E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.287

Case Series

MR Imaging of Brain in a Paediatric with Hypoxic Ischemic Encephalopathy, a Retrospective Study in Tertiary Care Center

Dr. Navya N.[1*], Dr. Tamsir Rongpipi[2], Dr. Anuja Saikia[3], Dr. Darpana Kalita[4], (Prof) Dr. Rupak Bhuyan[5]

[1*]Postgraduate Trainee, Department of Radiodiagnosis, Jorhat Medical College and Hospital, Jorhat, Assam, India.

[2]Assistant Professor, Department of Radiodiagnosis, Jorhat Medical College and Hospital, Jorhat, Assam, India.

[3]Assistant Professor, Department of Radiodiagnosis, Jorhat Medical College and Hospital, Jorhat, Assam, India.

[4]Assistant Professor, Department of Radiodiagnosis, Jorhat Medical College and Hospital, Jorhat, Assam, India.

[5]Professor & HOD, Department of Radiodiagnosis, Jorhat Medical College and Hospital, Jorhat, Assam, India.

Corresponding Author: Dr. Navya N.
Postgraduate Trainee, Department of Radiodiagnosis, Jorhat Medical College and Hospital, Jorhat, Assam, India.

Orcid Id: 0009-0002-6791-9743.

Received: 10.06.2026 Revised: 06.07.2026 Accepted: 05.08.2026 Published: 10.08.2026

ABSTRACT

Background: Hypoxic–ischemic encephalopathy (HIE) remains a major cause of morbidity and long-term neurodevelopmental impairment in children. While basal ganglia and cortical injuries are well described, white matter involvement represents a critical but variable manifestation of hypoxic–ischemic injury, particularly beyond the neonatal period. Magnetic resonance imaging (MRI) plays a pivotal role in detecting and characterizing these white matter changes and in understanding injury patterns related to hypoxia–ischemia.

Methods: This retrospective study included children under 18 years of age diagnosed with hypoxic–ischemic encephalopathy who underwent brain MRI at a tertiary care center over a defined study period. Conventional MRI sequences including T1-weighted, T2-weighted, FLAIR, and diffusion-weighted imaging were reviewed. White matter involvement was assessed for distribution, symmetry, extent, and predominant regions affected, including periventricular, subcortical, and deep white matter. Imaging findings were analysed descriptively and correlated with age at insult and clinical history.

Results: White matter abnormalities were identified in the majority of cases, with periventricular and subcortical white matter being the most frequently involved regions. Diffuse bilateral involvement was more common than focal changes. Younger children demonstrated a higher prevalence of periventricular white matter injury, while older children showed more extensive subcortical and deep white matter involvement. Associated cortical and deep gray matter abnormalities were observed in a subset of patients.

Conclusion: MRI demonstrates a broad spectrum of white matter changes in Paediatric hypoxic–ischemic encephalopathy, with patterns varying according to age and severity of insult. Recognition of these imaging patterns is essential for accurate diagnosis, prognostication, and clinical management. White matter assessment should be an integral component of MRI evaluation in children with suspected hypoxic–ischemic brain injury.

Keywords: Hypoxic–Ischemic Encephalopathy, Paediatric Mri, White Matter Injury, Periventricular Leukomalacia, Hypoxia, Ischemia.

INTRODUCTION

Hypoxic–ischemic encephalopathy (HIE) is a significant cause of acquired brain injury in children and remains a leading contributor to mortality and long-term neurological morbidity worldwide. It results from a reduction in

cerebral blood flow and oxygen delivery to the brain, leading to a cascade of metabolic failure, excitotoxicity, oxidative stress, inflammation, and subsequent neuronal and glial injury. The extent and pattern of brain damage depend on the severity, duration, and timing of the hypoxic–ischemic insult, as well as the developmental stage of the brain at the time of injury.[1]

While deep gray matter structures such as the basal ganglia and thalami are classically associated with severe hypoxic–ischemic injury, white matter involvement represents a crucial and often under-recognized component of paediatric HIE. The immature white matter is particularly vulnerable to hypoxia–ischemia due to high metabolic demands, selective vulnerability of oligodendrocyte precursors, and incomplete myelination.[2] In neonates and young children, this vulnerability commonly manifests as periventricular white matter injury, whereas older children may demonstrate more diffuse subcortical and deep white matter involvement.[3]

