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MAGNETIC RESONANCE IMAGING IN NEONATAL BIRTH ASPHYXIA: A RETROSPECTIVE EVALUATION OF HYPOXIC-ISCHEMIC BRAIN INJURY IN TERM AND PRETERM INFANTS

Radio-Diagnosis

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ABSTRACT

Background: Birth asphyxia is a major cause of neonatal morbidity and mortality, particularly affecting the central nervous system. Magnetic Resonance Imaging (MRI) plays a crucial role in the evaluation of hypoxic-ischemic brain injury by providing detailed anatomical and functional information.**Objective:** To evaluate the role of MRI in detecting the patterns and severity of brain injury in term and preterm neonates with clinical evidence of birth asphyxia.**Methods:** This retrospective study included 15 neonates (8 term, 7 preterm) with clinical and biochemical evidence of hypoxic-ischemic encephalopathy (HIE). MRI brain was performed using T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging (DWI), and MR spectroscopy sequences. Imaging findings were correlated with clinical stage and outcome. **Results:** MRI detected characteristic patterns of injury correlating with gestational age. Term infants predominantly showed basal ganglia-thalamic and perirolandic cortical injury patterns, while preterm infants exhibited periventricular leukomalacia and diffuse white matter injury. Restricted diffusion was noted in acute phases (<7 days), while ssschronic cases demonstrated cystic evolution and atrophy. MRI findings significantly correlated with the severity of encephalopathy and clinical outcome. **Conclusion:** MRI serves as a sensitive and specific modality for detecting, characterizing, and staging hypoxic-ischemic injury in both term and preterm neonates. Early MRI evaluation aids in prognostication and management planning.

KEYWORDS

Birth Asphyxia, Hypoxic-ischemic Encephalopathy, MRI, Neonatal Brain Injury, Diffusion-weighted Imaging, MR Spectroscopy

INTRODUCTION

Birth asphyxia remains a significant cause of neonatal brain injury, contributing to long-term neurodevelopmental disabilities such as cerebral palsy, epilepsy, and cognitive impairment. Despite advances in perinatal care, the incidence of hypoxic-ischemic encephalopathy (HIE) remains approximately 1–3 per 1000 live births in term infants and higher in preterm neonates [1,2].

Magnetic Resonance Imaging (MRI) provides superior soft tissue contrast, enabling precise evaluation of the location, extent, and timing of hypoxic-ischemic injury. Diffusion-weighted imaging (DWI) and MR spectroscopy (MRS) further enhance diagnostic accuracy by detecting early cytotoxic edema and metabolic disturbances [3,4].

This study retrospectively assesses the MRI patterns of hypoxic-ischemic injury in term and preterm neonates with birth asphyxia and correlates imaging findings with clinical outcomes.

Materials and Methods

Study Design: A retrospective observational study was conducted in the Department of Radiodiagnosis, GMC, Chhatrapati Sambhaji Nagar, over a period of 12 months. Institutional ethical clearance was obtained.

Study Population: A total of 15 neonates (8 term, 7 preterm) diagnosed clinically with birth asphyxia or hypoxic-ischemic encephalopathy (HIE) were included.

Inclusion Criteria: Neonates with evidence of perinatal asphyxia (Apgar score ≤6 at 5 minutes, metabolic acidosis, or need for resuscitation); MRI performed within 2–21 days of birth; availability of clinical and biochemical data.

Exclusion Criteria: Neonates with congenital malformations, metabolic disorders, or infections.

MRI Protocol: MRI was performed on a 1.5T system using axial and sagittal T1-weighted, axial and coronal T2-weighted, FLAIR, diffusion-weighted imaging sequences.

Data Analysis: MRI findings were analyzed for the pattern of injury (basal ganglia-thalamic, watershed, or white matter predominant), signal changes, and spectroscopic abnormalities, and correlated with gestational age and severity of HIE.

