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Original Research Paper

MRI in evaluating perinatal asphyxia – A closer look and a retrospective analysis

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ABSTRACT:
Perinatal asphyxia is the significant cause of long-term morbidity and mortality in neonates and contributes 20% in the causes of neonatal mortality in India. Asphyxia in the perinatal period is the most important cause of hypoxic ischemic encephalopathy (HIE). Due to difference in maturity at the time of hypoxic insult and extent of the insult, HIE presents with different MRI patterns. MRI is the most sensitive investigation in evaluation of these patterns of brain injury with additional significant role of Diffusion Weighted Imaging (DWI). Due to recent advances in MRI, it has become easy in determination of location of the injury, its extent and the evolution.

Keywords: Hypoxic ischemic encephalopathy (HIE), Magnetic Resonance Imaging (MRI), perinatal asphyxia, multi-cystic encephalomalacia, ulegyria.

INTRODUCTION:

Neonatal hypoxemic-ischemic encephalopathy resulting from perinatal asphyxia is one of the most common causes of cerebral palsy and other severe neurological deficits in children. It occurs in 1.5/1000 live births. Perinatal asphyxia is the most significant risk factor for HIE. Almost 20% of neonatal deaths in India are caused by perinatal asphyxia. It may occur either in utero or postnatally [1].

Hypoxia and hypercapnia result in reduced cerebral circulation which causes series of events including acidosis, release of inflammatory mediators, generation of free radicals and lipid peroxidation which result in impairment of cell metabolism, reperfusion and necrosis [4].

HIE presents differently depending upon the time of insult. There are three main MRI patterns of HIE; including basal ganglia and/or thalamic lesions, periventricular and subcortical leukomalacia, multi-cystic encephalomalacia [3],[8]. MRI with its extra advantage of Diffusion Weighted Imaging (DWI) plays an important role in identification, characterization, location and extent of the brain injury [7]. The knowledge of different MRI features of HIE helps clinicians to estimate the prognosis and in planning appropriate treatment based on the degree of motor impairment [9], [10].

AIM AND OBJECTIVES:

AIM:

- To retrospectively analyse the utility of MRI as a method of imaging to diagnose patients with hypoxic ischemic encephalopathy.

OBJECTIVES:

- To review the different MRI patterns of hypoxic ischemic encephalopathy.
- To classify different patterns on the basis of timing of the hypoxic ischemic insult.

MATERIALS AND METHODS:

- This is a retrospective study conducted in department of Radiodiagnosis, Mahatma Gandhi Mission Institute of Health Sciences, Navi Mumbai over a period of 1 year from December 2022 to December 2023 after receiving permission from concerned authorities (Head of the Department of Radiodiagnosis and Hospital Director) of MGM Hospital, Navi Mumbai.
- MRI records of total 25 patients of paediatric age group with history of perinatal asphyxia

presenting with chief complaints of seizure disorder, global developmental delay and cerebral palsy were assessed showing different features on MRI imaging.

- The patients' ages ranged from 5 days to 10 years All patients were scanned on TOSHIBA 1.5T MRI Machine.

DISCUSSION:

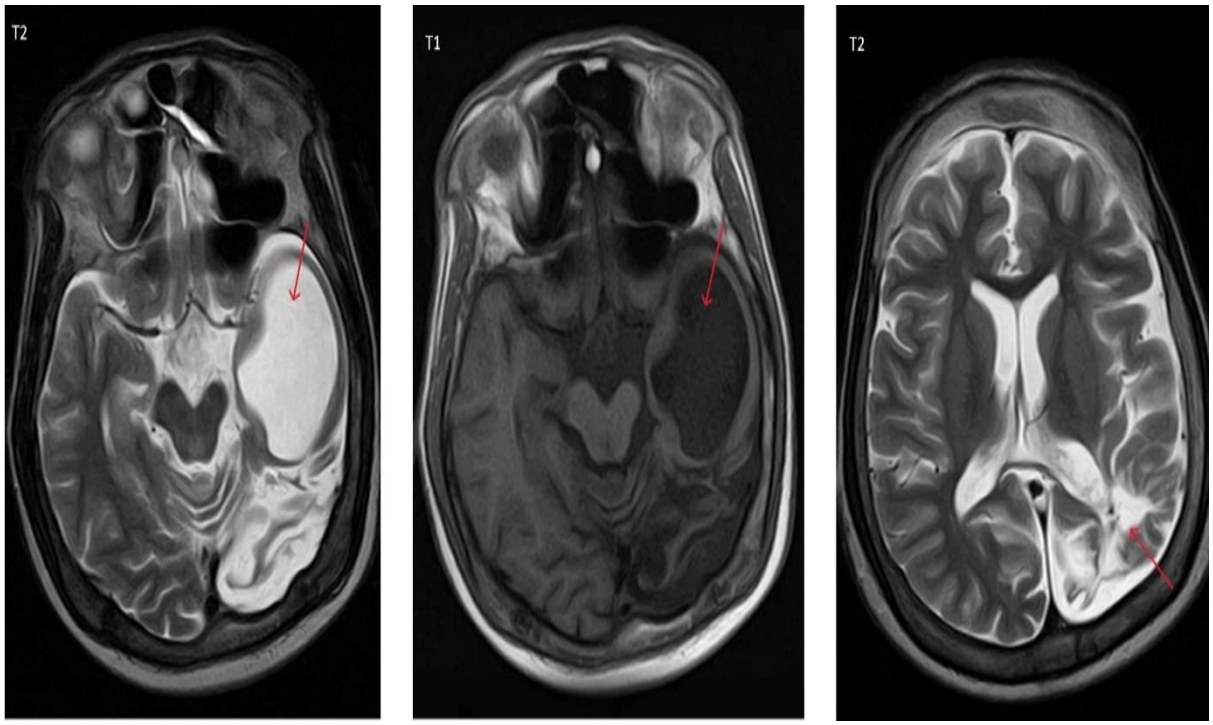
The patterns of hypoxic ischemic encephalopathy on MRI depends on the time of insult and are as follows [2], [3], [4], [5], [6], [7]:

1. **Before 28 weeks**:
The immature brain is not able to react with gliosis so it undergoes liquefaction [2], [7]. It presents with a parenchymal defect and enlargement of CSF spaces. On MRI it presents with Porencephalic cyst or hydranencephaly.

CASE-1:

A 10-year-old male patient presented with history of preterm delivery, birth asphyxia, seizures, inappropriate behaviour and mental retardation.

Figure:1



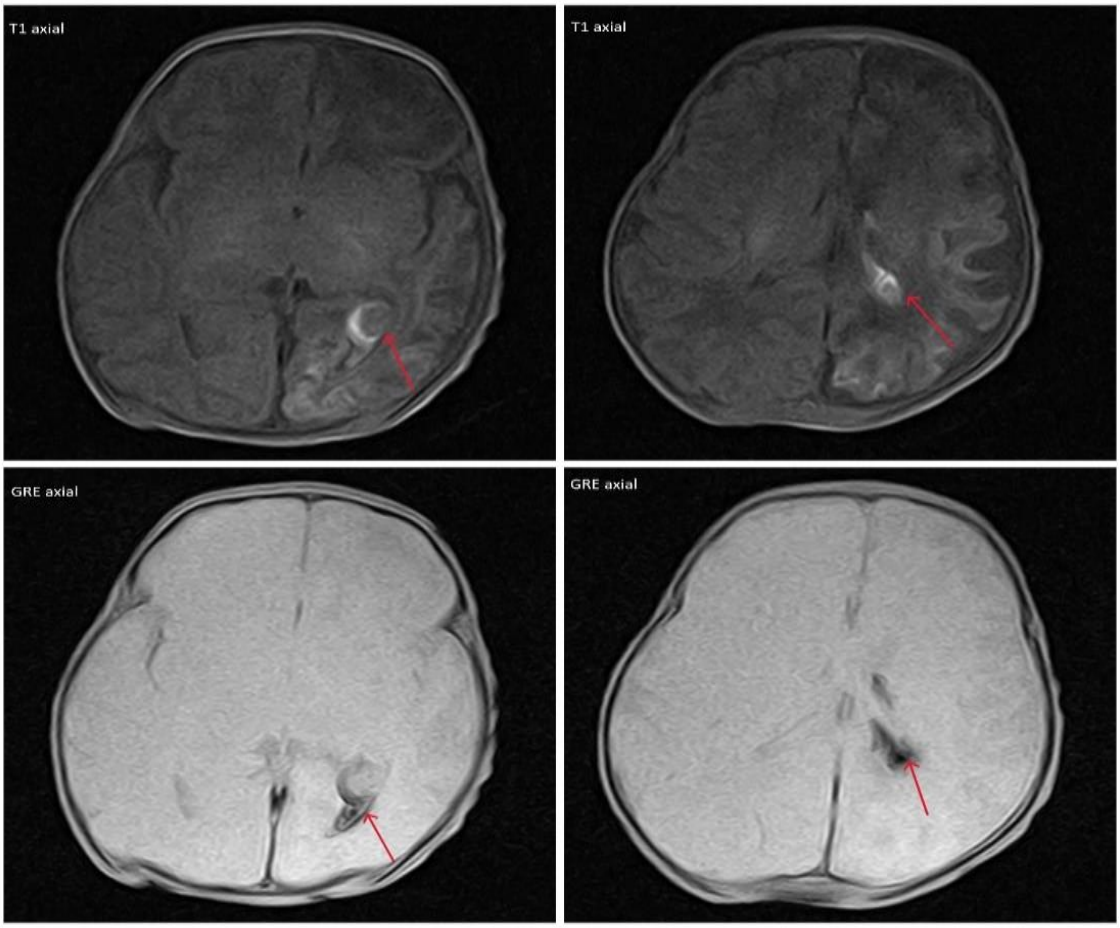
CSF signal intensity cystic lesion was noted in left temporal lobe with surrounding encephalomalacia involving left parieto-occipito-temporal lobes – features were suggestive of Porencephalic cyst.