Magnetic resonance imaging (MRI) is the imaging modality of choice for evaluating hypoxic–ischemic brain injury in children. MRI provides superior contrast resolution and multiplanar capability, allowing detailed assessment of white matter integrity and associated gray matter abnormalities. Conventional sequences such as T1-weighted, T2-weighted, and fluid-attenuated inversion recovery (FLAIR) images are valuable for detecting chronic white matter signal changes, volume loss, and gliosis, while diffusion-weighted imaging is particularly sensitive in the early stages of injury.[4]

White matter changes in HIE have important prognostic implications, as they are closely linked to long-term neurodevelopmental outcomes, including motor impairment, cognitive delay, and epilepsy. Despite this, most imaging-based studies have focused primarily on neonatal HIE or on deep gray matter injury patterns, with relatively fewer studies dedicated to systematic evaluation of white matter abnormalities across the broader Paediatric age group.[5] Furthermore, the spectrum and distribution of white matter injury in post-neonatal hypoxic–ischemic insults remain less clearly defined.

The present retrospective study aims to analyze the MRI patterns of white matter changes in children with hypoxic–ischemic encephalopathy, emphasizing their distribution and associated imaging findings. A better understanding of these patterns may aid in accurate diagnosis, improved prognostication, and optimized clinical management of Paediatric patients with hypoxic–ischemic brain injury.

Aims and Objectives

Aim

To evaluate the magnetic resonance imaging (MRI) patterns of brain in children with hypoxic–ischemic encephalopathy and to characterize the distribution and associated intracranial abnormalities in a retrospective cohort.

Objectives

Primary Objective

1. To assess the pattern and distribution of white matter abnormalities on MRI in children diagnosed with hypoxic–ischemic encephalopathy.

Secondary Objectives

1. To identify the most commonly affected white matter regions (periventricular, subcortical, and deep white matter).
2. To describe the MRI characteristics of white matter injury on T1-weighted, T2-weighted, FLAIR, and diffusion-weighted imaging (DWI) sequences.
3. To evaluate associated MRI findings, including cortical injury, basal ganglia and thalamic involvement, cerebral atrophy, ventricular dilatation, and cystic changes.
4. To compare the pattern of white matter injury across different Paediatric age groups.
5. To correlate MRI findings with the clinical history and timing of hypoxic–ischemic insult, where available.
6. To highlight the role of MRI in the diagnosis and assessment of Paediatric hypoxic–ischemic encephalopathy.

MATERIALS AND METHODS

Study Design and Population

The Retrospective observational study was conducted in the 'Department of Radiodiagnosis and Imaging Sciences, Jorhat Medical College and Hospital, Jorhat Assam for a period of 1 year from November 2024 to November 2025. Medical records and imaging data of children younger than 18 years diagnosed clinically with hypoxic–ischemic encephalopathy were reviewed over this study period. A total of 22 cases of either gender were included in the study who underwent MRI brain in 1.5 Tesla

General Electric (GE) MRI scanner in supine position with the coil.

Inclusion Criteria

1. Children aged 0–18 years.
2. Patients with a clinical diagnosis of hypoxic–ischemic encephalopathy (HIE) based on history, clinical examination, and treating physician's assessment.
3. Patients who underwent brain MRI at the study institution during the study period.
4. MRI examinations that included standard brain sequences such as T1-weighted, T2-weighted, FLAIR, and diffusion-weighted imaging (DWI)/ADC.
5. MRI studies of adequate image quality for assessment of white matter abnormalities.
6. Patients with complete clinical records and MRI data available for retrospective review.

Exclusion Criteria

1. Patients older than 18 years.
2. Patients with congenital brain malformations (e.g., lissencephaly, schizencephaly, holoprosencephaly).
3. Patients with inherited metabolic or neurodegenerative disorders known to affect cerebral white matter (e.g., leukodystrophies, mitochondrial disorders).
4. Patients with central nervous system infections such as meningitis or encephalitis.
5. Patients with traumatic brain injury as the primary cause of neurological deficits.
6. Patients with intracranial tumors or other structural brain lesions unrelated to hypoxic–ischemic injury.
7. MRI examinations with significant motion artifacts, incomplete imaging sequences, or poor image quality that precluded evaluation.
8. Patients with incomplete clinical information or unavailable imaging records.