RESULTS

Demographics

- **Total cases:** 15 (8 term, 7 preterm)
- **Mean gestational age:** 36 ± 3.5 weeks
- **Male:Female ratio:** 9:6
- **MRI timing:** Mean 8.2 days post-birth

Imaging Findings

Pattern	Term (n=8)	Preterm (n=7)
Basal ganglia–thalamic injury	6	2
Perirolandic cortical injury	5	0
Periventricular leukomalacia	1	5
Diffuse white matter injury	2	6
Watershed injury	3	2

- **Restricted diffusion** in acute lesions (<7 days) in 10/15 neonates
- **Lactate peaks** on MR spectroscopy in 11/15 neonates
- **Cystic evolution and atrophy** in 4 chronic-stage infants (>14 days)

Correlation with Outcome: MRI severity correlated significantly with the grade of encephalopathy and 6-month developmental outcome ($p<0.05$).

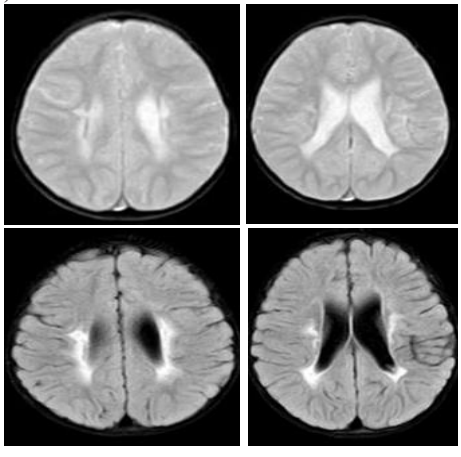

Fig 1: Preterm, low birth weight infant MRI brain showing - perirolandic, periventricular and peritrigonal hyperintensities in T2 WI (a, b) and FLAIR (c, d) images – suggesting hypoxic ischemic encephalopathy.

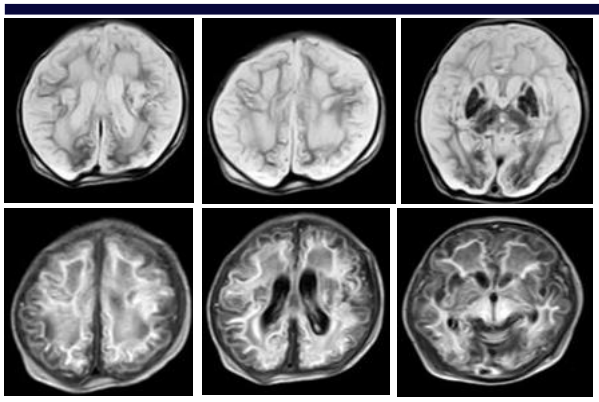

Fig 2 : Term, low birth weight infant MRI brain showing – Symmetrical & severe paucity of white matter with associated abnormal T2 WI (a, b, c) and FLAIR (d, e, f) hyperintensities in bilateral peri rolandic, centrum semiovale and per ventricular regions with peri trigonal white matter predominance & gangliocapsular regions. No e/o restricted diffusion noted on DW images in images – suggesting severe hypoxic ischemic encephalopathy.

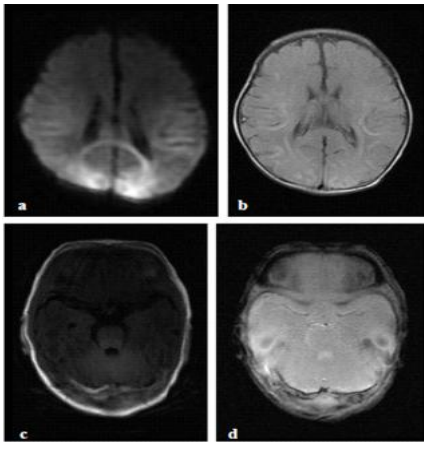

Fig. 3: WATERSHED PATTERN

a. Axial DWI: restricted diffusion in the splenium of the corpus callosum and the occipital white matter.
b. Axial T2 FLAIR: Hyper intensity in the corona radiata of bilateral parieto occipital lobes.
c. Axial T1 FLAIR: T1 hyper intensity in bilateral transverse sinuses.
d. Axial T2*: blooming in bilateral transverse sinuses suggestive of thrombosis.

