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Radio-Diagnosis

MRI PATTERNS IN NEONATAL HYPOXIC ISCHEMIC ENCEPHALOPATHY (HIE) : TERM VERSUS PRETERM : A SINGLE CENTRE DESCRIPTIVE STUDY.

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ABSTRACT **Background:** Hypoxic ischaemic encephalopathy (HIE) remains a major cause of neonatal mortality and long-term disability worldwide [1]. The burden is seen to be higher in socioeconomically disadvantaged countries, including India, where perinatal care challenges and high rates of prematurity persist [2]. Magnetic Resonance Imaging (MRI) plays a critical role in HIE enabling early diagnosis, guiding clinical management, monitoring disease progression, and serving as a robust prognostic tool [3]. Gestational age significantly influences the pattern and severity of brain injury, with term and preterm infants showing distinct MRI findings [4]. **Aim:** To describe and compare MRI patterns and key prognostic signs in term and preterm neonates with HIE. **Methods:** This descriptive study was conducted at our hospital over 5 years, including 30 babies diagnosed clinically with HIE. Participants were stratified into term (≥37 weeks) and preterm (<37 weeks) groups. All suspected babies underwent MRI brain using diffusion-weighted imaging (DWI), T1W, T2W & FLAIR sequences [5]. MRI findings were categorised into basal ganglia thalamic (BGT), watershed, white matter, cortical, and mixed injury patterns [6]. Prognostic signs including posterior limb of internal capsule (PLIC) signal abnormality, BGT lesion grading, and brainstem involvement were recorded and correlated with clinical severity (Sarnat staging) and short-term outcome [7]. **Results:** Term neonates predominantly exhibited BGT and parasagittal cortical injuries, often associated with absent PLIC signal in severe cases [8]. Preterm infants more frequently demonstrated periventricular white matter injury and intraventricular haemorrhage, with relative sparing of deep grey matter [9]. Abnormal PLIC signal and higher BGT grades strongly correlated with severe motor outcomes [10], while brainstem involvement was associated with increased mortality risk [11]. **Conclusion:** MRI is essential in HIE, guiding diagnosis, tracking disease evolution, and predicting outcomes [3]. Distinct term and preterm injury patterns reflect maturational differences [4], while absent PLIC signal and severe BGT injury serve as powerful early prognostic markers [7],[10].

KEYWORDS : Hypoxic ischaemic encephalopathy, MRI brain findings, Neonatal hypoxia, Preterm vs term.

INTRODUCTION

Hypoxic-ischaemic encephalopathy (HIE) is a major cause of neonatal mortality and long-term neurodisability, resulting from impaired cerebral blood flow and oxygen delivery during the perinatal period [1]. Survivors frequently face lifelong neurological sequelae, including cerebral palsy, epilepsy, and cognitive impairment [12], placing a considerable burden on families and healthcare systems [13].

Hypoxic ischaemic encephalopathy (HIE) is a serious neurological condition in the neonatal period, caused by a reduction in cerebral blood flow and oxygenation during the perinatal phase [14]. The spectrum of clinical manifestations ranges from mild weakness to permanent neurological disability including movement disorders, epilepsy, learning difficulties, or death in severe cases [15]. The prevalence of HIE is greater in low and middle income countries, such as India, where prematurity remains common and perinatal care lacks resources [2],[16]. Though both term and preterm infants are affected, the pattern of cerebral injury varies with gestational age, which can be attributed to differences in brain structure, vascular supply, and metabolic vulnerability [4,17].

Magnetic Resonance Imaging (MRI) has come a long way in the evaluation of neonatal HIE [18]. Now, with advances in imaging protocols, it is not just limited to diagnosing the condition; detailed mapping of the affected brain regions enables monitoring of lesion evolution and treatment planning [3,19]. Subtle changes can be detected, particularly with diffusion-weighted sequences [20].

