

EVALUATION OF INVOLVEMENT OF DIFFERENT SITES OF BRAIN BY MRI IN 1-5 YEAR AGE GROUP CHILDREN WHO SUFFERED FROM HYPOXIC ISCHAEMIC ENCEPHALOPATHY IN THE NEONATAL PERIOD

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ABSTRACT

BACKGROUND

Perinatal cerebral hypoxic-ischemic insults result in neonatal brain injury with serious long-term neuro developmental sequelae. Magnetic resonance imaging (MRI) of the brain is an ideal and safe imaging modality for suspected hypoxic-ischemic injury.¹ The outcome for patients with hypoxic-ischemic brain Injury (HIBI) is often poor. It is important to determine prognosis as soon as possible for better management. Clinical diagnosis is not so much helpful and ancillary investigations, particularly imaging are needed to understand the severity of brain injury and the likely outcome.

MATERIALS AND METHODS

50 children who have suffered from hypoxic ischaemic encephalopathy during neonatal period were included in this study. The frequencies of involvement of different sites were evaluated by MRI of brain.

RESULTS

Of those 50 patients, Parieto-occipital cortex involvement seen in 16 patients. Periventricular white matter (WM) involvement seen in 7 cases, thalamus and basal ganglia involvement seen in 5 and 6 cases respectively. Subcortical WM involvement seen in 4 cases. Corpus callosum, brain-stem, and cerebellum involvement seen in 3 patients each.

CONCLUSION

After the data collection, we have come to a conclusion that parieto-occipital cortex has been most frequently involved among the different sites that are usually affected in HIE.

KEYWORDS

Hypoxic Ischaemic Encephalopathy, Watershed, Parieto-Occipital Lobe, MRI, 1-5 year age group, Signal Intensity.

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BACKGROUND

Neonatal encephalopathy, following severe birth asphyxia or perinatal hypoxia is referred to as hypoxic ischaemic encephalopathy (HIE).² Insufficient cerebral blood flow (ischemia) and decreased oxygen level in the blood (hypoxia) lead to loss of normal cerebral autoregulation. This results in diffuse brain injury and thereby causes hypoxic-ischemic encephalopathy (HIE). The outcomes of HIE are devastating and permanent, making it a major burden for the patient, the family, and society. It is critical to identify and develop therapeutic strategies to reduce brain injury in new-borns with HIE. The underlying pathophysiology of perinatal HIE is difficult to study in the human.³ While the

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exact cause is not always identified, antecedents include cord prolapse, uterine rupture, abruptio placenta, placenta previa, maternal hypotension, breech presentation, or shoulder dystonia. The manifestations of perinatal HIE in early postnatal life include abnormal fetal heart rate tracings, poor umbilical cord gases ($\text{pH} < 7.0$ or base deficit $\geq 12 \text{ mmol/L}$), low Apgar scores, presence of meconium stained fluid or the need for respiratory support within the first several minutes of postnatal life.^{4,5} Cerebral ischaemia occurs as a consequence of cerebral oedema (which compresses cerebral vessels) and also reduced cerebral perfusion due to myocardial dysfunction as a result of hypoxic cardiomyopathy. Cerebral hypoxia-ischemia (asphyxia) occurring in the fetus and new born infant is a major cause of acute mortality and chronic neurological disability in survivors.^{6,7,8} Following severe birth asphyxia, 25% infants are likely to develop the syndrome of HIE.^{9,10,11} Neonatal encephalopathy is estimated as 3 per 1000 live birth and HIE alone accounts for 1.5 per 1000 live birth. Neonatal HIE has been graded clinically by Sarnat and Sarnat into mild (stage1), moderate (stage2), severe (stage3).^{12,13,14} First line investigation for the detection of



these lesions is trans-cranial ultrasound, but its role is limited for older children when anterior fontanelles are closed. Although USG is non-invasive, readily available and reproducible, it can only reveal the most severe hypoxic-ischemic lesions and lacks prognostic significance. Although CT scan is also helpful, it has the disadvantage of high radiation hazards and resolution is inferior to MRI. MRI is the most sensitive and specific modality to determine hypoxia induced changes.¹⁵ The main role of MRI is to exclude structural anomalies and in assessing the extent and nature of injury. Thereby, it helps in prognosticating the outcome and planning neurodevelopmental therapy. In our study we have evaluated the involvement of different sites of brain by MRI in 1-5 year age group children suffered from hypoxic ischaemic encephalopathy in the neonatal period.

