



SPECTRUM OF IMAGING FINDINGS IN HYPOXIC ISCHEMIC INJURY

Radio-Diagnosis

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ABSTRACT

Introduction: Perinatal asphyxia is a common cause for hypoxic ischemic encephalopathy that can lead to morbid neuronal outcomes, developmental delays or even death. Transcranial ultrasound is a commonly used modality to assess hemorrhage and hypoxic changes. Due to increased sensitivity in detecting cortical injury and less inter observer variability MRI is a better modality to detect hypoxic ischemic encephalopathy changes. **Aims and Objectives:** To enumerate the imaging spectrum in hypoxic ischemic injury **Material and Methods:** Data of 50 patients presenting to Department of Radio diagnosis and Imaging in Vydehi institute of Medical Sciences and research Centre, Bangalore with different symptoms and history of perinatal hypoxia who underwent MRI Brain were utilized. The data obtained was coded and entered in Microsoft Excel Spreadsheet. Further statistical analysis was done using SPSS version 20.0 software **Result:** Various findings were found in patients with history of perinatal history like Periventricular leukomalacia, subcortical white matter hyperintensities, Gliosis, Ventriculomegaly, Basal ganglia and Brain stem involvement, hemorrhage, Corpus callosal atrophy. **Conclusion:** The most common finding was periventricular and subcortical white matter changes and atrophy of the corpus callosum, followed by gliotic changes with ventriculomegaly. Cystic changes were seen in less number of people who has a more severe ischemic insult.

KEYWORDS

INTRODUCTION

Hypoxic-ischemic brain injury occurring in antenatal, perinatal or early postnatal period constitutes an important diagnostic problem in both prematurely born and term. Over the past several years magnetic resonance imaging (MRI) has become relatively easily accessible. On the basis of the central nervous system MRI, the experienced radiologist are able to determine the location of the hypoxic-ischemic lesions, their extent and evolution. Therefore they can help clinicians to answer the question whether the brain damage of the newborn is responsible for its clinical condition and they can contribute to determining the prognosis of the infant's future development.

Neonatal hypoxic-ischemic encephalopathy (HIE) is a devastating condition that may result in death or severe neurologic deficits in children. The pattern of brain injury depends on the severity and duration of hypoxia and degree of brain maturation.

Mild to moderate Hypoxic injury results in periventricular leukomalacia and germinal matrix bleed in preterm neonates, and parasagittal watershed infarcts in full-term neonates. Severe Hypoxic ischemic injury involves deep gray matter in both term and preterm infants. The current article reviews the etiopathophysiology and clinical manifestations of HIE, role of imaging in the evaluation of the condition, patterns of brain injury in term and preterm neonates.

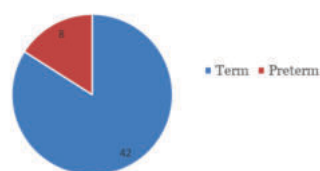
METHODS AND MATERIALS

A retrospective study of 50 patients presenting to Department of Radio diagnosis and Imaging in Vydehi institute of Medical Sciences and research Centre, Bangalore with different symptoms and history of perinatal hypoxia who underwent MRI Brain from Jan 2022 to October 2023.

RESULTS

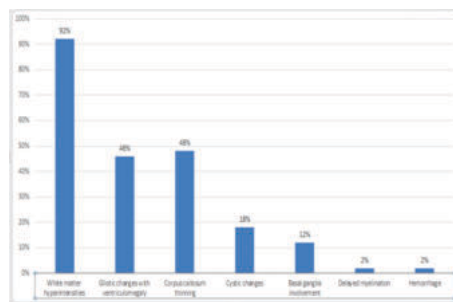
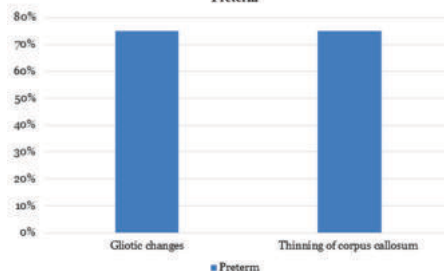
- Out of the 50 patients white matter hyper intensities with history of perinatal asphyxia and varied symptoms, 46 patients (92 %) had (periventricular and subcortical white matter).
- Gliotic changes with ventriculomegaly and corpus callosum thinning was seen in 23 (46 %) and 24 patients (48%) respectively.
- Cystic changes were seen in 9 patients (18 %).
- Basal Ganglia involvement was seen in 6 patients (12%).
- Delayed myelination and haemorrhage was seen in one patient each(2%).
- 8 out of the 50 patients had history of preterm delivery in whom gliotic changes and thinning of corpus callosum were a more common finding (75 %).

