia.

RESULTS

The study was performed to evaluate the role of MRI to characterise patterns of CNS involvement in pre term and term infants/children. 30 patients with history of perinatal asphyxia and/or fetal distress fulfilling the inclusion criteria were studied in which 18 (60 %) were male and 12 (40 %) were female patients. According to the age of the patients, the study population was sub-divided into 3 groups. 14 patients (46.67%) patients were in the age group of 1-5 yrs, 9 patients (30 %) were < 1 yr and 7 patients (23.33 %) were in the age group of 5-11 yrs. In our study, 18 (60 %) patients were term babies and 12 (40 %) were preterm. Our study demonstrated that preterm patients had predominant periventricular leucomalacia type of pattern of involvement. Areas of fluid signal intensity in the periventricular white matter adjacent to frontal horn of right lateral ventricle- suggestive of periventricular leucomalacia (Figure 1). Progressive necrosis results in loss of periventricular white matter, passive ventriculomegaly (revealing irregular margins of the bodies and trigones of lateral ventricles). Our study also revealed the term infants had significant involvement of subcortical and/or periventricular white matter and involvement of basal ganglia/thalamic region. Areas of subcortical and periventricular white mater T2WI/FLAIR hyperintensities are seen in bilateral frontal and parietal lobes (Figure 3). Areas of altered signal intensity in bilateral temporal lobes and bilateral basal ganglia with gyriform pattern of hyperintensity in both cerebral hemispheres. These areas reveal restricted diffusion on DWI (Figure 4). Term infant revealing areas of cortical atrophy and multicystic encephalomalacia in bilateral frontal and parietal lobes (Figure 5). The risk factors for HIE were neonatal, maternal, placental and unknown risk factors. The neonatal risk factors (like low birth weight) were found in 17 (56.66 %) patients, maternal causes (like nulliparity, short stature, previous caesarean delivery and overweight) were found in 10 (33.33 %) patients, placental causes (like placenta previa and abruptio placenta) found in 3 (10 %) patients and 5 (16.6 %) patients revealed no significant risk factor. APGAR score at 5 minutes found to be <3 in 5 (16.66 %) patients, 4-7 in 12 (40 %) patients and >7 in 13 patients (43.34 %).

Table 7. Imaging Findings In Term Neonates

Findings	No of patients	Percentage (%)
White matter (subcortical and/or periventricular) hyperintensities	7	38.88
T2/FLAIR hyper intensities in bilateral thalami and basal ganglia	4	22.22
Encephalomalacia in bilateral cerebral hemispheres	5	27.77
Acute infarcts in cerebral hemispheres	1	5.55
Cerebral atrophy	3	16.66
Periventricular leucomalacia	2	11.11

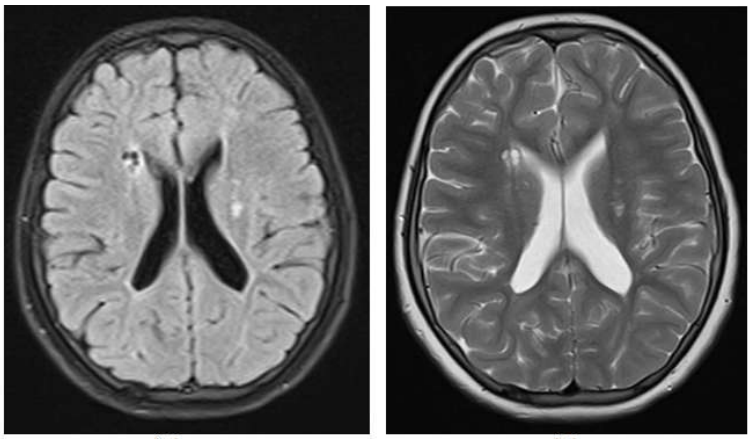

Figure 1: Periventricular leucomalacia – classic pattern of hypoxic-ischemic encephalopathy in preterm infants/children (A) FLAIR, axial (B) T2WI, axia