Aims and Objectives

This prospective study was carried out with the aim of generating information regarding the role of MRI-

- To detect different sites of brain involvement in 1-5 year age group children who have suffered from neonatal hypoxic ischaemic encephalopathy.
- To determine the commonly affected areas in HIE

MATERIALS AND METHODS

Inclusion Criteria

1-5 year age group children who have suffered from neonatal hypoxic encephalopathy.

Exclusion Criteria

- children who could not be sedated.
- children who have metallic implants not compatible with MRI.
- parents did not give consent inspite of proper Explanation.

The study was carried out in our Department of Radiodiagnosis, NBMC&H for a time period of 1 year among 50 children of 1-5 year age group who have suffered from hypoxic ischaemic encephalopathy during neonatal period. Our study was institution based observational study and purposive sampling done and cross-sectional study design was applied.

These patients were referred for neurological assessment because of follow up of hypoxic-ischaemic brain insults in the neonatal period. All the patients underwent clinical assessment, appropriate investigation and MR imaging. We have not administered IV gadolinium as part of study. Only non-contrast MRI study were done.

- We had used the MRI machine-1.5 tesla m/s GE make, BRIVO model

MRI Protocols¹⁶

Literature suggested the following protocol of MRI scans after the hypoxic-ischemic event:

- Axial T1-and T2-weighted images, slice thickness 4 mm.
- Sagittal T1-weighted images, slice thickness: 1.5–3 mm.
- Coronal T2-weighted images, slice thickness: 4 mm.

- DWI.
- Venous MRA.
- Arterial MRA.
- MR spectroscopy (MRS).
- Alternative scan after administration of a contrast medium if CNS infection is suspected (after examination of renal function).

In everyday clinical practice at our centre the scans are performed using shortened protocol which in majority of cases, allow us to obtain diagnostic images free from motion artefacts associated with the child awakening:

- Coronal T2-weighted images.
- Axial T1-weighted images.
- Axial GRE/T2*-weighted images.
- DWI.
- Sagittal T2-weighted images.
- Axial T2-weighted images.
- Axial FLAIR images.

RESULTS

In our study we included 50 children in the age group 1-5 years. Out of them 30 were male and 20 were female child. Out of those 50 children, 35 were in 1-3 year age group and 15 were in 3-5 year age group.

We found brain parenchyma was normal in 3 cases and altered parenchymal signal was found in 47 cases. Out of those 47 cases, Parieto-occipital cortex involvement seen in 16 patients. Periventricular WM involvement was seen in 7 cases, thalamus and basal ganglia involvement in 5 and 6 cases respectively. Subcortical WM involvement was found in 4 patients. Corpus callosum, brain-stem, and cerebellum involvement in 3 patients each.

Total No. of Children	50
Sex	M=30, F=20
1-3 yr. Age Group	35
3-5 yr. Age Group	15

Table 1. Distribution of Patients According to Sex and Age Group

Imaging Findings of Brain	No. of Cases
1. Altered Parenchymal Signal:	
a. Central	
-Periventricular WM	7
-Basal ganglia	6
-Thalamus	5
-Corpus callosum	3
-Brain stem	3
-Cerebellar WM	3
b. Peripheral	
-Parieto-Occipital Cortex	16
-Subcortical WM	4
2. Brain Parenchyma Normal:	3