Term VS Preterm



FINDINGS	NO OF PATIENTS
White matter hyperintensities	46
Basal ganglia involvement	6
Hemorrhage	1
Delayed myelination	1
Ventriculomegaly with gliosis	23
Cystic changes	9
Thinning of corpus callosum	24

Preterm



DISCUSSION

Imaging findings in hypoxic ischemic encephalopathy are highly variable and depend on a number of factors, including brain maturity, severity and duration of insult, and type and timing of imaging studies. MR imaging is an accurate modality for evaluating neonatal HIE. DWI is a promising technique for early detection of PVL, before any abnormality appears on conventional MRI. Diffusion-weighted images will demonstrate increased signal intensity in the region of the ventrolateral thalami and basal ganglia (particularly the posterior putamina), in the peri rolandic regions, and along the corticospinal tracts.

Diffusion-weighted MR imaging performed during the first 24 hours will often lead to underestimation of the ultimate extent of injury, indeed, normal findings at diffusion weighted imaging (prominent role of apoptosis) performed in the first 24 hours, although uncommon, have been reported. Abnormalities seen at diffusion weighted imaging generally peak at 3–5 days and subsequently “pseudo normalize” by about the end of the 1st week, although decreased ADC values can persist in injured regions into the 2nd week.

Perinatal hypoxic insult may result in hypoxic ischemic encephalopathy (HIE), which is a devastating condition that may result in death or severe neurologic deficits in children.

Neuroimaging with cranial ultrasound (US), computed tomography and magnetic resonance imaging are used to assess patients with HIE. Investigation of choice for the detection of these lesions is transcranial ultrasound, as it is non-invasive, readily available and reproducible, but it may only reveal the severe hypoxic-ischemic lesions and may have inter observer variability.

The pattern of brain injury depends on the severity and duration of hypoxia and degree of brain maturation. Mild to moderate HI injury results in periventricular leukomalacia and germinal matrix bleed in preterm neonates, and parasagittal watershed infarcts in full-term neonates. Mild to moderate HI injury results in Periventricular leukomalacia, that may be focal or diffuse. Severe HI injury involves deep gray matter in both term and preterm infants.

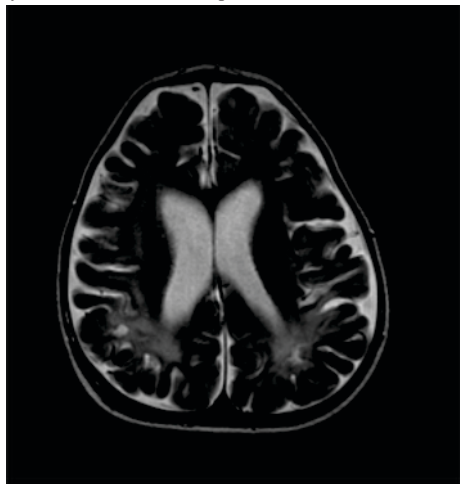


Figure 1: MRI Axial T2w Image Demonstrating Periventricular Leucomalacia

Germinal matrix hemorrhage (GMH) is typically seen in preterm infants with HI injury. Subsequent reperfusion to ischemic brain tissue may result in GMH from rupture of weak capillaries and increased venous pressure.

Thalami, brainstem, and cerebellum in the immature brain have high metabolic activity and hence are more susceptible to injury in severe HI injury. Acute and severe hypoxia in term infants results in damage to the gray matter, especially the basal ganglia and thalamus. Frequently the signal from the cerebral cortex and white matter is also altered. Extensive multicystic involvement of white matter, quite often accompanied by necrotic cavities within the basal ganglia and thalamus is typical for a prolonged and severe hypoxia.

Parasagittal brain injury is diagnosed less frequently than selective

neuronal necrosis and is characteristic for mild or moderate, subacute hypoxia. It is located on the border of the vascular territories – parasagittally, bilaterally and includes the cerebral cortex and the adjacent white matter.

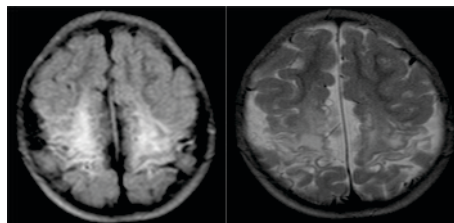


Figure 2: MRI Axial FLAIR and T2w Image Subcortical White Matter Intensities.