Clinical Data Collection

Demographic data including age and sex were recorded. Clinical history was reviewed to identify the presumed hypoxic–ischemic event, such as perinatal asphyxia, respiratory failure, cardiac arrest, drowning, or severe hypotension. Available neurological findings at presentation and follow-up were documented from medical records.

MRI Acquisition Protocol

All MRI examinations were performed on a 1.5-T scanner using a standardized Paediatric brain imaging protocol.

Imaging protocol

- Axial T1-weighted FLAIR
- Axial T2 Propeller
- Coronal and Sagittal T2
- Axial fluid-attenuated inversion recovery (FLAIR)
- Axial diffusion-weighted imaging (DWI) with corresponding axial apparent diffusion coefficient (ADC) maps.
- Axial GRE

Slice thickness and imaging parameters were adjusted according to patient age and scanner specifications. Sedation was administered when necessary under Paediatric anaesthesia supervision.

Image Analysis

MRI studies were independently reviewed by two experienced radiologists blinded to detailed clinical outcomes. Discrepancies were resolved by consensus. White matter changes were assessed for signal intensity alterations, symmetry, and distribution. White matter involvement was categorized into periventricular, subcortical, and deep white matter regions. The extent of involvement was classified as focal or diffuse and unilateral or bilateral. Associated findings such as cortical injury, deep gray matter involvement, volume loss, cystic changes, and gliosis were also documented.

Timing of MRI and Injury Pattern Assessment

MRI timing in relation to the hypoxic–ischemic event was noted and classified as early (<7 days), subacute (7–21 days), or chronic (>21 days). Imaging features were interpreted in relation to the timing of injury to distinguish acute diffusion abnormalities from chronic white matter signal changes and atrophy.

Statistical Analysis

Data were compiled using a standardized data collection sheet. Descriptive statistics were used to summarize demographic variables and imaging findings. Categorical variables were expressed as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation. Statistical analysis was performed using standard statistical software. A p-value of <0.05 was considered statistically significant. Statistical analysis was performed using SPSS software version 23.0.

RESULTS

A total of 22 Paediatric patients with hypoxic–ischemic encephalopathy were included in the study. The study population consisted of children ranging from infancy to 18 years of age. Most patients had a documented history of a hypoxic–ischemic event, including perinatal asphyxia, respiratory failure, or cardiac arrest. All MRI examinations were of diagnostic quality.

	Number of Patients	Percentage%
Male	12	54.5%
Female	10	45.5%

Table 1: Gender Distribution among the Study Population (N = 18)

	Number of Patients	Percentage (%)
Infants (<1 year)	8	36.4%
Toddlers (1-3 years)	6	27.3%
Children (3–10 years)	5	22.7%
Adolescents (>10 years)	3	13.6%

Table 2: Age Distribution among the Study Population (N = 18)

Distribution of White Matter Abnormalities

White matter abnormalities were detected on MRI in all patients. The majority demonstrated bilateral and diffuse involvement. The periventricular white matter was the most commonly affected region, followed by subcortical and deep white matter.

White Matter Region	Number of Patients	Percentage (%)
Periventricular white matter	15	68.2%
Subcortical white matter	4	18.2%
Deep white matter	3	13.6%
Diffuse involvement	14	63.6%
Focal involvement	8	36.4%
Bilateral involvement	18	81.8%
Unilateral involvement	4	18.2%

Table 3: Distribution of White Matter Involvement on MRI

Age-Wise Pattern of White Matter Injury

Age-related differences in white matter involvement were observed. Younger children predominantly showed periventricular white matter injury, whereas older children more frequently exhibited subcortical and deep white matter involvement, often with diffuse distribution.

Age Group	Predominant white Matter Pattern	Number of Patients
<3 years	Periventricular predominance	11
3–10 years	Both periventricular and subcortical	4
>10 years	Subcortical and deep white matter	3

Table 4: Age-Wise Distribution of White Matter Injury Patterns

MRI Signal Characteristics

All affected white matter regions demonstrated hyperintense signal on T2-weighted and FLAIR images. Loss of normal white matter volume and gliotic changes were noted in patients imaged during the chronic phase. Diffusion restriction was observed in patients imaged during the early or subacute phase of injury.

MRI Feature	Number of Patients	Percentage (%)
T2/FLAIR hyperintensity	22	100%
Diffusion restriction	3	13.6%
White matter volume loss	13	59.1%
Gliotic changes	6	27.3%
Cystic white matter changes	3	13.6%

Table 5: MRI Signal Characteristics of White Matter Injury

Associated MRI Findings

In addition to white matter abnormalities, associated gray matter and structural changes were observed in several patients. Cortical involvement and deep gray matter injury were more frequently seen in patients with severe hypoxic–ischemic insult.