2. **28 to 32 weeks**:

The prematurity and the number of capillaries in the germinal matrix show direct relationship and the insult in this period of time presents with germinal matrix or intraventricular haemorrhage.

CASE-2:
A 5-days-old male preterm new-born with history of birth asphyxia and neonatal seizures.

Figure-2:

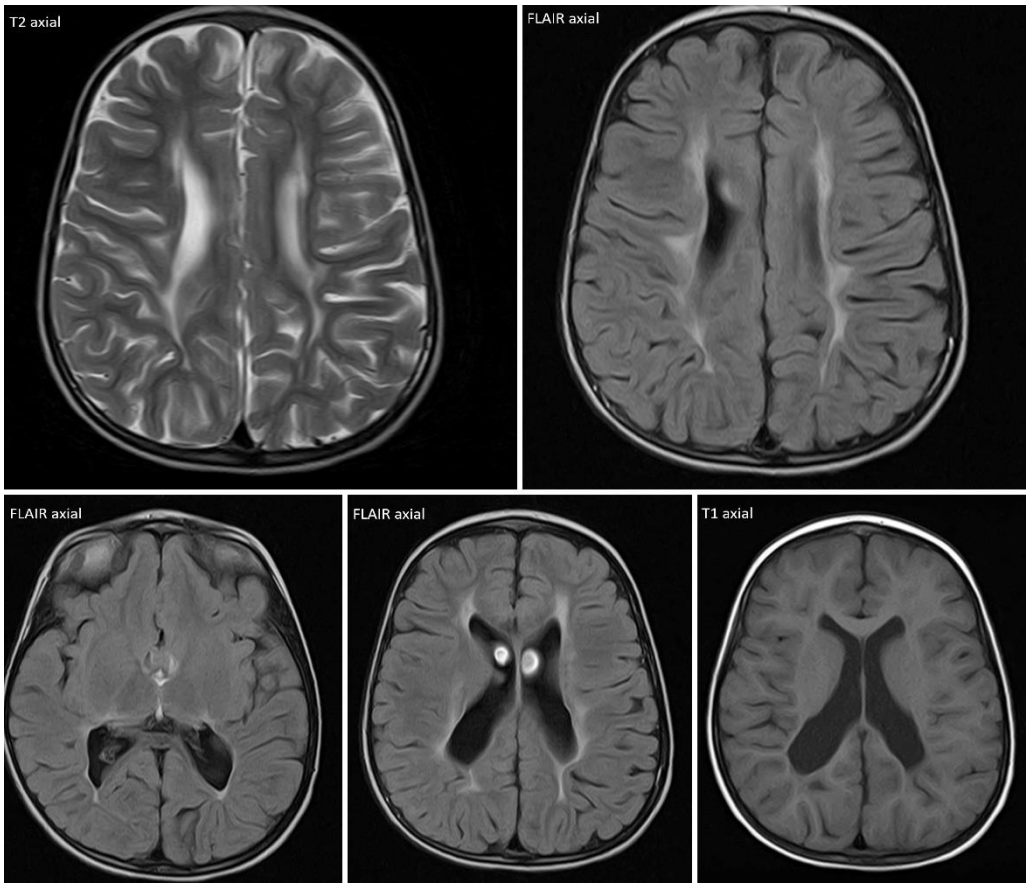


Intraventricular haemorrhage involving the body and occipital horn of left lateral ventricle appearing hyperintense on T1W images and showing blooming on GRE. There was also marked atrophy of left cerebral hemisphere with cortical laminar necrosis.

3. **32 to 36 weeks:**
   Periventricular leukomalacia: Partial asphyxia results in coagulation necrosis with white matter loss and gliosis occurs involving the white matter in the periventricular and subcortical areas. Typically, the lesions are symmetrical.

CASE-3:
A 4-year-old female patient with history of birth asphyxia presented with complaints of global developmental delay.