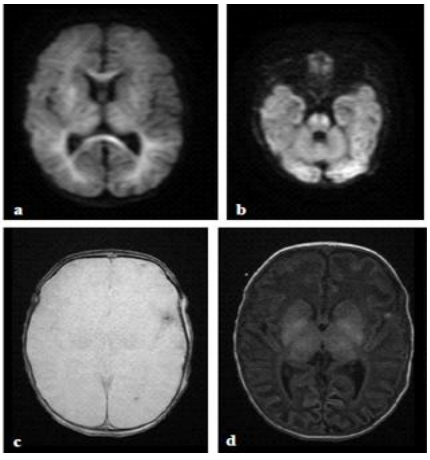

Fig. 4: BASALGANGLIA-THALAMUS PATTERN

a. Axial DWI: restricted diffusion in the basal ganglia, thalami, periventricular white matter and the corpus callosum.
b. Axial DWI: Restricted diffusion in the brainstem along the corticospinal tract.
c. Axial T2*: blooming present in the cortex of the left temporoparietal lobes and the occipital horn of the left lateral ventricle, suggestive of parenchymal and intraventricular hemorrhage, respectively.
d. Axial T1: T1 hyper intensity in the left temporoparietal lobes, suggestive of hemorrhage; cortical highlighting along the occipital lobes, myelination is age appropriate.

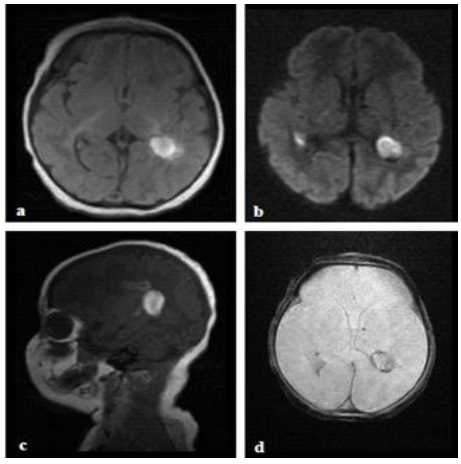

Fig 5: PERIVENTRICULAR PATTERN

a. Axial DWI : rounded lesions showing restricted diffusion in the lateral ventricles and periventricular white matter.
b. Axial T2 FLAIR : T2 FLAIR hyperintense intraventricular lesions.
c. Sagittal T1 FLAIR : T1 hyperintense intraventricular lesions.
d. Axial T2* : the T1 & T2 hyperintense lesions show blooming, suggestive of sub acute intraventricular hemorrhage.

DISCUSSION

MRI effectively delineates both temporal evolution and anatomical distribution of hypoxic ischemic brain injury in neonates. In term neonates, high metabolic activity in the basal ganglia and perirolandic cortex explains the predominant injury in these regions, whereas in preterm neonates, vulnerability of immature white matter leads to periventricular leukomalacia [5,6].

Diffusion-weighted imaging detects early cytotoxic edema before conventional MRI changes appear [7]. MR spectroscopy, with elevated lactate and reduced NAA, indicates anaerobic metabolism and neuronal injury [8].

Our findings align with prior studies that describe gestational age–dependent patterns of HIE [9–11]. Early MRI provides prognostic information and assists in planning therapy such as hypothermia [12].

CONCLUSION

MRI, especially with diffusion and spectroscopic sequences, is a critical tool for detecting and prognosticating hypoxic-ischemic injury in both term and preterm infants. Understanding gestational-age–specific patterns enhances diagnostic accuracy and management.

Ethical Clearance

Approved by Institutional Ethics Committee, Government Medical College, Chhatrapati Sambhajinagar. Parental consent obtained for imaging inclusion.

Conflict of Interest

No conflict of interest declared.

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