Associated Findings	Number of Patients	Percentage (%)
Cortical involvement	6	27.2%
Basal ganglia / thalamic involvement	2	9%
Cerebral atrophy	8	36.4%
Ventricular dilatation	5	22.7%

Table 6: Associated MRI Findings

Timing of MRI and Evolution of Findings

MRI examinations were performed at different intervals following the hypoxic–ischemic event. Three patients underwent MRI within the first 7 days (early phase), two patients were imaged between 7 and 21 days (subacute phase), and 17 patients underwent MRI more than 21 days after the insult (chronic phase).

In the early phase, diffusion-weighted imaging demonstrated restricted diffusion involving the periventricular white matter, consistent with acute cytotoxic edema. During the subacute phase, T2-weighted and FLAIR images showed increasing white matter hyperintensity with partial resolution of diffusion restriction. In the chronic phase, MRI demonstrated persistent T2/FLAIR hyperintensity, white matter volume loss, gliosis, ventricular dilatation, and cerebral atrophy, indicating irreversible injury and chronic sequelae.

Timing of MRI	Number of patients	Percentage (%)	Predominant MRI findings
Early (<7 days)	3	13.6	Diffusion restriction in periventricular white matter; subtle T2 hyperintensity
Subacute (7–21 days)	2	9.1	T2/FLAIR hyperintensity; decreasing diffusion restriction
Chronic (>21 days)	17	77.3	White matter volume loss, gliosis, ventricular dilatation, cerebral atrophy

Table 7. Timing of MRI and Evolution of Imaging Findings (N =22)

MRI Timing	Diffusion Restriction (+), n	Diffusion Restriction (−), n	Total	P-value
Early (<7 days)	3	0	3	
Subacute (7–21 days)	0	2	2	
Chronic (>21 days)	0	17	17	
Total	3 (13.6)	19 (86.4)	22	**<0.001***

Table 8. Association Between MRI Timing and Diffusion Restriction

Diffusion restriction was observed exclusively in patients who underwent MRI within the first 7 days after hypoxic–ischemic injury. No diffusion restriction was identified in patients imaged during the subacute or chronic phases. Fisher's exact test demonstrated a highly significant association between MRI timing and the presence of diffusion restriction ($p < 0.001$).

Representative Cases

Case 1-

Area of cystic encephalomalacia along with volume loss, adjacent gliosis and shrunken cerebral cortex noted in bilateral parieto-occipital, left temporal and left frontal regions - s/o sequelae to severe Hypoxic ischemic encephalopathy.

