ISSN: 2581 – 3633

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at: *https://www.ijmacr.com*

Volume – 9, Issue – 4, August – 2026, Page No.: 560 – 564

Role of MRI in Evaluation of Hypoxic Ischemic Encephalopathy (HIE)

[1]Dr. Aditya Singh, Resident, Department of Radiodiagnosis, S.P. Medical College and Associated Group of Hospitals, Bikaner.

[2]Dr. GL Meena, Senior Professor, Department of Radiodiagnosis, S.P. Medical college and Associated Group of Hospitals, Bikaner.

[3]Dr. Banwari Lal Nayak, Professor, Department of Radiodiagnosis, S.P. Medical college and Associated Group of Hospitals, Bikaner.

[4]Dr. Sant Kumar, Resident, Department of Radiodiagnosis, S.P. Medical College and Associated Group of Hospitals, Bikaner.

[5]Dr. Keerti Singh, Resident, Department of Radiodiagnosis, S.P. Medical College and Associated Group of Hospitals, Bikaner.

Corresponding Author: Dr. Aditya Singh, Resident, Department of Radio-diagnosis, S.P. Medical College and Associated Group of Hospitals, Bikaner.

How to citation this article: Dr. Aditya Singh, Dr. GL Meena, Dr. Banwari Lal Nayak, Dr. Sant Kumar, Dr. Keerti Singh, "Role of MRI in Evaluation of Hypoxic Ischemic Encephalopathy (HIE)", IJMACR – August – 2026, Volume – 9, Issue – 4, P. No. 560 – 564.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Hypoxic-ischemic encephalopathy (HIE) is one of the most common causes of cerebral palsy (CP) and other severe neurological deficits in children. It is caused by inadequate blood flow and oxygen supply to the brain resulting in focal or diffuse brain injury.

Methods: Cross sectional Study was carried out under the department of Radio diagnosis at SP Medical College & Hospital, Bikaner on Infants with clinically diagnosed hypoxic-ischemic encephalopathy (HIE) undergoing MRI.

Results: We found that the sensitivity and specificity of MRI in the diagnosis of HIE is 94%. The negative and positive predictive values were 91% and 96%, respectively. The diagnostic accuracy of MRI is calculated to be around 95%, which makes MRI the best imaging modality for diagnosing hypoxic-ischemic encephalopathy.

Conclusion: In this study, Sarnath staging of HIE is a good predictor in identifying poor long-term neurological outcomes, and it also correlates with the findings of MRI. MRI has high sensitivity and specificity in the evaluation

of hypoxic ischemic encephalopathy. It is non-invasive and has no radiation hazards. It offers excellent grey-white matter resolution, which the other modalities cannot. MRI should be done in all patients with suspected HIE, thus helping in early identification and initiation of therapeutic hypothermia, thereby preventing the long-term complication of brain injury.

Keywords: HIE, MRI, CP

Introduction

Hypoxic ischemic encephalopathy (HIE) is a clinically significant neurological disorder that arises due to impaired oxygenation and perfusion of the neonatal brain, typically occurring in the perinatal or early postnatal period[1]. This condition continues to be a major source of mortality and long-term morbidity among infants, often resulting in cerebral palsy, cognitive impairment, and epilepsy[2]. The burden of HIE is not uniform worldwide; its incidence ranges from 1–2 per 1,000 live births in high-resource countries to as high as 26–40 per 1,000 live births in low-resource settings[3]. Such disparities highlight global inequities in perinatal care and underscore the critical need for timely recognition and management of neonatal asphyxia and its sequelae[4].

The pathogenesis of HIE involves a number of overlapping mechanisms. These include primary energy failure caused by diminished oxygen and glucose delivery to the brain, a cascade of excitotoxic, oxidative, and inflammatory responses, and delayed cell death pathways, including both necrosis and apoptosis[5]. The pattern and severity of brain injury following a hypoxic-ischemic insult are influenced by gestational age, the nature and duration of the insult, and the timing of clinical intervention[6]. Term infants are especially prone to injuries involving the basal ganglia, thalami, and perirolandic cortex following acute profound insults, while prolonged partial asphyxia often results in watershed cortical injury. Conversely, preterm infants are more likely to develop peri ventricular white matter injury[7].