In term neonates, injury to basal ganglia thalamic (BGT) regions and perirolandic cortex following acute profound hypoxia are common [21], while preterm infants exhibit periventricular white matter injury and haemorrhagic lesions [22].Certain imaging signs, such as loss of the normal high signal in the posterior limb of the internal capsule (PLIC) or extensive BGT/midbrain involvement, have been linked with poor motor outcomes and higher risk of cerebral palsy [7,10,23].

In this study, we aim to map the MRI patterns of brain injury in both term and preterm babies with HIE, with special mention to signs that can help predict future outcomes.

Review of Literature

The understanding of brain injury patterns in neonatal hypoxic-ischaemic encephalopathy (HIE) has grown steadily over the last three decades.

In 1995, Barkovich et al. was the first to show that MRI could link cerebral injury patterns to both type and timing of the insult [24]. This work described two hallmark MRI patterns in term babies: injury to the basal ganglia and thalamus in cases of sudden, severe hypoxia, and watershed cortical injury in situations of prolonged hypoxia.

In 1998, Rutherford et al. worked further on this and identified the role of MRI in prognostication [7]. They described two signs: absence of the normal posterior limb of the internal capsule (PLIC) signal as a powerful early indicator of later spastic cerebral palsy, and that severe basal ganglia thalamic (BGT) injury was proportional to motor disability.

In 2005, Miller et al. highlighted the impact of gestational age on cerebral injury patterns [4]. Their results showed that preterm infants more often sustain periventricular white matter injury, while term infants tend to have more cortical and deep grey matter involvement.

In 2012, the NICHD trial by Shankaran et al. described structured MRI scoring systems and advocated the use of MRI within the first two weeks, strongly predicting neurodevelopmental outcomes at 18-25 months [25].

In 2013, Ancora et al. described how diffusion-weighted imaging (DWI) could detect brain injury within 24-48 hours after the hypoxic–ischaemic event, much earlier than conventional MRI sequences [20].

In 2020, Lally et al. examined MRI findings in preterm infants undergoing therapeutic hypothermia and reported a higher frequency of white matter injury and intracranial haemorrhage compared with term infants, underscoring the need for gestation-specific interpretation in the cooling era [26].

In 2021, Weeke et al. studied multicentre cohorts and confirmed basal ganglia-thalamic predominance in term HIE and white matter/watershed involvement in preterm HIE [21].

In 2023, Wu et al. described scoring systems derived from term cohorts

and concluded they cannot be directly applied to preterm infants, as this could overestimate the severity of cerebral injury [27].

MATERIALS AND METHODS

This was a single-centre, descriptive retrospective study conducted in our radiology department over a defined study period of 5 years. We have taken newborns diagnosed clinically with hypoxic–ischaemic encephalopathy (HIE), divided them into term and preterm groups based on gestational age at the time of birth.

Inclusion Criteria

We included neonates who had a clear clinical diagnosis of HIE. underwent MRI for the same.

Exclusion Criteria

Babies with conditions that mimic hypoxic ischaemic injury patterns. These are major congenital brain malformations, chromosomal abnormalities, or metabolic disorders.

MRI Protocol

Machine used: 1.5 Tesla Siemens Magnetom Avanto MRI machine.

Sequences included conventional T1 weighted, T2 weighted, and FLAIR imaging, along with diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) maps.

Pattern Classification

Two experienced radiologists independently reviewed the scans.

Injury patterns were classified as:

- Basal ganglia thalamic (BGT) injury
- Watershed cortical injury
- Periventricular white matter injury
- Mixed patterns

For prognostic analysis, we specifically looked for:

- Absence of the normal bright signal in the posterior limb of the internal capsule (PLIC) on T1-weighted images.
- Severe BGT involvement on T1/T2/DWI sequences.
- Midbrain involvement
- Outcome Assessment
- Clinical outcomes were recorded at discharge and during follow-up visits including mortality, presence of cerebral palsy, and developmental milestones.

Statistical Analysis

Data were entered into a spreadsheet and analysed using appropriate statistical software. Categorical variables were compared between term and preterm groups using Chi-square or Fisher's exact tests, and continuous variables were compared with t-tests or Mann Whitney U tests as applicable.