Table 2. Distribution of Cases According to Imaging Findings

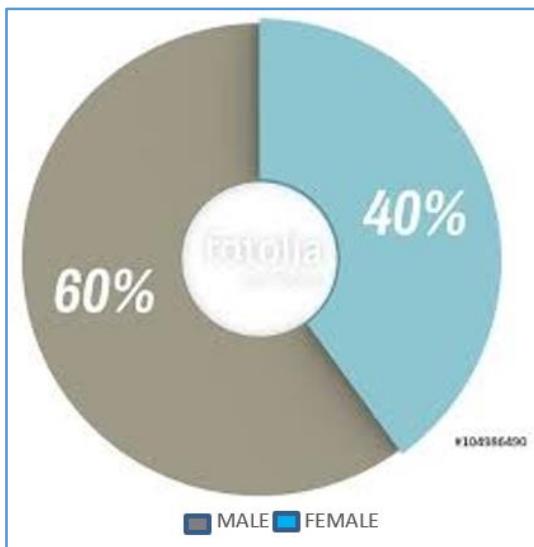


Figure 1. Distribution of Patients According to Sex

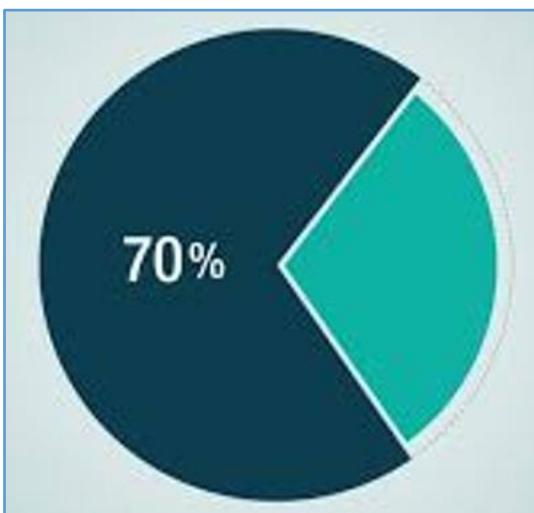


Figure 2. Distribution of Patients According to Age Group

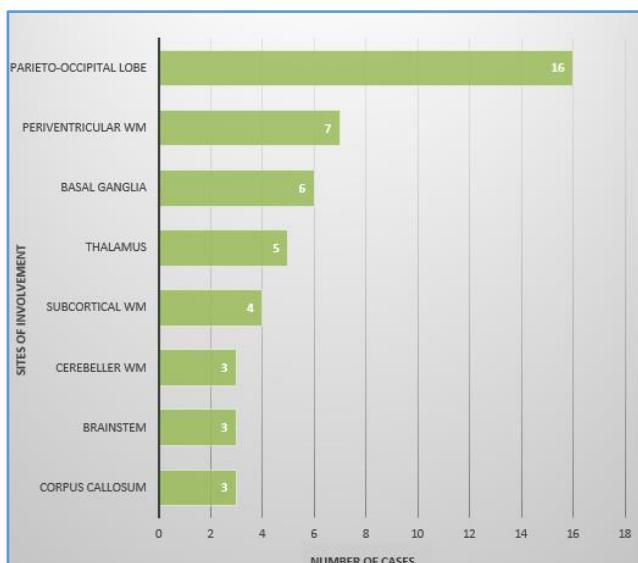


Figure 3. Distribution of Cases According to Sites of Involvement of Brain

Image Gallery: MRI Sequences Showing Parieto-occipital Involvement in Hypoxic Ischaemic Encephalopathy

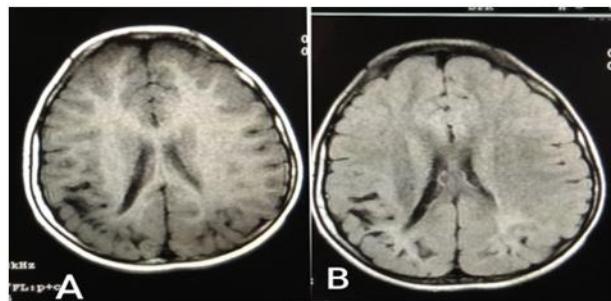


Figure 4 (A and B). T1 Weighted Axial Images and T2 Flair Showing Bilateral Parieto Occipital Involvement (Right > left)

(A and B)- T2 Weighted and FLAIR Axial Images Showing Parieto-occipital Involvement

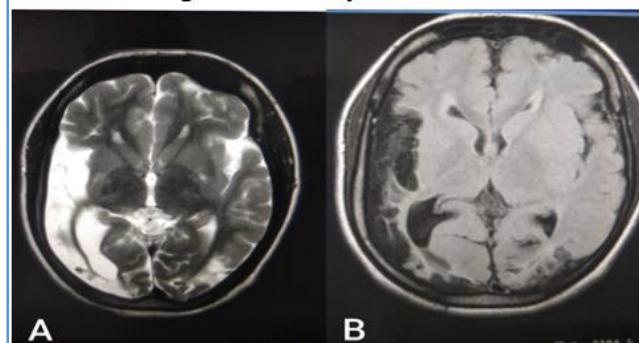


Figure 5 (A and B). T2 Weighted Images and T2 Flair Showing Predominantly Right Parieto Occipital Involvement.

