

# International Journal of Radiology and Diagnostic Imaging



E-ISSN: 2664-4444  
P-ISSN: 2664-4436  
[www.radiologypaper.com](http://www.radiologypaper.com)  
IJRDI 2022; 5(4): 25-29  
Received: 14-08-2022  
Accepted: 19-09-2022

**Dr. Virag Shethna**  
3<sup>rd</sup> year, Department of  
Radiodiagnosis, Amc Met  
Medical College and Lg  
Hospital, Maninagar,  
Ahmedabad, Gujarat, India

**Dr. Dipti A Shah**  
Professor and Head,  
Department of Radiodiagnosis,  
Amc Met Medical College and  
Lg Hospital, Maninagar,  
Ahmedabad, Gujarat, India

**Corresponding Author:**  
**Dr. Virag Shethna**  
3<sup>rd</sup> year, Department of  
Radiodiagnosis, Amc Met  
Medical College and Lg  
Hospital, Maninagar,  
Ahmedabad, Gujarat, India

## A study of magnetic resonance imaging findings in patients with encephalopathies in pediatric age group

**Dr. Virag Shethna and Dr. Dipti A Shah**

DOI: <http://dx.doi.org/10.33545/26644436.2022.v5.i4a.282>

### Abstract

**Introduction:** Magnetic resonance (MR) imaging is used with increasing frequency to evaluate the child and neonatal brain because it can provide important diagnostic and prognostic information that is needed for optimal treatment and appropriate counselling. Moreover, to accurately interpret the findings, specific knowledge is needed about the normal MR imaging appearances of the physiologic processes of myelination, cell migration and sulcation, as well as patterns of injury, in the neonatal brain at various stages of gestational development.

### Materials and Methods

- **Inclusion Criteria:** Patients of pediatric age group (neonate to 14 years of age) having seizure and confusion, undergoing magnetic resonance imaging for suspicion of encephalopathy and diagnosed as the same in magnetic resonance imaging.
- **Exclusion Criteria:** Patients with central nervous system pathologies other than encephalopathies. Patients having metallic implants and pacemaker. Patients having claustrophobia.
- **Study type:** Retrospective study (Record based study)

**Results and Conclusion:** In the study, most common type of encephalopathy was hypoxic-ischemic encephalopathy (48.7%) followed by hypoglycemic encephalopathy (36.5%) followed by metabolic encephalopathy. Out of 20 patients having MRI findings of HIE, 60% patients had diffusion restriction and FLAIR hyperintensity predominantly in basal ganglia region. Out of 15 patients having hypoglycemic encephalopathy, 66.6% patients had restricted diffusion predominantly in cerebral cortex. Out of 6 patients having metabolic encephalopathy, 66.6% patient had restricted diffusion predominantly in centrum semiovale.

Despite recent advances in the MR imaging-based characterization of these conditions clinical history must be reviewed carefully for information that may help identify the cause of injury.

**Keywords:** MRI, encephalopathy, basal ganglia, cerebellum, diffusion restriction, DWI

### Introduction

Magnetic resonance (MR) imaging is used with increasing frequency to evaluate the child and neonatal brain because it can provide important diagnostic and prognostic information that is needed for optimal treatment and appropriate counselling. Moreover, to accurately interpret the findings, specific knowledge is needed about the normal MR imaging appearances of the physiologic processes of myelination, cell migration and sulcation, as well as patterns of injury, in the neonatal brain at various stages of gestational development.

Hypoxic-ischemic injury, the most common cause of childhood and neonatal encephalopathy, has characteristic appearances that depend on the severity and duration of the insult as well as the stage of brain development. Diffusion-weighted MR imaging and MR spectroscopy depict abnormalities earlier than conventional MR imaging sequences. However, diffusion-weighted imaging, if performed in the first 24 hours after the insult, might lead to underestimation of the extent of injury.

When the MR findings are atypical, the differential diagnosis of encephalopathy also should include congenital and infectious diseases. Despite recent advances in the MR imaging-based characterization of these conditions, the clinical history must be borne in mind to achieve an accurate diagnosis.