Magnetic resonance imaging (MRI) has emerged as the gold standard for the assessment of HIE due to its ability to detect subtle structural and metabolic changes in the infant brain with high sensitivity and specificity[8]. Conventional MRI sequences, including T1- and T2-weighted imaging, allow for visualization of evolving cerebral edema, patterns of myelination, and chronic sequelae such as cystic encephalomalacia and gliosis[9]. Diffusion-weighted imaging (DWI) provides exceptional sensitivity for acute cytotoxic edema, often detecting injury within hours of the hypoxic-ischemic insult[10]. On the other hand, MR spectroscopy (MRS) enables metabolic assessment, with alterations such as increased lactate and reduced N-acetylaspartate serving as biomarkers of neuronal injury[11].

The timing of MRI is a crucial consideration in HIE evaluation. DWI changes typically emerge within the first few hours after insult and may normalize ("pseudo normalize") after 7–10 days, potentially leading to underestimation of injury when imaging is delayed[12]. T1 and T2 abnormalities often become more conspicuous during the subacute phase, persisting as chronic markers of injury. Thus, a well-timed MRI study between days 3 and 7 after the hypoxic ischemic event is considered optimal for diagnostic accuracy[9].

Hypoxic-ischemic encephalopathy remains one of the leading causes of neonatal mortality and long-term neuro developmental disability in infants. Although MRI has been established as the most sensitive tool for detecting both acute and chronic brain injuries, the spectrum and distribution of MRI findings can vary with timing of imaging, severity of insult, and local patient populations.

In many centers, especially in low- and middle-income countries, systematic documentation of MRI patterns and their correlation with stages of HIE is limited. Generating local data on the radiological profiles of HIE will not only enrich clinical understanding but also assist clinicians in prognostication, parental counseling, and planning long-term rehabilitation strategies. Hence, this study is needed to describe the role of MRI in evaluating both acute and chronic forms of HIE in infants.

Material and Methods

- **Study type**: Descriptive type of observational study
- **Study design**: Cross sectional Study
- **Study location**: This study was carried out under the department of Radio diagnosis at SP
- Medical College & Hospital, Bikaner
- **Study population**: Infants with clinically diagnosed hypoxic-ischemic encephalopathy (HIE) undergoing MRI

Inclusion Criteria

1. **Age:** Neonates and infants from birth to 12 months of age.
2. **Clinical suspicion or diagnosis of HIE:** History of perinatal asphyxia in neonates or a documented hypoxic-ischemic event in infants (Sarnat stage I–III). with associated neurological abnormalities suggestive of hypoxic-ischemic encephalopathy (e.g. seizures, altered sensorium, hypotonia, poor feeding).
3. **MRI evaluation:** Infants undergoing MRI brain as part of routine clinical evaluation for suspected or confirmed HIE within a defined period following the hypoxic-ischemic event.
4. **Clinical stability:** Hemodynamic ally stable and fit to undergo MRI, as certified by the treating pediatrician/ neonatologist.
5. **MRI safety:** No contraindications to MRI.
6. **Ethical consent:** Written informed consent obtained from parents or legal guardians as per IEC/ICMR guidelines, with assurance of confidentiality and right to withdraw.

Exclusion Criteria

1. Major congenital brain malformations (e.g., lissencephaly, holoprosencephaly) or known chromosomal/syndromic disorders affecting brain development.
2. Confirmed CNS infection (e.g., TORCH, meningitis/ encephalitis) or inborn errors of metabolism/metabolic encephalopathies unrelated to perinatal hypoxia-ischemia.
3. Primary intracranial pathology unrelated to HIE, such as significant traumatic brain injury or large vascular malformations.
4. MRI contraindications: non - MRI compatible implants/ devices, severe cardio respiratory instability precluding safe imaging, or inability to monitor safely during the scan.
5. Prior intracranial surgery before the index MRI.
6. Parental refusal of consent or withdrawal at any time.
7. Non-diagnostic MRI (e.g., severe motion artifact or incomplete core sequences) — exclude from final analysis; record separately for accountability.

Statistical analysis

Data thus collected was entered in Microsoft Excel Sheet by investigator herself on same day so as to minimize data entry bias if any.

1. Linear variables were summarised as mean and standard deviation whereas nominal/categorical variables was presented as proportions (percentage). Ordinal variables were described as median and inter quartile range.

2. Unpaired T test, one way ANOVA test and other parametric tests was used for analysis of linear variables while nominal/categorical variables was analysed by using chi square test and fisher exact test. Man Whitney U test and Kruskal Wali′s tests was used for ordinal variables.
3. P value <0.05 was taken as significant.
4. SPSS 26.0 version software was used for all statistical calculations.