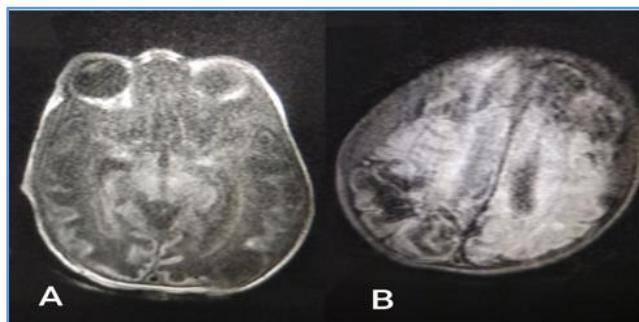


Figure 6 (A and B). T1 Weighted Images and T2 Flair Showing Marked Involvement of Right Parieto Occipital Lobe

DISCUSSION

MRI Pattern of Hypoxic Ischaemic Lesions^{16,17,18}

The subject literature describes three main MR patterns of hypoxic-ischemic lesions-

1. Periventricular leukomalacia– PVL,
2. Basal ganglia and/or thalamic lesions – BGTL,
3. Multicystic encephalopathy – MCE accompanied by injury to the basal ganglia, thalamus and/or cerebral cortex

Hypoxic-ischaemic encephalopathy (HIE) has varying anatomical patterns dependent on the type of insult, the degree and duration of cerebral hypoxia, or presence and degree of hypoperfusion. Profound insults can affect the entire cerebral cortex or just the periorolanic cortex, the cerebellum and the deep grey matter structures. Less severe insults may affect only the watershed regions^{19,20,21}

Mild to moderate hypoperfusion results in germinal matrix haemorrhages and periventricular leukomalacia in preterm neonates. Parasagittal watershed territory infarcts are seen in full-term neonates. Severe insult preferentially damages the deep grey matter in both term and preterm infants. However associated frequent periorolanic injury is seen in term neonates.^{22,23}

A study on brain injury pattern in neonatal encephalopathy published in American Journal of Neuroradiology in July 2013 found prolonged partial asphyxia results in a pattern of injury that primarily involves the watershed zones between the major intra vascular boundary zones, whereas acute profound asphyxia results in the basal ganglia-predominant pattern of brain injury that involves the basal ganglia, thalamus, brain stem, sensorimotor cortex and cortico-spinal tracts respectively.²⁴

In our study among 50 cases, we also found 16 cases with involvement of parieto-occipital cortex which is a predominant water-shed zone that correlates with the above studies.

In our study second most commonly involved area was periventricular WM, followed by basal ganglia, thalamus, subcortical WM, corpus callosum, brain-stem, cerebellar cortex in decreasing order of frequency.

A study published by Eman et al at dept. of Radiodiagnosis, Ain Shams University, Cairo in January 2016 on comparison between MRI and cranial USG in identification of HIE found thalamus to be most frequently involved.²⁵

Perinatal metabolic disease such as kernicterus and severe hypoglycaemia should be differentiated from classic HIE. Other conditions, such as infections, non-accidental injury and rarer metabolic diseases can be misinterpreted as HIE in their early course when diffuse brain swelling is still the predominant MRI feature. Diffusion techniques can help to differentiate different types of diffuse brain oedema.^{26,27}

CONCLUSION

From our study, we have come to a conclusion that parieto-occipital cortex (water-shed zone) has been most frequently involved area among the different sites that are usually involved in HIE.