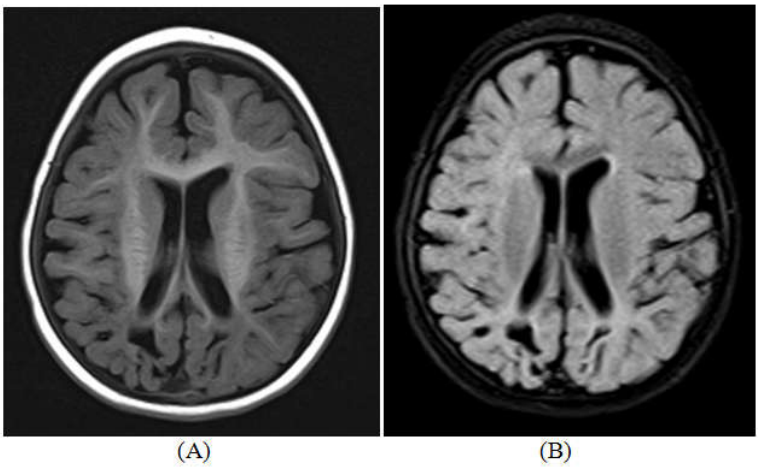

Figure 2. Periventricular leucomalacia with cystic encephalomalacia (A) T1WI, axial (B) FLAIR, axial

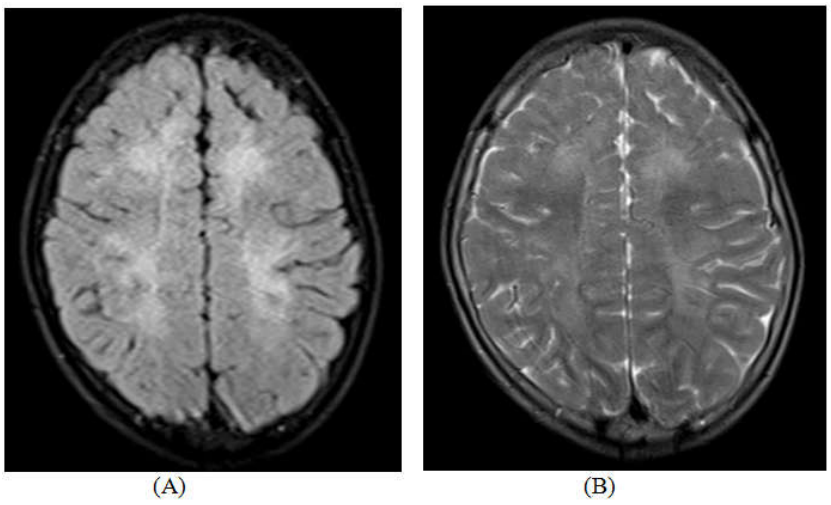

Figure 3. Sub-cortical and periventricular hyperintensities (A) T2 FLAIR, axial (B) T2WI axial