**Aims and objectives:** To evaluate magnetic resonance imaging findings in encephalopathies in pediatric age group (Neonate to 14 years of age)

**Methodology**

- **Inclusion Criteria:** Patients of pediatric age group (neonate to 14 years of age) having seizure and confusion, undergoing magnetic resonance imaging for suspicion of encephalopathy and diagnosed as the same in magnetic resonance imaging.
- **Exclusion Criteria:** Patients with central nervous system pathologies other than encephalopathies. Patients having metallic implants and pacemaker. Patients having claustrophobia.
- **Study type:** Retrospective study (recordbased study)
- **Study Time:** October 2021 to march 2022
- **Need of consent: consent not required for retrospective study:** A total of 41 cases clinically suspected of encephalopathy & diagnosed of encephalopathy by magnetic resonance imaging of brain, at the department of Radiodiagnosis, AMC MET Medical College, Maninagar, Ahmedabad were included in this study.
- MRI examinations were performed by using Siemens Magnetom Essenza 1.5T MRI.

**Routine sequences performed in all participants**

Multiplanar scout sections were obtained for planning sequences. Imaging of whole brain MR from vertex to foramen magnum including base of skull using axial, coronal and sagittal sections with the following sequences was done.

**T1WI T2WI**

TR:490msec TR: 4300msec  
TE:8.9msec TE:101msec

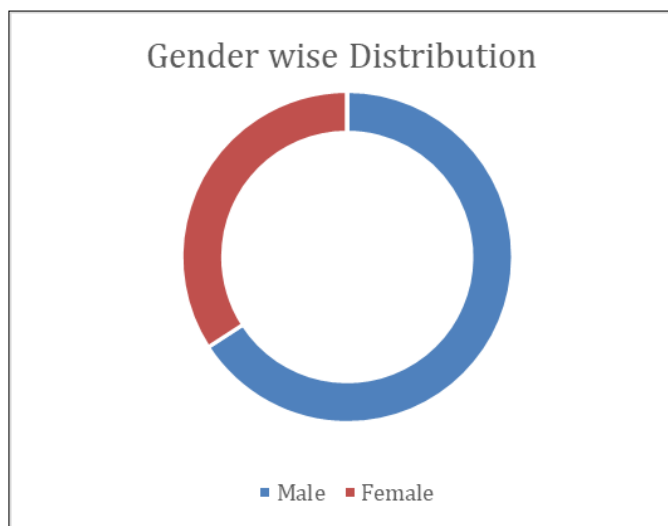
**FLAIR DWI**

TR:8000msec TR: 3800msec  
TE:80msec TE:113msec  
b :500,1000,1500

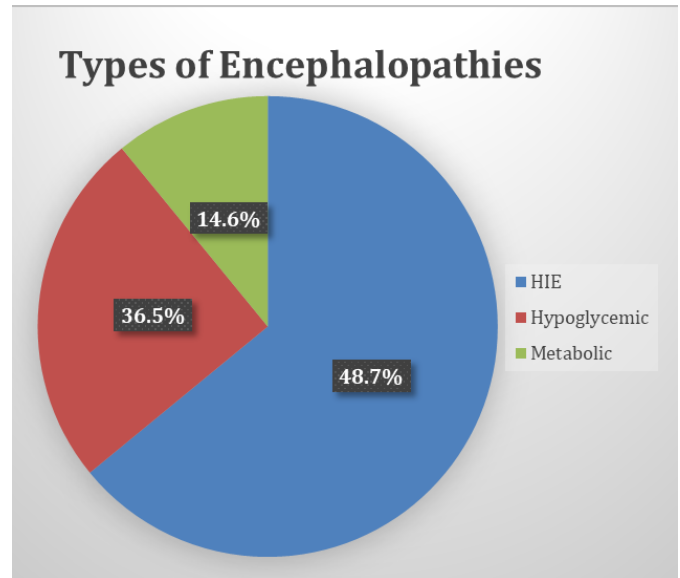
**SWI:**

TR: 49msec  
TE: 40msec

**Results**



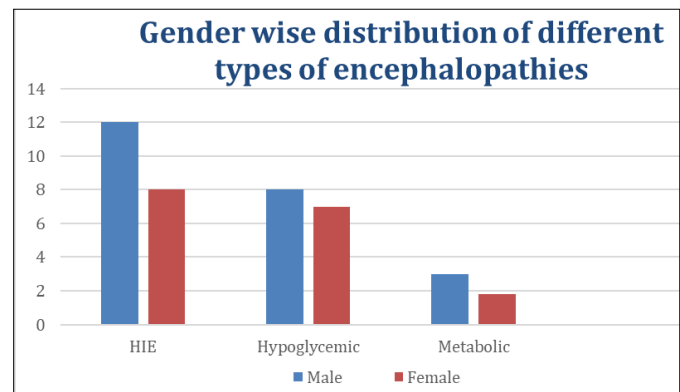
**Fig 1:** Out of 41 patients included in the study, 23 were males & 18 were females.



**Fig 2:** In the following study, most common type of encephalopathy is hypoxic-ischemic encephalopathy followed by hypoglycemic encephalopathy.

**Table 1:** Gender wise distribution of different types of encephalopathy

	Male	Female	Total