In preterm neonates periventricular white matter is most vulnerable to the HI injury. Progressive necrosis results in loss of periventricular white matter, passive ventriculomegaly (irregular margins of the bodies and trigones of lateral ventricles) and thinning of corpus callosum. Cavitation and cyst formation may occur in end-stage PVL and is best seen on FLAIR sequence. Periventricular cysts are usually $\leq 2-3$ mm, larger cysts have poorer prognosis. Gliosis, porencephalic cyst, dilated ventricle, neuroparenchymal volume loss and non visualization of posterior body and splenium of corpus callosum.

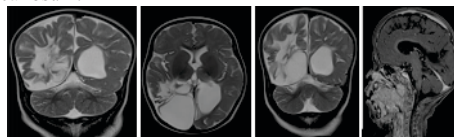


Figure 3: MRI coronal and axial images showing gliosis, porencephalic cyst, dilated ventricle, neuroparenchymal volume loss and non visualization of posterior body and splenium of corpus callosum.

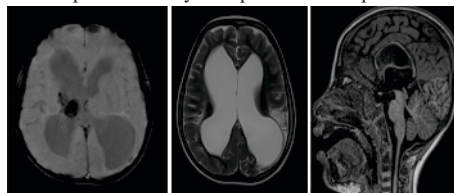


Figure 4: MRI axial SWI and T2 axial and sagittal Chronic haemorrhage in right thalamus, Hydrocephalus with volume loss and gliotic changes, thinning of corpus callosum.

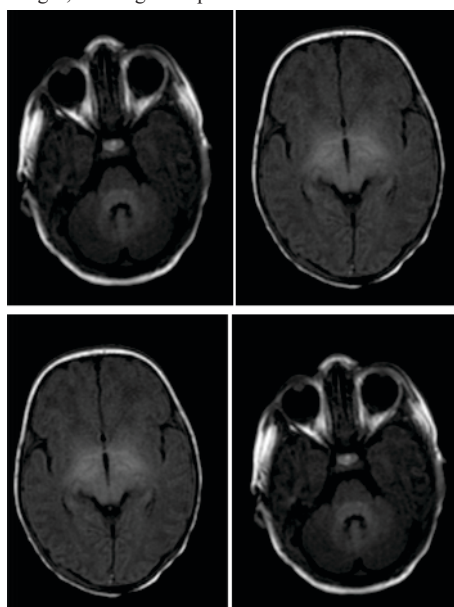


Figure 5: MRI FLAIR images showing symmetrical altered signal intensities in bilateral perirolandic regions, putamen and thalamus & also in the mid brain, pons and upper medulla

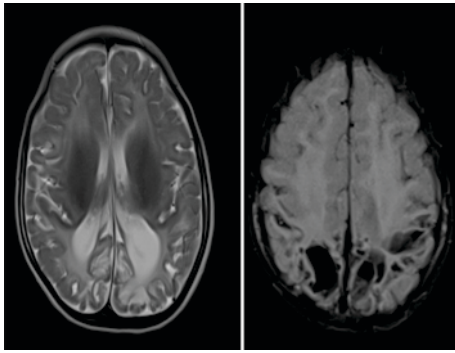


Figure 6: MRI T2 and FLAIR axial images showing Gliosis, porencephalic cyst, dilated ventricle, neuroparenchymal volume loss and non visualization of posterior body and splenium of corpus callosum.

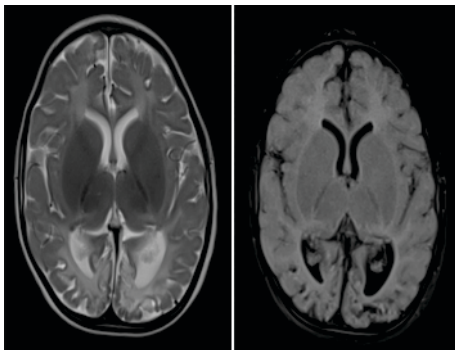


Figure 7: MRI T2 and FLAIR axial images Volume loss with cystic encephalomalacic changes in bilateral parietal and occipital lobes. Confluent T2W/FLAIR white hyperintensities in all visualised lobes - S/o delayed myelination.

CONCLUSION

- Hypoxic ischemic injury can pose a difficult diagnostic problem from a neuroimaging standpoint, especially in newborns.
- MRI is a sensitive, non-invasive modality with no radiation exposure that can be used to assess hypoxic ischemic lesions in patients with history of perinatal asphyxia as it has excellent grey white matter resolution and multi planar imaging capabilities.
- The most common finding was white matter hyperintensities, followed by corpus callosum thinning and gliotic changes with ventriculomegaly.
- Cystic changes, basal ganglia involvement, haemorrhage and delayed myelination were seen in lesser number of cases.

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