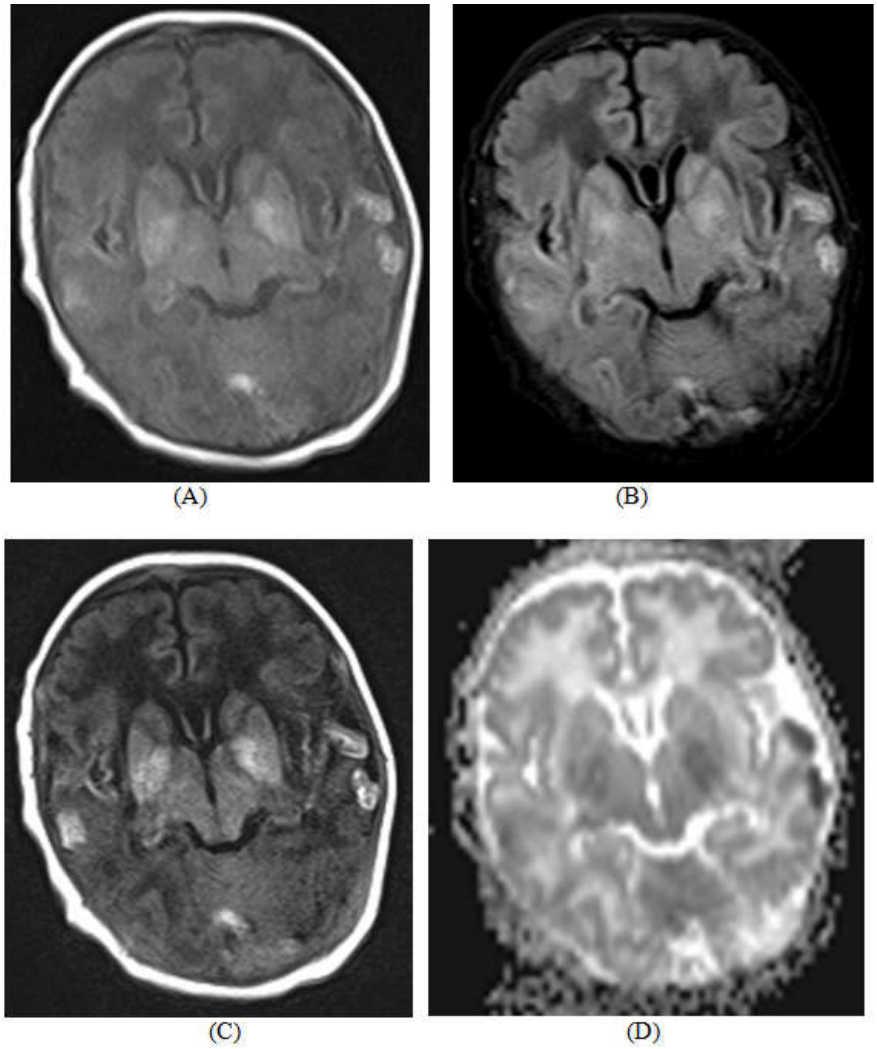

Figure 4. Hypoxic ischemic pattern in term infants (A) T1 FLAIR, axial (B) T2 FLAIR, axial (C) DWI sequence (D) ADC sequence

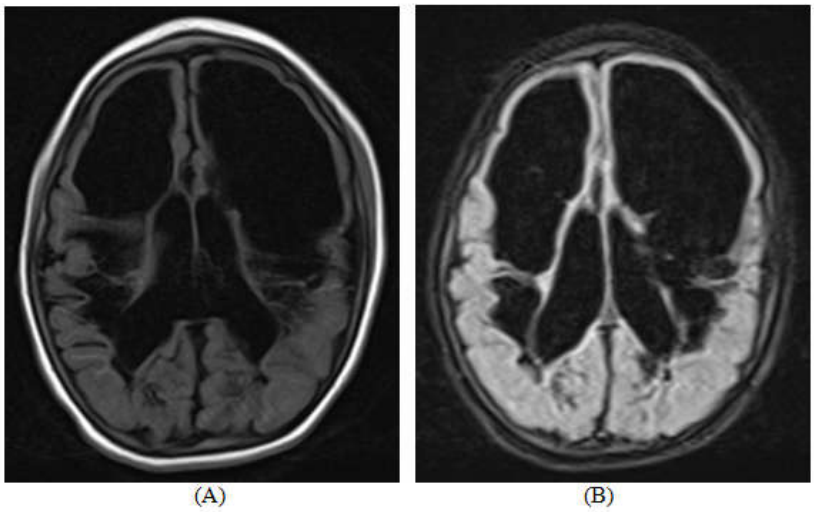

Figure 5. Multicystic encephalopathy (A) T1 FLAIR, axial (B) T2 FLAIR, axial

DISCUSSION

Perinatal hypoxia can results in neonatal hypoxic–ischemic encephalopathy with serious long-term neurological problems. The pattern of brain injury depends on brain maturity at the time of hypoxic insult, severity/duration of insult and severity of hypoxia.

Time of imaging after the hypoxic event also influences the imaging findings. MRI of the brain is the modality of choice for the evaluation of suspected cases hypoxic–ischemic injury (Barkovich, 2000). The pattern of hypoxic ischemic encephalopathy may differ in preterm (<36 weeks of gestation) and term neonates (>36 weeks of gestation). However; at times overlapping features may exist (Barkovich, 2005).

Basically; there are main three MRI patterns of hypoxic-ischemic encephalopathy.

- Periventricular leucomalacia– PVL
- Basal ganglia and/or thalamus lesions – BGTL
- Multicystic encephalopathy – MCE accompanied by injury to the basal ganglia, thalamus and/or cerebral cortex (Truwit, 1990).