Hypoxic-ischemic encephalopathy (HIE)	12(52%)	8(44%)	20
Hypoglycemic encephalopathy	8(34.7%)	7(38.8%)	15
Metabolic encephalopathy	3(13%)	3(16.6%)	6
Total	23(56%)	18(44%)	41

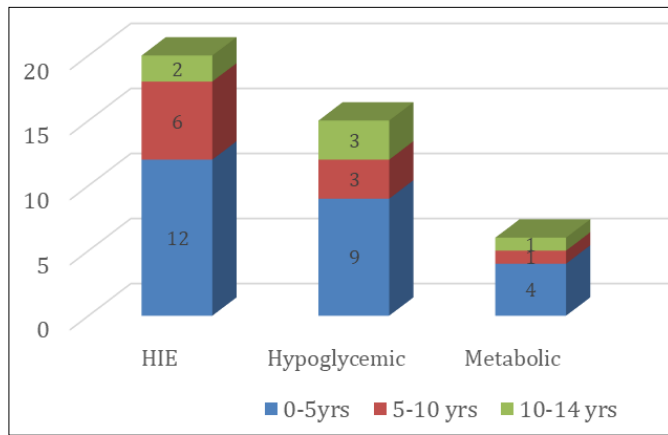


**Fig 3:** Out of total 41 patients having encephalopathy, 23 patients were males and 18 were females.

**Table 2:** Age wise distribution of different types of encephalopathy

Age (in years)	HIE	Hypoglycemic	Metabolic
0-1	8(40%)	5(33.3%)	2(33.3%)
1-5	4(20%)	4(26.6%)	2(33.3%)
5-10	6(30%)	3(20%)	1(16.6%)
10-14	2(10%)	3(20%)	1(16.6%)
Total	20(48.7%)	15(36.5%)	6(14.6%)

In the following study, hypoxic ischemic encephalopathy and hypoglycemic encephalopathy were predominantly found in 0 to 1 year of age group, while metabolic encephalopathy was predominantly seen in 0 to 5 year of age group.



**Fig 4:** Age wise distribution of different types of encephalopathies

**Table 3:** Distribution of MRI changes (FLAIR and DWI Hyperintensity) in hypoxic-ischemic encephalopathy

Site of involvement	No. of patients	Percentage
Basal ganglia	12	60%
Thalamus	8	40%
Internal capsule	8	40%
Subcortical white matter	5	25%
Periventricular white matter	9	45%

In hypoxic ischemic encephalopathy, there was predominant involvement of basal ganglia region, followed by thalamus and internal capsule in the following study.

**Table 4:** Distribution of MRI changes (FLAIR and DWI hyperintensity) in hypoglycemic encephalopathy

Site of involvement	No. of patients	Percentage
Basal ganglia	7	46.6%
Cerebral cortex	10	66.6%
Internal capsule	6	40%
Hippocampus	3	20%

In hypoglycemic encephalopathy, there was predominant involvement of cerebral cortex, followed by basal ganglia region in the following study.