Figure-3:



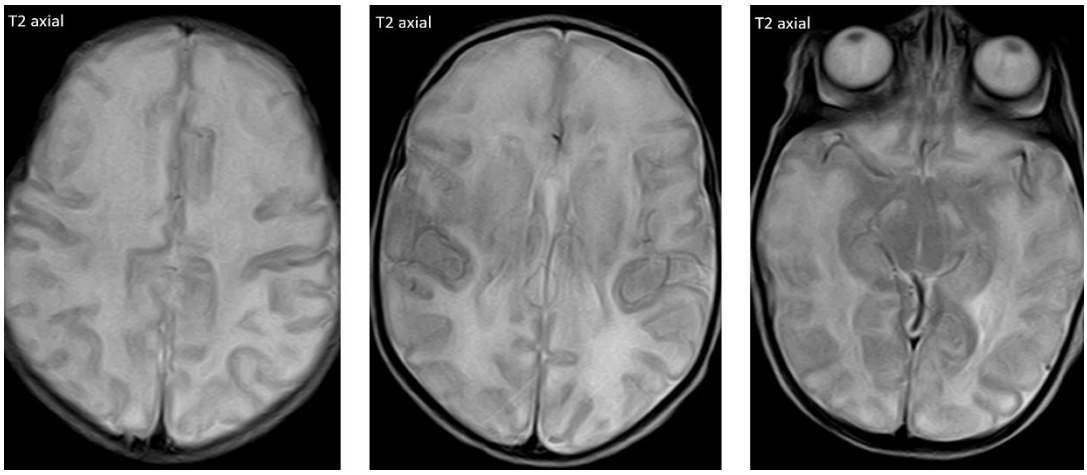
Symmetrical T2W and FLAIR hyper intensities were noted involving the bilateral periventricular white matter with mild prominence of bilateral lateral ventricles. The margins of bodies and trigones of bilateral lateral ventricles appear irregular with volume loss of periventricular white matter.

4. Term new-born:

Basal ganglia- thalamus pattern: Acute profound asphyxia results in lesions in high oxygen demand areas. It involves ventrolateral thalami, posterior putamen and cortical spinal tracts from peri-Rolandic cortex to posterior limb of internal capsule [3].There is strong association between the degree of basal ganglia involvement and the severity of motor impairment.

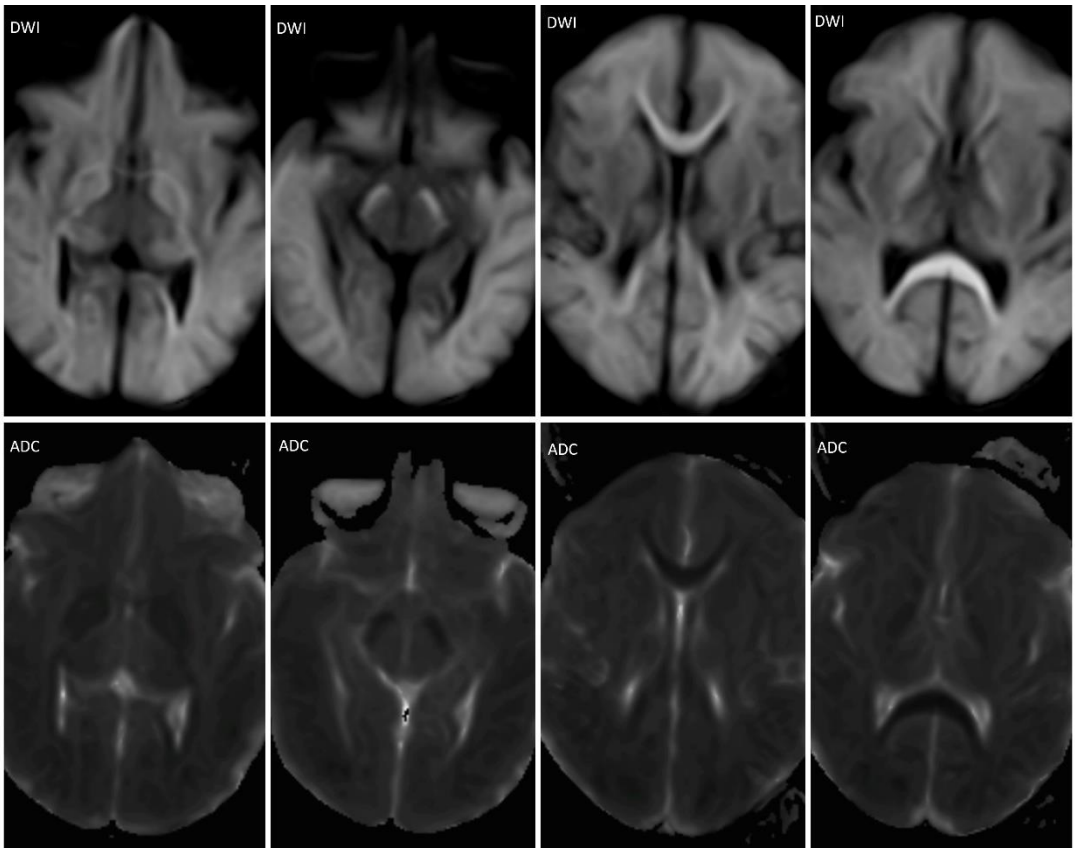
CASE-4:
A 10-days-old male patient with history of severe birth asphyxia presented with multiple episodes of convulsions, poor activity and hypotonia.