RESULTS

A total of 30 patients were included, comprising 20 term infants (67%) and 10 preterm infants (33%).Most of the cases underwent beyond one year of age, on chronic MRI scans performed beyond one year of age, the most frequent injury pattern was mixed involvement (53%), followed by periventricular white matter injury (23%), basal ganglia thalamic (BGT) involvement (13%), and watershed cortical injury (10%).

When stratified by gestational age, mixed patterns were common in both term and preterm groups. Periventricular white matter injury was seen only in term infants (7/20), while BGT-only involvement was also confined to term infants (4/20). Watershed-only patterns were seen exclusively among preterm infants (3/10).

On diffusion-weighted imaging (DWI), true diffusion restriction was observed in 17% of cases, while a further 17% showed T2 shine-through. Ulegyria was identified in 3 term infants with MRI scans beyond one year. Brainstem involvement was uncommon, seen in only 10% of cases.

Acute phase prognostic markers such as posterior limb of the internal capsule (PLIC) hyperintensity could not be assessed in most of these chronic scans. It was present in 1 case which gotten MRI brain within 2 weeks of life.

Cerebral palsy (CP) (6%) was present across all injury patterns, most frequently in patients with mixed involvement, followed by periventricular white matter injury and BGT patterns. Other common long term outcome in our study is developmental delay(20%) which did not show any predilection to specific pattern.

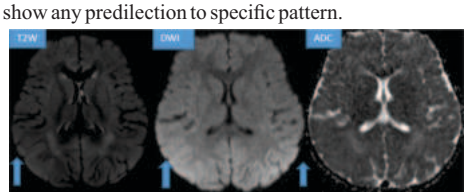

Figure 1: Periventricular hyperintensities on T2W & FLAIR showing no diffusion restriction in a case hypoxic ischaemic encephalopathy in a term infant.

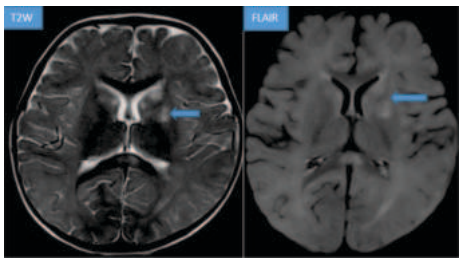

Figure 2: Bilateral basal ganglia hyperintensities on T2W/FLAIR sequences in a case hypoxic ischaemic encephalopathy in a term infant.

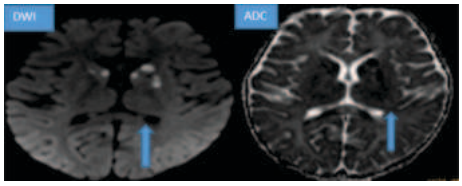

Figure 3: Bilateral basal ganglia hyperintensities showing restricted diffusion on DWI/ADC sequences in a case hypoxic ischaemic encephalopathy in a term infant.

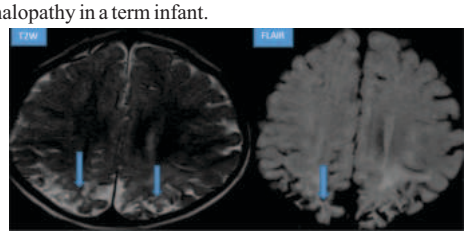

Figure 4: Mushroom shaped gyria (Ulegyria) in a case of chronic hypoxic ischaemic encephalopathy in a term infant.

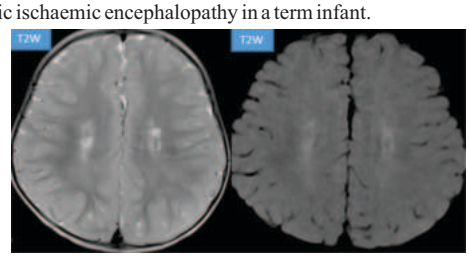

Figure 5: Watershed involvement in a case hypoxic ischaemic encephalopathy in a preterm infant

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