REFERENCES

- [1] Lawn JE, Cousens S, Zupan J. 4 million neonatal deaths: when? Where? Why? *Lancet* 2005;365(9462):891-900.
- [2] Chao CP, Zaleski CG, Patton AC, et al. Neonatal hypoxic-ischaemic encephalopathy: multimodality imaging findings. *RadioGraphics* 2006;26 Suppl 1:S1-S3.
- [3] Lai MC, Yang SN. Perinatal hypoxic-ischemic encephalopathy. *J Biomed Biotechnol* 2011;2011:609813.
- [4] Allen KA, Brandon DH. Hypoxic ischemic encephalopathy: pathophysiology and experimental treatments. *Newborn Infant Nurs Rev* 2011;11(3):125-133.
- [5] Sie LT, van der Knaap MS, Oosting J, et al. MR patterns of hypoxic-ischemic brain damage after prenatal, perinatal or postnatal asphyxia. *Neuropediatrics* 2000;31(3):128-136.
- [6] Vannucci RC. Hypoxic-ischemic encephalopathy. *Am J Perinatol* 2000;17(3):113-120.
- [7] Maller AI, Hankins LL, Yeakley JW, et al. Rolandic type cerebral palsy in children as a pattern of hypoxic-ischemic injury in the full-term neonate. *J Child Neurol* 1998;13(7):313-321.
- [8] Okumura A, Kato T, Kuno K, et al. MRI findings in patients with spastic cerebral palsy. II: correlation with type of cerebral palsy. *Dev Med Child Neurol* 1997;39(6):369-372.
- [9] Fatemi A, Wilson MA, Johnston MV. Hypoxic-ischemic encephalopathy in the term infant. *Clin Perinatol* 2009;36(4):835-858.
- [10] Valk J, Vermeulen RJ, van der Knaap MS. Post-hypoxic-ischemic encephalopathy of neonates. In: van der Knaap MS, Valk J, eds. *Magnetic resonance of myelination and myelin disorders*. Springer-Verlag: Berlin Heidelberg 2005:718-748.
- [11] Flodmark O, Barkovich AJ. Imaging of the infant brain. In: Lagercrantz H, Hanson M, Evrard P, et al, eds. *The newborn brain*. Cambridge University Press 2002:289-316.
- [12] Sarnat H, Sarnat M. Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. *Arch Neurol* 1976;33(10):695-705.
- [13] Campistol J, Poo P, Fernandez Alvarez E, et al. Parasagittal cerebral injury: magnetic resonance findings. *J Child Neurol* 1999;14(10):683-685.
- [14] Pasternak JF, Gorey MT. The syndrome of acute near-total intrauterine asphyxia in the term infant. *Pediatr Neurol* 1998;18(5):391-398.
- [15] Triulzi F, Baldoli C, Righini A. Neonatal hypoxic-ischemic encephalopathy. In: Tortori-Donati P, Rossi A, Biancheri R, eds. *Pediatric Neuroradiology*. Brain. Springer: Berlin-Heidelberg 2005:234-255.
- [16] Cabaj A, Bekiesińska-Figatowska M, Mądzik J. MRI patterns of hypoxic-ischemic brain injury in preterm and full term infants – classical and less common MR findings. *Pol J Radiol* 2012;77(3):71-76.
- [17] Triulzi F, Parazzini C, Righini A. Patterns of damage in the mature neonatal brain. *Pediatr Radiol* 2006;36(7):608-620.
- [18] Rutherford M, Biarge MM, Allsop J, et al. MRI of perinatal brain injury. *Pediatr Radiol* 2010;40(6):819-833.

- [19] White ML, Zhang Y, Helyey JT, et al. Anatomical patterns and correlated MRI findings of non-perinatal hypoxic-ischaemic encephalopathy. *Br J Radiol* 2013;86(1021):20120464.
- [20] Rutherford M. The asphyxiated term infant. In: Rutherford M, ed. *TMR of the neonatal brain*. London-Toronto: W.B. Saunders 2002:99-128.
- [21] Barkovich AJ, Truwit CL. Brain damage from perinatal asphyxia: correlation of MR findings with gestational age. *Am J Neuroradiol* 1990;11(6):1087-1096.
- [22] Mutch L, Alberman E, Hagberg B, et al. Cerebral palsy epidemiology: where are we now and where are we going? *Dev Med Child Neurol* 1992;34(6):547-551.
- [23] Tong KA, Ashwal S, Obenaus A, et al. Susceptibility-weighted MR imaging: a review of clinical applications in children. *Am J Neuroradiol* 2008;29(1):9-17.
- [24] Wong DS, Poskitt KJ, Chau V, et al. Brain injury patterns in hypoglycemia in neonatal encephalopathy. *Am J Neuroradiol* 2013;34(7):1456-1461.
- [25] Genedi EAS, Osman NM, El-deeb MT. Magnetic resonance imaging versus transcranial ultrasound in early identification of cerebral injuries in neonatal encephalopathy. *Egyptian J of Radiology and Nuclear Medicine* 2016;47(1):297-304.
- [26] Cabaj A, Bekiesińska-Figatowska M, Mądzik J. MRI patterns of hypoxic-ischemic brain injury in preterm and full term infants – classical and less common MR findings. *Pol J Radiol* 2012;77(3):71-76.
- [27] Barkovich AJ, Hajnal BL, Vigneron D, et al. Prediction of neuromotor outcome in perinatal asphyxia: evaluation of MR scoring systems. *Am J Neuroradiol* 1998;19(1):143-149.