Figure-4:



Symmetrical T2W hyper intensities were noted involving the white matter of bilateral frontal, parietal, temporal, occipital lobes showing restricted diffusion on DWI.

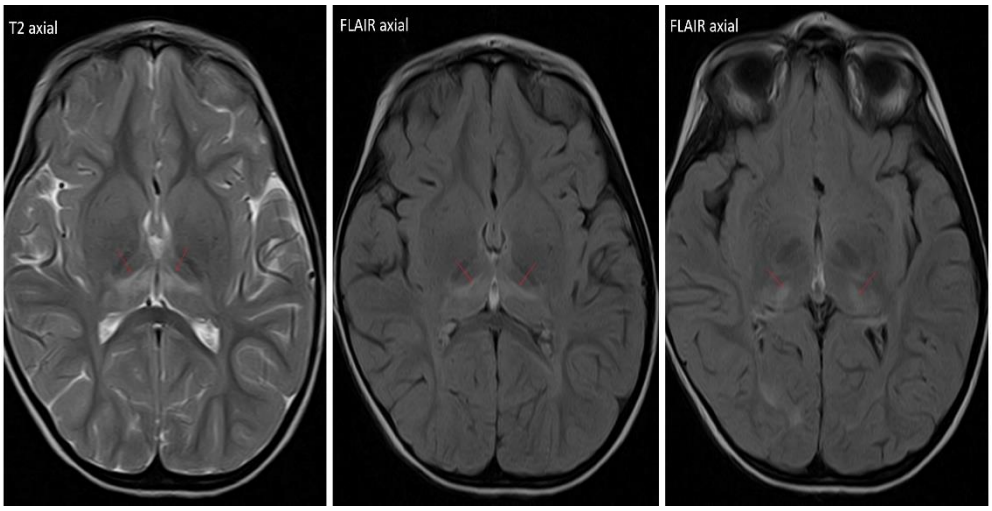
Figure-5:



Restricted diffusion with corresponding signal drop on ADC was also noted involving the bilateral basal ganglia, bilateral thalami, bilateral cortico-spinal tracts along the internal capsule and cerebral peduncles and the entire corpus callosum.

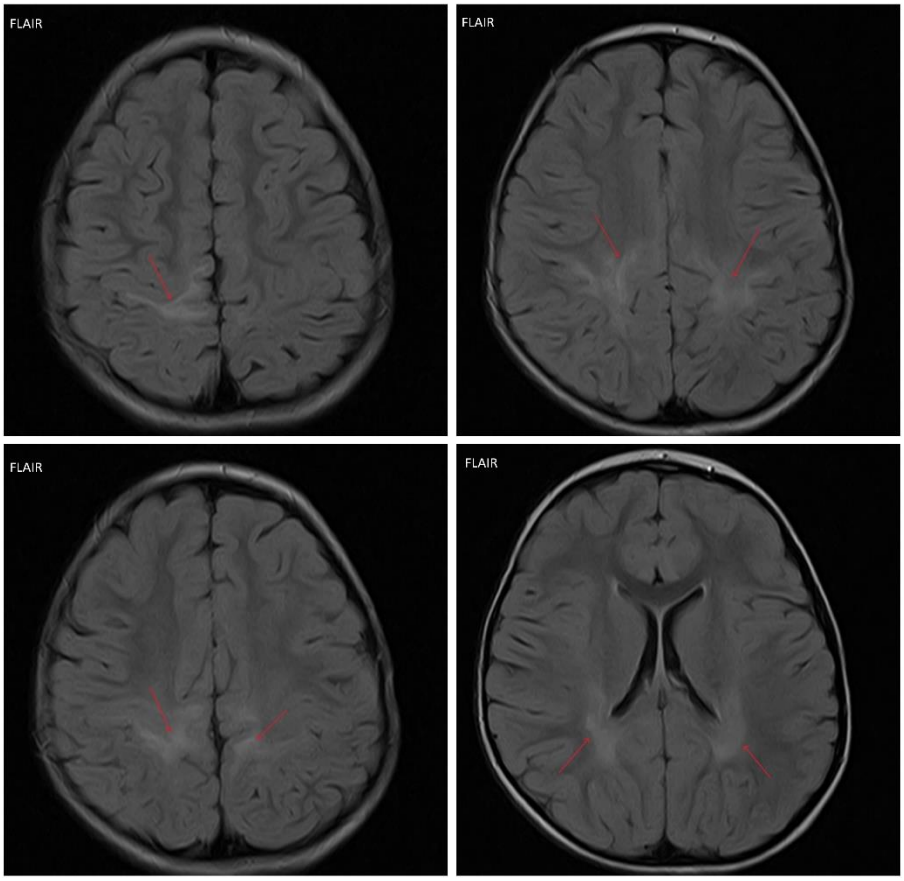
CASE-5:
A 4-year-old female patient with history of birth asphyxia and history of NICU admission presented with complaints of seizure and developmental delay.