**Table 5:** Distribution of MRI changes (FLAIR and DWI hyperintensity) in metabolic encephalopathy

Site of involvement	No. of patients	Percentage
Centrum semiovale	4	66.6%
Cerebellar white matter	2	33.3%
Internal capsule	3	50%
Brainstem	2	33.3%

In metabolic encephalopathy, there was predominant involvement of centrum semiovale region in the following study.

**Discussion**

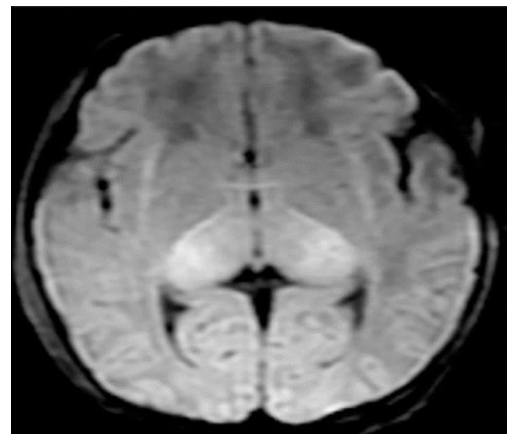
**Normal myelination**

Normal myelination in the brain is well depicted on T1-weighted MR images. Myelination causes shortening of T1 relaxation time in white matter structures because of the increased amount of glycolipids (e.g. galactocerebroside) and decreased number of free water molecules, with the latter change occurring because of hydrogen binding to glycolipids and cholesterol. As a general rule, myelination in the brain proceeds from the caudal to the rostral aspect,

from the posterior to the anterior aspect, and from the center to the periphery. Thus, the dorsal brainstem, which contains the medial longitudinal fasciculus and medial lemniscus, undergoes myelination at 24–28 weeks of gestation, before the ventral brainstem. The subthalamic nucleus and the ventrolateral nucleus of the thalamus also have undergone myelination by 28 weeks of gestation. The posterior portion of the posterior limb of the internal capsule should show signal hyperintensity on T1-weighted images obtained at 36–37 weeks of gestation. Myelination of the corticospinal tract progresses in the cranial direction and is seen in the central corona radiata and perirolandic white matter at 38–40 weeks. In the preterm brain, T2-weighted imaging may show myelin in gray matter structures (vestibular nuclei, medial geniculate bodies, lateral geniculate bodies, inferior olivary nucleus, and inferior colliculi) before T1-weighted imaging does, whereas T1-weighted imaging is better at depicting myelin in white matter tracts of the medial longitudinal fasciculus, medial lemniscus and lateral lemniscus.

**Normal migrational milestones**

Until 28–30 weeks of gestational age, the germinal matrix is depicted on T2-weighted images as a low-signal-intensity band that extends along the ventricular wall. The band is most prominent at the caudothalamic groove (ganglionic eminence). Before 33 weeks of gestational age, the white matter has a laminated appearance, with three alternating bands of different signal intensity on T2-weighted images.



**Fig 5:** Normal posterior limb of internal capsule hyperintensity at birth (T1W MRI sequence)

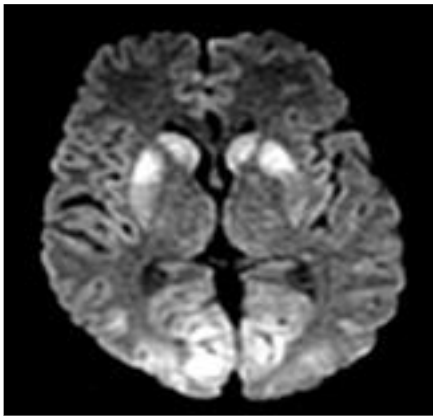
**Causes of Encephalopathy in the pediatric age group**

- Hypoxic-Ischemic Encephalopathy (HIE)
- Hypoglycaemic encephalopathy
- Metabolic encephalopathy

**Hypoxic-ischemic encephalopathy**

- Hypoxic-ischemic injury is a major cause of death and cerebral palsy in children, occurring in two to nine of 1000 live births. Hypoxic-ischemic injury in full-term infants accounts for approximately 15%–20% of neonatal mortality, and 25% of those who survive demonstrate significant deficits in subsequent development. In preterm infants, hypoxic-ischemic injury is even more common; it occurs in 5% of infants born before 32 weeks of gestational age. Up to 19% of infants born before 28 weeks of gestation develop cerebral palsy.