E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.287

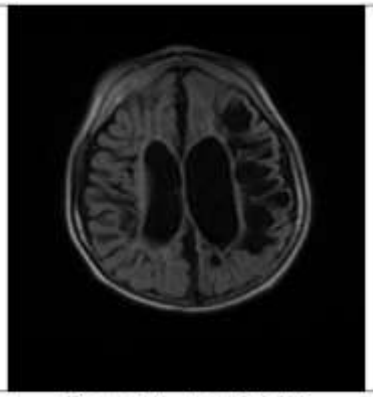

Figure 1.1- Axial FLAIR

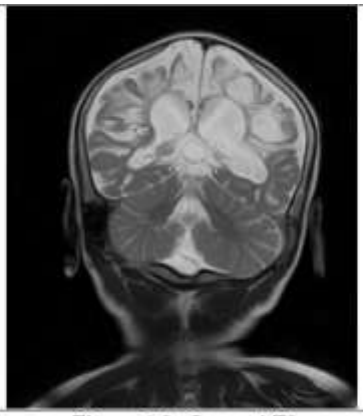

Figure 1.2- Coronal T2

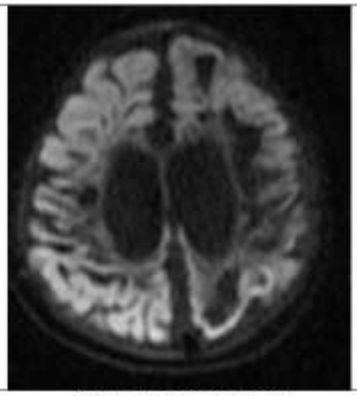

Figure 1.3- Axial DWI

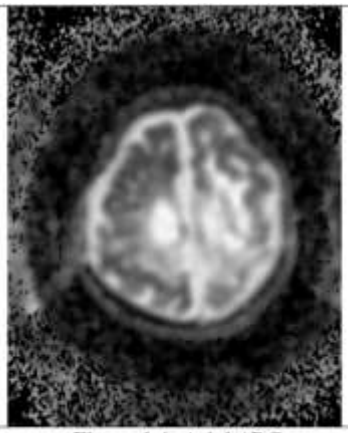

Figure 1.4- Axial ADC

Case 2

T2/FLAIR white matter hyperintensities involving the periventricular region of right occipital lobe with atrophied deep portion of adjacent gyri.

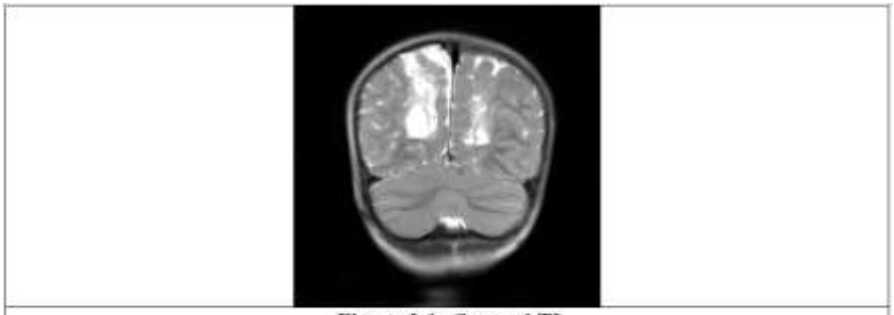

Figure 2.1- Coronal T2

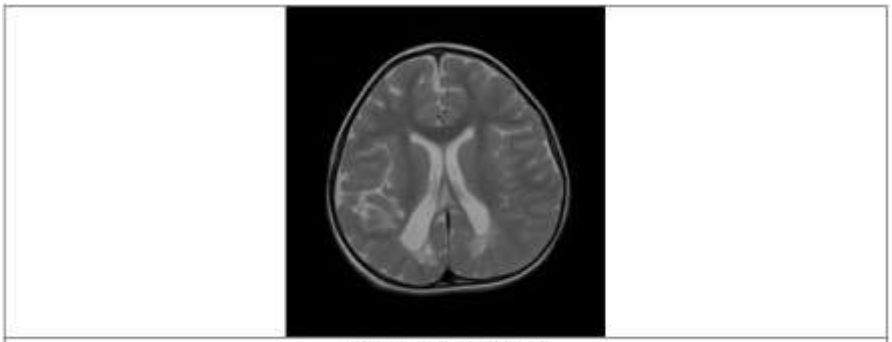

Figure 2.2- Axial T2

DISCUSSION

Hypoxic–ischemic encephalopathy (HIE) represents a heterogeneous pattern of brain injury in children, with white matter involvement constituting a major determinant of long-term neurological outcome. In the present retrospective study, MRI demonstrated a wide spectrum of white matter abnormalities in all included patients, emphasizing the vulnerability of developing cerebral white matter to hypoxic–ischemic insults across paediatric age groups.

In our cohort, periventricular white matter was the most frequently involved region, particularly in younger children. This finding is consistent with previous studies that have demonstrated selective vulnerability of immature oligodendrocytes and the periventricular watershed zones during early brain development.[6] Barkovich et al. described periventricular white matter injury as a characteristic manifestation of hypoxic–ischemic injury in neonates and infants, attributed to incomplete myelination and reduced autoregulatory capacity in these regions.[7]

Older children in our study more commonly exhibited subcortical and deep white matter involvement, often with diffuse distribution. This age-dependent variation in injury pattern has been previously reported and is thought to reflect maturational changes in cerebral metabolism, vascular supply, and myelination.[8] Rutherford et al. observed that post-neonatal hypoxic–ischemic events tend to produce more extensive hemispheric white matter injury compared with the predominantly periventricular pattern seen in neonates.[4]