Figure-6:



Symmetrical T2W and FLAIR hyperintense areas involving the bilateral thalami.

Figure-7:

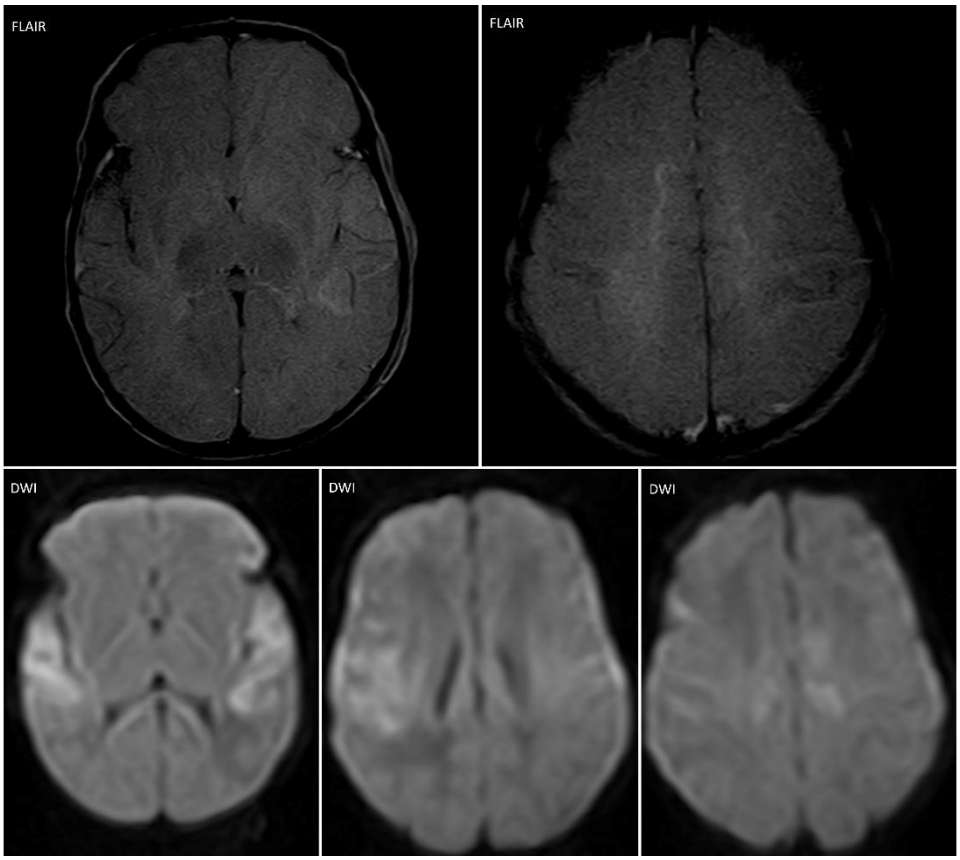


Symmetric T2W and FLAIR hyper intensities were also noted involving the subcortical, periventricular and deep white matter of the bilateral parietal lobes and the peri-Rolandic white matter.

Cortical-subcortical white matter injury: Prolonged partial asphyxia results in cortical-subcortical lesions in watershed areas with parieto-occipital and posterior temporal lobes affected more often than the frontal lobes [3]. Severe motor impairment is uncommon in these group of patients and often show normal outcome.

CASE-6:
A 1-year-old male patient with history of birth asphyxia presented with complaints of delayed milestones and seizure.

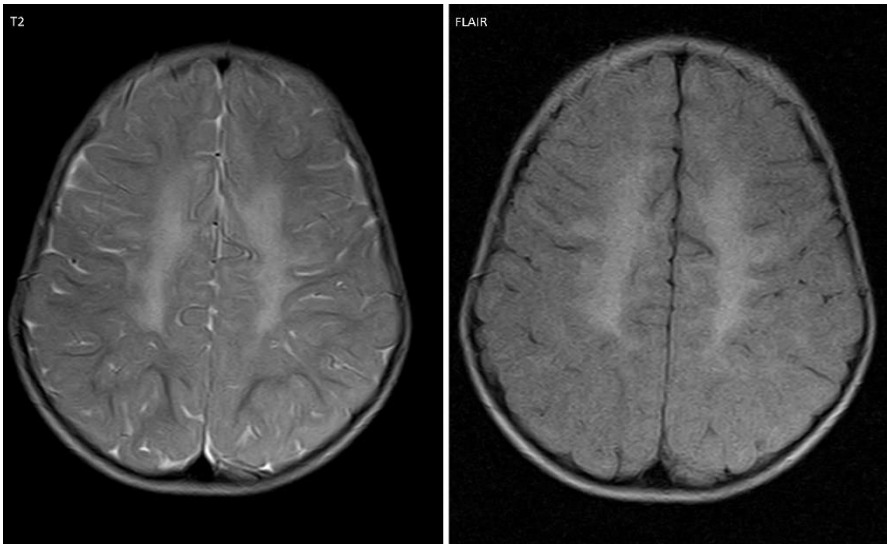
Figure-8:



Symmetric FLAIR hyper intensities involving bilateral temporal and parasagittal areas of bilateral fronto-parietal lobes with areas of restricted diffusion involving the bilateral para-falcine fronto-parietal lobes (watershed) and peri-sylvian area of bilateral tempo-parietal lobes.