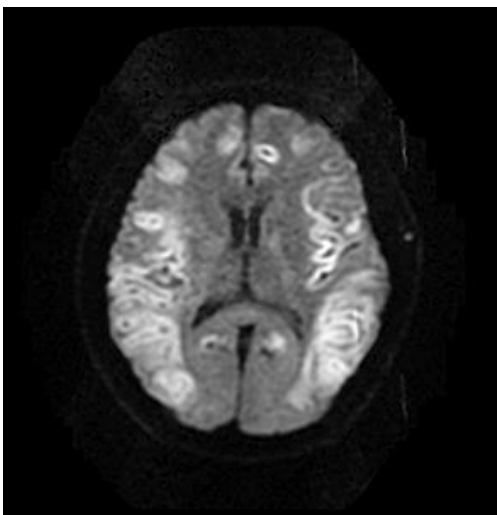
- Approximately 50% of cases of cerebral palsy occur in infants born prematurely. Severe hypoxic-ischemic insults to the premature brain typically injure the thalamus, anterior part of the vermis and dorsal brainstem.
- Involvement of the basal ganglia, hippocampus, cerebellum, and corticospinal tracts also may be seen. Injury to the basal ganglia (which undergo myelination at 35–36 weeks of gestation) is usually less extensive than that to the thalamus, which undergoes myelination at 24–25 weeks of gestation.
- Diffusion weighted images will demonstrate increased signal intensity in the ventrolateral thalami and basal ganglia (particularly the posterior putamina), in the perirolandic regions and along the corticospinal tracts.



**Fig 6:** Bilateral symmetrical hyperintensities in head of caudate nuclei, putamen and cortical grey matter in DWI sequence in a patient diagnosed with hypoxic ischemic encephalopathy.

#### Hypoglycemic Encephalopathy

- Hypoglycemic encephalopathy occurs due to prolonged severe hypoglycemia.
- On MR imaging, it can manifest as bilateral areas of increased signal on T2 and FLAIR images affecting the posterior limb of internal capsule, cerebral cortex, hippocampus and basal ganglia.



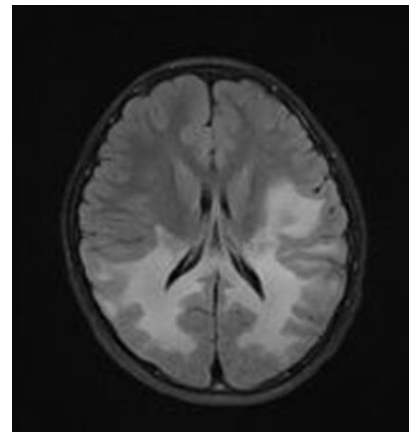
**Fig 7:** Bilateral cortical hyperintensities in DWI sequence in a patient diagnosed with hypoglycemic encephalopathy.

#### Metabolic encephalopathy

- Metabolic encephalopathy is defined as an alteration in

consciousness caused by diffuse or global brain dysfunction from impaired cerebral metabolism.

- The main metabolic causes of neonatal encephalopathy are congenital lactic acidosis (the commonest of these is pyruvate dehydrogenase deficiency), urea cycle disorders, and amino acidurias. Maple syrup urine disease is related to a deficiency in the metabolism of branched-chain amino acids. The most severe, classic neonatal form of this disease is characterized by early postnatal onset and rapidly progressive neurologic deterioration.
- The MR imaging appearance is highly characteristic, with restricted water diffusion in the cerebellar white matter, dorsal brainstem, cerebral peduncles, posterior limb of the internal capsule, and posterior centrum semiovale.