All patients in the present study demonstrated T2-weighted and FLAIR hyperintensity within the affected white matter, with chronic cases showing volume loss and gliosis. These findings are well documented in the literature and reflect the evolution of hypoxic–ischemic injury from cytotoxic edema to irreversible tissue loss. The timing of MRI had a significant influence on the imaging appearance. Diffusion restriction was observed exclusively in patients who underwent MRI within the first seven days after hypoxic–ischemic injury (3/3 patients) and was absent in the subacute and chronic groups. This association between MRI timing and diffusion restriction was highly statistically significant (Fisher's exact test, $p < 0.001$). These findings are consistent with the work of Cowan et al. and Rutherford et al., who demonstrated that diffusion-weighted imaging is most sensitive during the acute phase of hypoxic–ischemic injury and that diffusion abnormalities gradually normalize over the subsequent days, while T2-

weighted and FLAIR abnormalities become increasingly conspicuous.[4]

Cortical involvement and deep gray matter injury were observed in a subset of patients, particularly those with severe hypoxic–ischemic insults. Similar associations have been reported by Martinez-Biarge et al., who demonstrated that combined white matter and deep gray matter injury is indicative of more profound hypoxia and is associated with poorer neurological outcomes.[5] The presence of cerebral atrophy and ventricular dilatation in chronic cases in our study further supports the role of MRI in assessing long-term sequelae of hypoxic–ischemic injury.

The findings of this study underscore the importance of systematic evaluation of white matter changes on Paediatric brain MRI in suspected HIE. While deep gray matter injury has traditionally been emphasized, white matter abnormalities are equally significant and may occur in isolation or in combination with gray matter involvement. Recognition of these patterns can aid in timing the insult, assessing severity, and guiding prognostication.

Limitations

The limitations of this study include its retrospective design and relatively small sample size. Clinical outcome correlation was limited by the availability of follow-up data. Advanced imaging techniques such as diffusion tensor imaging and quantitative ADC analysis were not included, which may have provided additional insight into microstructural white matter damage.

CONCLUSION

Magnetic resonance imaging is an essential tool for evaluating white matter injury in children with hypoxic–ischemic encephalopathy. In this retrospective study, white matter abnormalities were consistently identified, with the periventricular white matter being the most commonly affected region, followed by the subcortical and deep white matter. Bilateral and diffuse patterns of involvement predominated, while associated cortical and deep gray matter abnormalities were observed in patients with more severe injury.

The distribution of white matter changes varied with age, reflecting differences in brain maturation and selective vulnerability to hypoxic–ischemic insults. Conventional MRI sequences, together with diffusion-weighted imaging, effectively demonstrated both acute and chronic changes, allowing characterization of the extent and evolution of injury.

Recognition of characteristic MRI patterns of white matter involvement is important for accurate diagnosis, assessment of injury severity, and prognostication. A systematic evaluation of white matter abnormalities should therefore be incorporated into routine MRI interpretation in children with suspected hypoxic–ischemic encephalopathy. Larger prospective studies incorporating advanced MRI techniques and long-term neurodevelopmental follow-up are warranted to further define imaging biomarkers that can improve outcome prediction and guide patient management.

REFERENCES

1. Ferriero DM. Neonatal Brain Injury. N Engl J Med 2004;351(19):1985-95.
2. Back SA, Miller SP. Brain injury in premature neonates: a primary cerebral dysmaturation disorder? Annals of Neurology 2014;75(4):469–86.
3. Barkovich AJ, Raybaud C. Pediatric neuroimaging. 6th edn. Philadelphia Baltimore New York London Buenos Aires Hong Kong Sydney Tokyo: Wolters Kluwer 2019: p. 1.
4. Cowan F, Rutherford M, Groenendaal F, et al. Origin and timing of brain lesions in term infants with neonatal encephalopathy. The Lancet 2003;361(9359):736-42.
5. Martinez-Biarge M, Diez-Sebastian J, Rutherford MA, et al. Outcomes after central grey matter injury in term perinatal hypoxic-ischaemic encephalopathy. Early Human Development 2010;86(11):675-82.
6. Volpe JJ. Brain injury in the premature infant: Overview of clinical aspects, neuropathology, and pathogenesis. Seminars in Pediatric Neurology 1998;5(3):135-51.
7. Barkovich AJ, Hajnal BL, Vigneron D, Sola A, Partridge JC, Allen F, et al. Prediction of neuromotor outcome in perinatal asphyxia: evaluation of MR scoring systems. AJNR Am J Neuroradiol 1998;19(1):143-9.
8. Back SA, Rosenberg PA. Pathophysiology of glia in perinatal white matter injury. Glia 2014;62(11):1790-815.