CASE-7:
A 1-year-old male patient with history of birth asphyxia and NICU admission came with chief complaints of developmental delay.

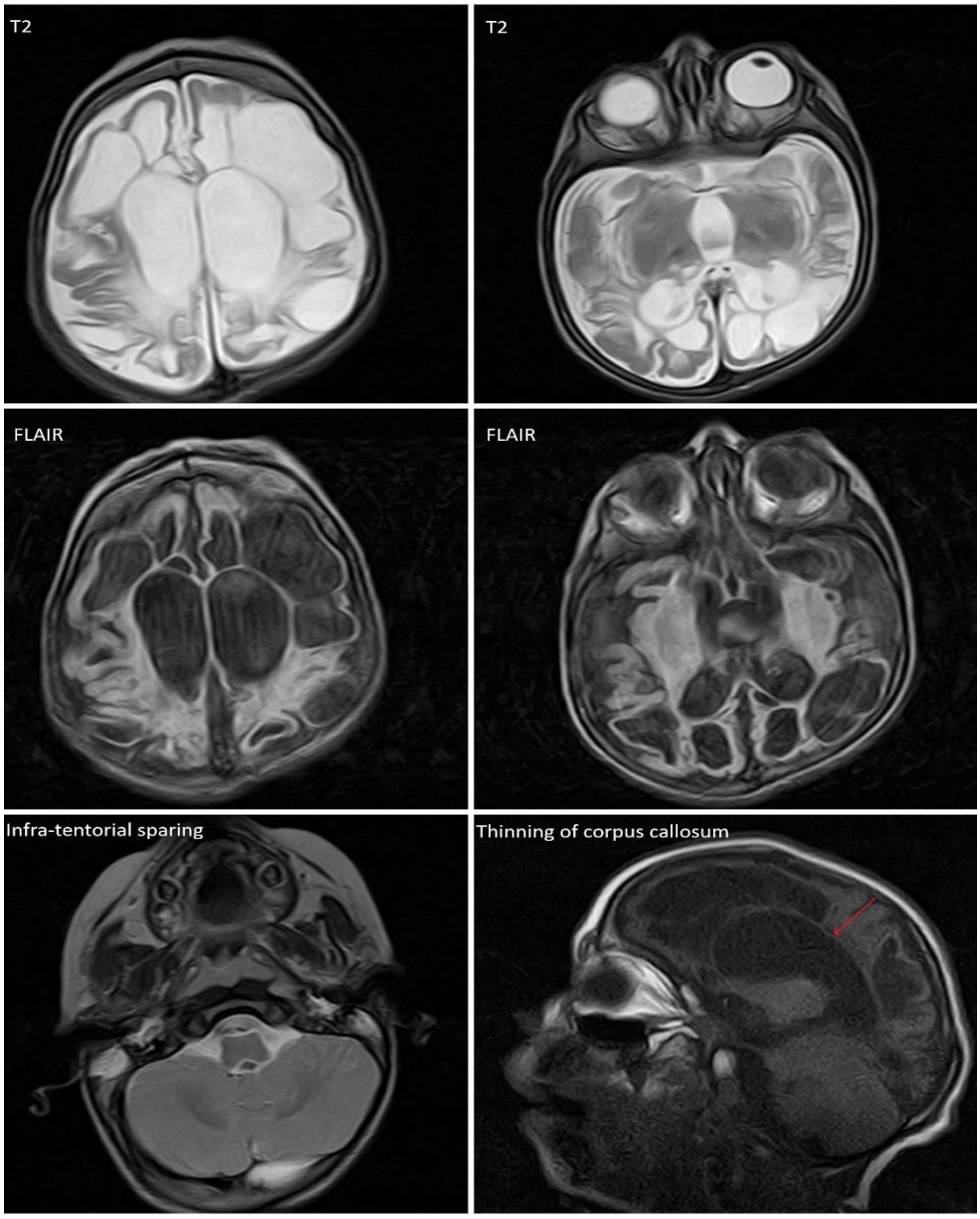
Figure-9:



Symmetric confluent T2/FLAIR hyperintense areas involving cortical and subcortical white matter of bilateral para-falcine fronto-parietal lobes (watershed).
Multi-cystic encephalomalacia: Profound asphyxia results in liquefactive necrosis of brain causing its softening, cystic degeneration and surrounding gliosis [6].

CASE-8:
A 1-year-old male patient with history of birth asphyxia presented with rolling of neck behind and tightness of limbs since birth.