Results

Table 1: Distribution of study parameters

Age group In days	< 28 days	16
	1-3 months	39
	>3 months	45
Gender	Male	61
	Female	39
Birth weight in kg		7.15 ± 2.36 kg
HIE (I:II:III)		26 : 51 : 23
MRI Severity (Normal: Mild: Moderate: Severe)		12: 18 : 44 : 26

Table 2: Cross tabulation clinical staging and MRI findings

Clinical stage	Normal	Mild	Moderate	Severe	p-value
I	11	13	1	1	0.001
II	1	3	31	16	
III	0	2	12	9	

We found that the sensitivity and specificity of MRI in the diagnosis of HIE is 94%. The negative and positive predictive values were 91% and 96%, respectively. The diagnostic accuracy of MRI is calculated to be around 95%, which makes MRI the best imaging modality for diagnosing hypoxic-ischemic encephalopathy.

Discussion

This study evaluated the various MRI findings in suspected Hypoxic Ischemic Encephalopathy and compared the different MRI patterns with clinical outcomes. In addition, we also set out to demonstrate the sensitivity of MRI in evaluating birth asphyxia babies. In a study conducted by Robertson et al.,[12], they concluded that 100% of babies with stage I HIE had normal neurodevelopmental outcomes at 3.5 years. For babies with stage II HIE, 71% had normal outcomes, and for stage III HIE babies 0% had normal outcomes at 3.5 years.

In a study conducted by Biarge et al. [13] with 175 term babies with birth asphyxia, they found that the severity of BGT lesions was strongly associated with the severity of motor Impairment. These findings were also correlated with the study conducted by Volpe et al.[9] He stated that among a group of infants with presumed hypoxic-ischemic encephalopathy, the 25% with predominantly basal ganglia-thalamic injury had a poorer outcome than the 45% with a predominantly para sagittal watershed pattern or the 30% with a normal neonatal MRI.

Conclusion

In this study, Sarnath staging of HIE is a good predictor in identifying poor long-term neurological outcomes, and it also correlates with the findings of MRI. MRI has high sensitivity and specificity in the evaluation of hypoxic ischemic encephalopathy. It is non-invasive and has no radiation hazards. It offers excellent grey-white matter resolution, which the other modalities cannot. MRI should be done in all patients with suspected HIE, thus helping in early identification and initiation of therapeutic hypothermia, thereby preventing the long-term complication of brain injury.

Reference

1. Volpe JJ. Hypoxic - ischemic encephalopathy: clinical aspects. In: Neurology of the Newborn. 5th ed. Philadelphia: Saunders; 2008.

2. Shankaran S. Hypoxic-ischemic encephalopathy and novel strategies for neuro protection. Clin Perinatol. 2012; 39(4):919–29.

3. Lee AC, Kozuki N, Blencowe H, et al. Intra partum-related neonatal encephalopathy incidence and impairment at regional and global levels: a systematic review. PLoS Med. 2013; 10(7)\:e1001470.

4. Lawn JE, Cousens S, Zupan J. 4 million neonatal deaths: When? Where? Why? Lancet. 2005; 365 (9462):891–900.

5. Ferriero DM. Neonatal brain injury. N Engl J Med. 2004; 351:1985–95.

6. Gunn AJ, Bennet L. Fetal hypoxia insults and patterns of brain injury: insights from animal models. Clin Perinatol. 2009; 36(3):579–93.

7. Volpe JJ. Brain injury in premature infants: a complex amalgam of destructive and developmental disturbances. Lancet Neurol. 2009; 8(1):110–24.

8. Cowan FM, de Vries LS. The internal capsule in neonatal imaging. Semin Fetal Neonatal Med. 2005; 10 (5): 461–74.

9. Barkovich AJ. MR imaging of the neonatal brain. Neuro imaging Clin N Am. 2006; 16(1):117–35.

10. Robertson NJ, Thayyil S. Magnetic resonance spectroscopy in neonatal encephalopathy. Semin Fetal Neonatal Med. 2015; 20 (2):129–35.

11. Robertson C, Finer N. Term infants with hypoxic-ischemic encephalopathy: outcome at 3.5 years. Developmental Medicine & Child Neurology. 1985 Aug; 27(4):473-84.

12. Martinez-Biarge M, Diez-Sebastian J, Kapellou O, Gindner D, Allsop JM, Rutherford MA, Cowan FM. Predicting motor outcome and death in term hypoxic-ischemic encephalopathy. Neurology. 2011 Jun 14; 76 (24):2055-61.