In preterm neonates with immature brain, periventricular white matter (supplied by ventriculopetal penetrating arteries) is most susceptible to the hypoxic injury. Mild to moderate hypoxic ischemic injury results in PVL, which may be focal (adjacent to frontal horns and trigones) or diffuse. Progressive necrosis results in loss of periventricular white matter, passive ventriculomegaly (revealing irregular margins of the bodies and trigones of lateral ventricles) (Truwit, 1990; Valk *et al*.,2005; Martin, 1995; Sie *et al*., 2000). Cyst formation and cavitation is suggestive of end-stage PVL which is best demonstrated on FLAIR sequence Periventricular cysts are usually small (≤2–3 mm) while larger cysts carries bad prognosis. DWI is the best sequence for early detection of PVL before any abnormality appears on conventional MRI. Germinal matrix hemorrhage (GMH) is classically seen in preterm infants with hypoxic ischemic injury. Based on severity, GMH can be graded into subependymal hemorrhage (Grade 1), intraventicular hemorrhage without ventricular dilatation (Grade 2), intraventicular hemorrhage with ventricular dilatation (Grade 3) and parenchymal extension of the bleed with coexisting venous infarction (Grade 4). Gradient recalled echo-T2-weighted sequences are sensitive to detect GMH. Coexisting germinal matrix hemorrhage and periventricular white matter injury may be seen. Thalami, brainstem, and cerebellum in the immature brain have high metabolic activity, hence are more susceptible to injury in severe hypoxic ischemic injury (Sie *et al*., 2000; Heinz, 2009). Severe hypoxic-ischemic insult causes injury to metabolically active tissues such as ventrolateral thalami, posterior putamina, hippocampi, brainstem, corticospinal tracts, and sensory-motor cortex. BG injury is more common than parasagittal pattern and carries the bad prognosis. DWI and proton MRS demonstrate basal ganglia/thalamus injury earlier than conventional MRI or other imaging modality.

MR scan of term infants with chronic HIE may reveal cortical atrophy and multicystic encephalomalacia (Sie *et al*., 2000; Heinz, 2009). Prospective study performed by Harshad (2017) on 101 term/preterm patients revealed preterm infants had predominant periventricular leucomalacia type of involvement. Our study also revealed the similar results. According to study performed by Dr Harshad the term infants had predominant basal ganglia and thalamus type pattern of involvement. Our study showed significant involvement of subcortical and/or periventricular white matter and basal ganglia/thalamus region in case of term infants. Study performed by Astra Cabaj *et al* (Astra Cabaj *et al*., 2012) revealed periventricular leucomalacia as a chief pattern of involvement in preterm infants and symmetrical/asymmetrical subcortical white matter hyperintensities or basal ganglia/thalamus as chief pattern of involvement in term infants. Our study also revealed similar findings. The study by Astra Cabaj *et al* also showed that, neonatal causes were present in 53 (52.5%) patients, maternal causes were seen in 22(21.8%) patients, no significant risk factors were found in 20(19.8%) patients and placental causes were found in 6(5.9%) patients.

Our study revealed neonatal causes in 56.66 % patients, maternal causes in 33.33 % patients, placental factors in 10 % cases and no significant risk factors in 16.66 % of patients. Study performed by Lena Liljestrom *et al*. (2016) shown nulliparity, short stature, previous caesarean delivery, overweight, gestational age, birth weight and occiput posterior presentation were all independently associated with hypoxic ischemic encephalopathy. The risk of HIE is increased with decreasing maternal height and increasing body mass index. Our study also showed similar causative maternal factors responsible for HIE. Prospective study of 45 neonates/children with history of premature birth and perinatal hypoxia performed by Alam (2010) revealed that MR imaging could identify early and late stages of ischemic infarction in periventricular white matter. In the clinical background of premature birth and perinatal hypoxia PVL is considered specific. Our study correlated well with the findings of the above mentioned study. Review of article published by Sonia K Ghei *et al*. (2014) revealed in term infants with relatively short duration of hypoxia (<10 mins) are common and usually have no major neurologic consequences. However; hypoxic-ischemic insults for long duration are often associated with clinical and imaging consequences. Mild to moderate hypotension or partial hypoxia for prolonged duration results in peripheral pattern (parasagittal or watershed pattern) of involvement. The basal ganglia–thalamus pattern is relatively less common and occurs in case of profound hypotension or severe hypoxia. As these injuries evolve, cortical thinning and reduction of the underlying white matter are seen in the affected areas. There is resultant ex-vacuo dilatation adjacent lateral ventricles; predominantly in the trigones and occipital horns.

Conclusion

MRI of the brain is the modality of choice for the diagnosis and follow up of infants/children suspected/diagnosed with hypoxic-ischemic encephalopathy (HIE). The pattern of brain injury depends on brain maturity at the time of hypoxic insult, severity/duration of insult and severity of hypotension.

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