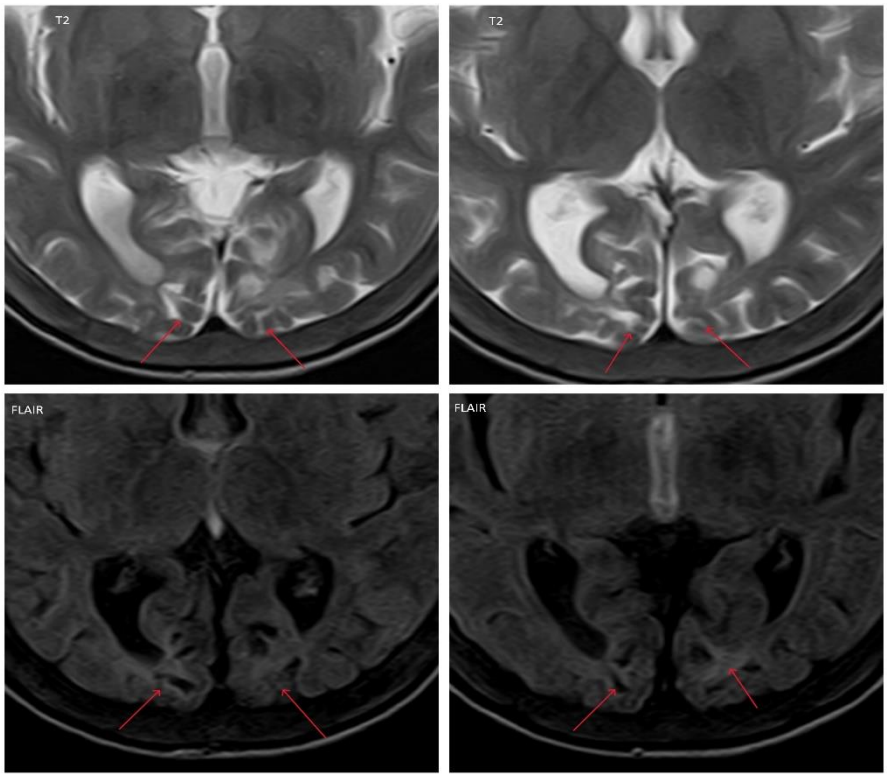
Figure-10:



Multiple cysts of variable size and shape situated bilaterally in the supra-tentorial cerebral white matter and inner cortex with relative sparing of bilateral basal ganglia. There was associated irregular ex-vacuo dilatation of bilateral lateral and third ventricles. There was sparing of infra-tentorial region including cerebellum and brainstem with significant thinning of corpus callosum (red arrow).
Ulegyria: Prolonged asphyxia also leads to atrophy and flat shrunken cortex, thinning of gyral stems with widening of sulci at the deep end.

CASE-9:
A 10-year-old male patient a known case of cerebral palsy with history of birth asphyxia.

Figure-11:



Cortical thinning with prominence of adjacent cortical sulci and underlying subcortical T2W and FLAIR hyper intensities were noted involving the bilateral para-median occipital lobes representing focal atrophy with underlying gliosis – ulegyria.

The treatment of HIE is primarily supportive with correction of the underlying cause. The supportive care includes maintenance of adequate ventilation, metabolic status and vitals, and control of brain edema and seizure [11], [12], [13].

RESULT:

MRI records of 25 patients (11 females and 14 males) with history of birth asphyxia were reviewed and different MRI patterns of hypoxemic ischemic encephalopathy were studied. Their ages ranged from 5 days to 10 years. Out of 25 patients, 1 patient showed porencephalic cyst on MRI, 02 patients showed intraventricular haemorrhage, 02 patients showed basal ganglia-thalamus pattern, 08 patients showed periventricular leukomalacia, 06 patients showed cortical-subcortical white matter involvement, 04 patients showed changes of multi-cystic encephalomalacia and 02 patients showed ulegyria.

CONCLUSION:

MRI remains the best investigation in diagnosis of the hypoxic ischemic encephalopathy with high prognostic value. There are different patterns of signal changes on MRI depending upon the time, duration and severity of the insult and the brain maturation. The typical patterns include basal ganglia-thalamus pattern, periventricular leukomalacia and the cortical-subcortical white matter injury resulting into subsequent encephalomalacia and gliosis. The knowledge of these different MRI patterns helps radiologists in identification, characterization, localization and extent of the insult, determination of the severity, its correlation with the degree of motor impairment, estimating the prognosis and guiding the clinician to plan for appropriate treatment and rehabilitation.

Early diagnosis and timely intervention are the main objective in management of suspected HIE. Treatment is mainly supportive including adequate ventilation, metabolic status, vitals and control of seizures. Prognosis depends on severity and gestational age. Term infants with mild injury show good prognosis and complete recovery, however 20% of infants may die in neonatal period and another 25% may develop significant neurological deficit. Preterm infants have poor prognosis.

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