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Radiology

KEYWORD:
Hypoxic ischemic encephalopathy, preterm, term, periventricular leukomalacia, posterior limb of internal capsule.

SPECTRUM OF MRI FINDINGS IN HYPOXIC ISCHEMIC ENCEPHALOPATHY PATIENTS

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ABSTRACT

INTRODUCTION: Hypoxic-ischemic brain injury is one of the most devastating of all brain insults. The incidence of HIE is 2.5 per 1000 term live births and approximately 7 per 1000 preterm births. Imaging findings in perinatal HII vary with insult severity and duration of hypo perfusion event, state of brain maturation, metabolic demands of key anatomic structures, state of maturity of glutamate receptors and gestational age. Patterns of injury in preterm includes profound or central, perinatal white matter damage, germinal matrix-intraventricular haemorrhage, periventricular hemorrhagic infarction and cerebellar injury. Aims of this study are to see the different pattern/distribution of injury in preterm and term born patients.

MATERIAL AND METHOD: This retrospective study of 100 patients was conducted at Gajra Raja Medical College, Gwalior, Madhya Pradesh in one year duration. Distribution of injuries in term born patients were watershed areas (36.1%), cortex (8.3%), periventricular white matter (34.7%), basalganglia and thalami (16.7%), brainstem (1.4%), cerebellum (2.8%), germinal matrix and intraventricular (0%) and diffuse involvement (0%). Distribution in preterm born patients were watershed areas (32.2%), basal ganglia and thalami (25%), periventricular white matter (17.9%), germinal matrix and intraventricular (10.7%), cortical (7.1%), diffuse involvement (7.1%), brainstem (0%) and cerebellum (0%). Other associated findings include microcephaly, ulegyria, corpus callosum thinning, intra or extra axial or scalp collections, congenital anomalies and intracranial infections.

CONCLUSION: Most common location involved in term and preterm born patients of our study is watershed areas. Most common associated imaging findings were corpus callosum thinning followed by ulegyria. Other significant associated imaging findings are microcephaly, collections, congenital anomalies and infections.

INTRODUCTION:

Hypoxic-ischemic brain injury is one of the most devastating of all brain insults. The incidence of HIE is 2.5 per 1000 term live births[1] and approximately 7 per 1000 preterm births[2]. Despite improvements in perinatal care, hypoxic–ischemic injury (HII) results in 23% of world's neonatal deaths[3] and causes permanent neurological deficits in 25% of the affected term neonates[4].

Cerebral ischemia is simply diminished blood flow. Ischemia can be focal or global. Focal ischemia refers to decreased or absent perfusion in a particular vascular territory, usually secondary to arterial stenosis or occlusion. Ischemia may or may not proceed to frank infarction. Global ischemia occurs when overall cerebral perfusion drops below the level required to maintain normal brain function. Hypoxia or hypoxemia refers to reduced blood oxygenation. In contrast to ischemia, cerebral hypoxia is almost always global. In the initial stages of hypoxia, cardiac output and cerebral blood flow (CBF) may be maintained normally but blood oxygenation is deficient. Prolonged systemic hypoxemia results in cardiac hypoxia, which in turn diminishes output. Depressed cardiac output eventually causes global brain hypo-perfusion and ischemia[5].

The term global hypoxic-ischemic injury (HII) is used to describe the pathologic and imaging findings of CNS hypoxia with or without global (not focal) brain ischemia. In practice, both factors often act in concert. In asphyxia, brain injury is a consequence of ischemia superimposed on hypoxia. Pure hypoxia in the absence of ischemia generally does not cause frank brain necrosis unless the hypoxic state is prolonged.

MRI is the most sensitive and specific modality for HIE even though there are other modalities used to diagnose like ultrasonography (USG) and computed tomography (CT). USG remain the first investigation of choice because of the cost advantage, portability and availability. USG can detect haemorrhage, cystic encephalomalacia, periventricular leukomalacia (PVL) and hydrocephalus. It is less reliable in PVL and cerebral edema[6]. Computed Tomography is the less commonly used modality for evaluation of HIE because of poor parenchymal contrast resolution and radiation exposure.

Imaging findings in HII are highly variable. The effect of HII on the mature brain of older children and adults differs significantly from its impact on the developing brain. Other factors such as insult duration and severity as well as timing of studies relative to onset also affect the imaging appearance of global cerebral HII.

CYTOTOXIC EDEMA:

One concept that is helpful to understand the changes that are seen in stroke is the development of cytotoxic edema associated with ischemia. When regional cerebral blood flow (CBF) decreases below 15 to 18 mL/100 g per minute, electrical activity within human neurons ceases[6,7]. With further decreases in CBF the associated decrease in availability of oxygen and glucose results in decreased production of adenosine triphosphate (ATP)[8,9].

With the loss of ATP production, Na+/K+ ATPase an enzyme that is important to cell homeostasis fails. This permits the unbalanced influx of extracellular calcium and sodium and secondarily the influx of extracellular water into cells. This increased intracellular water is termed cytotoxic edema.

The formation of cytotoxic edema is postulated to be the cause of most CT and MRI findings seen in acute ischemic stroke.