**Fig 7:** Flair image showing hyperintensity in posterior centrum semiovale

#### Conclusion

- In the study, most common type of encephalopathy was hypoxic-ischemic encephalopathy (48.7%) followed by hypoglycemic encephalopathy (36.5%).
- Out of total 41 patients having encephalopathy, 20(48.7%) patients had hypoxic-ischemic encephalopathy, 15(36.5%) patients had hypoglycemic encephalopathy and 6(14.6%) patients had metabolic encephalopathy.
- Out of 20 patients having MRI findings of HIE, 60% patients had diffusion restriction and FLAIR hyperintensity predominantly in basal ganglia region.
- Out of 15 patients having hypoglycemic encephalopathy, 66.6% patients had restricted diffusion predominantly in cerebral cortex.
- Out of 6 patients having metabolic encephalopathy, 66.6% patient had restricted diffusion predominantly in centrum semiovale.

#### References

1. Huang BY, Castillo M. Hypoxic-ischemic braininjury: imaging findings from birth to adulthood. *Radio Graphics*. 2008;28(2):417-439.
2. Barkovich AJ. Brain and spine injuries in infancy and childhood. In: *Pediatric neuroimaging*. 4th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; c2005. 190-290.
3. Liauw L, van der Grond J, van den Berg-Huysmans AA, Palm-Meinders IH, van Buchem MA, van Wezel-Meijler G. Hypoxic-ischemic encephalopathy:

- diagnostic value of conventional MR imaging pulse sequences in term-born neonates. *Radiology*. 2008;247(1):204-212.
4. Rutherford M, Counsell S, Allsop J, *et al*. Diffusionweighted magnetic resonance imaging in term perinatal brain injury: a comparison with site of lesion and time from birth. *Pediatrics*. 2004;114(4):1004-1014.
  5. Robertson RL, Ben-Sira L, Barnes PD, *et al*. MR line-scan diffusion-weighted imaging of term neonates with perinatal brain ischemia. *AJNR Am J Neuroradiol*. 1999;20(9):1658-1670.
  6. Rutherford MA, Pennock JM, Counsell SJ, *et al*. Abnormal magnetic resonance signal in the internal capsule predicts poor neurodevelopmental outcome in infants with hypoxic-ischemic encephalopathy. *Pediatrics*. 1998;102(2 pt 1):323-328.
  7. O'Shea TM. Cerebral palsy in very preterm infants: new epidemiological insights. *Ment Retard Dev Disabil Res Rev*. 2002;8(3):135-145.
  8. Huang BY, Castillo M. Hypoxic-ischemic brain injury: imaging findings from birth to adulthood. *Radio Graphics*. 2008;28(2):417-439.
  9. Chao CP, Zaleski CG, Patton AC. Neonatal hypoxicischemic encephalopathy: multimodality imaging findings. *Radio Graphics*. 2006;26(suppl 1):S159-S172.
  10. Paneth N, Pinto-Martin J, Gardiner J, *et al*. Incidence and timing of germinal matrix/intraventricular hemorrhage in low birth weight infants. *Am J Epidemiol*. 1993;137(11):1167-1176.
  11. Kirton A, Deveber G, Pontigon AM, Macgregor D, Shroff M. Presumed perinatal ischemic stroke: vascular classification predicts outcomes. *Ann Neurol*. 2008;63(4):436-443.
  12. Roelants-van Rijn AM, Groenendaal F, Beek FJ, Eken P, van Haastert IC, de Vries LS. Parenchymal brain injury in the preterm infant: comparison of cranial ultrasound, MRI and neurodevelopmental outcome. *Neuropediatrics*. 2001;32(2):80-89.
  13. Kuban K, Sanocka U, Leviton A, *et al*. White matter disorders of prematurity: association with intraventricular hemorrhage and ventriculomegaly. The Developmental Epidemiology Network. *J Pediatr*. 1999;134(5):539-546.

**How to Cite This Article**

Shethna V, Shah DA. A study of magnetic resonance imaging findings in patients with encephalopathies in pediatric age group. *International Journal of Radiology and Diagnostic Imaging*. 2022;5(4):25-29.

DOI: <https://doi.org/10.33545/26644436.2022.v5.i4a.282>

**Creative Commons (CC) License**

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.