Magnetic Resonance Imaging Technique, Protocol And Basics Of Interpretation:

MR imaging examination was indicated for medical reasons and MR spectroscopy was considered a part of the MR

examination. An appropriate size neonatal head coils used. Signal-to-noise ratio (SNR) increases, as the diameter of the coil decreases. As there is higher water content, lower protein and lipid contents of neonatal brain, the standard MR sequences used in an adult brain should be optimized for use in neonates. It is achieved by increasing the repetition time (TR) of both T1 and T2 weighted images (WI). Ideally for T1WI, the standard TR (400-500 ms) is increased to 800-850 ms and for T2WI the standard TR (3500-5000 ms) is increased to 9000-10000 ms[10]. This optimizes the gray-white differentiation and SNR of the images.

MRI images accompanying this article was done in a Philips-Ingenia 1.5-Telsa dstream scanner. T1 and T2 WI in axial plane, T2WI in coronal and sagittal planes, Diffusion WI (DWI) in axial plane with apparent diffusion coefficient (ADC) map generation, T2* images (gradient-echo and SWI) and fluid-attenuated inversion recovery (FLAIR) in axial plane are the key protocol employed. Axial T1WI is ideal for detecting myelination, ischemia, subacute hemorrhage and abnormal anatomy. Axial T2WI provides good contrast between white and grey matter, also useful in delineating white matter signal abnormalities. Gradient-echo T2* or susceptibility weighted images (SWI) is ideal for demonstrating hemorrhage and calcification, distinguishing it from ischemic gray matter lesions and astrogliosis.

Between 24 hours to 7 days of insult DWI with ADC maps is more sensitive for the detection of cytotoxic edema than conventional T1 or T2WI. The ischemic areas show restricted diffusion in this time period, which is manifested as bright signal on DWI and corresponding reduced signal intensity on ADC maps. DWI abnormalities generally peak at 3–5 days after the insult and subsequently normalize despite the tissue injury. Early DWI is excellent for the detection of white matter injury. However, few (approximately 15%) of the basal ganglia and thalamic lesions show normal DWI because of the antenatal insult or delayed cell death[10,11].

For abnormally increased T2 conditions like gliosis and cystic lesions the FLAIR sequence is useful. It is less sensitive to differentiate the signal intensities between gliosis and unmyelinated white matter. After myelination the sequence is useful for demonstrating periventricular and cortical gliosis[12,29].

MR spectroscopy is the highly sensitive and specific sequence than any other techniques for HII in first 24 hrs. It is performed at short echo time (35 ms) and intermediate echo time (135-145 ms) and recommended as an additional sequence in full term neonates. In term neonates with HII it detects lactate peak at 0.9-1.3 ppm. Characteristically double peak at 1.3 ppm on spectra obtained with short TE and inversion below the baseline on spectra obtained with intermediate TE seen, which distinguishes it from adjacent lipid peak at 0.9–1.3 ppm. Premature neonates usually show lower N-acetylaspartate (NAA) and higher lactate peaks. As the brain matures, NAA will increase and lactate will diminish. Therefore, it is not recommended in preterm infants. Increased lactate-to-choline and lactate-to-creatinine ratios and reduced concentrations of NAA and choline in the basal ganglia of full-term neonates with asphyxia are predictive of worse neurologic outcome[13]. Normal CSF shows lactate peak so care should be taken not to include CSF in MRS voxel. MRS should always be interpreted along with conventional and diffusion weighted MRI. HII to gray matter (deep gray matter and cortex) results in characteristic T1 hyperintensity and variable T2 signal intensity. White matter injury results in abnormal T1 hyperintensity without marked T2 hypointensity, denoting astrogliosis, and low T1 with high T2 signal intensity denoting cavitation or edema.(10) In general DWI is most useful in the first week of life and conventional T1 and T2WI are most diagnostically useful from the second week onwards. The recommended timing for MRI is between 5 and 14 days from birth. Early neonatal imaging (before 5 days) may underestimate the injury, however, it is useful in taking decision on ventilated patients[10].

Normal Magnetic Resonance Imaging Appearances Of Neonatal Brain:

Neonatal brain differs from adult by degree of myelination. Myelination starts in utero from second trimester and completed by 18-24 postnatal months (14). Myelination normally follows a topographical pattern, progressing from central to peripheral, inferior to superior and posterior to anterior. Myelin is produced by oligodendrocytes. Myelin is rich in lipids, including cholesterol, glycolipids and phospholipids.

Myelinated white matter appears as hyperintense on T1W images and hypointense on T2W images in comparision with cortex **(Figure 1)**. The high myelin contents of cholesterol and galactocerebroside, tight boundation of water molecules to macromolecules within myelin sheaths makes white matter appear hyperintense on T1W images.

Imaging findings in perinatal HII vary with insult severity and duration of hypo perfusion event, state of brain maturation, metabolic demands of key anatomic structures, state of maturity of glutamate receptors and gestational age [15]. All these above mentioned factors in aggregate affect the pattern vulnerability to HII. Actively myelinating structures are particularly susceptible to hypoxic-ischemic insults[16,17]. The prenatal brain is developmentally immature, so imaging findings in these premature infants differ from findings in those born at or near term[18].

Patterns And Distribution Of Brain Injury:

Atleast 10-15 minutes of hypoxia-ischemia is required to induce brain damage during perinatal period. The usual sites of brain injury in HII are cortex, subcortical and periventricular white matter, medial temporal lobe, internal capsule, basal ganglia and thalami.

Mild-to-moderate Or Less Severe Asphyxia Pattern Of Brain Injury:

In less severe pattern of hypoperfusion, the injury predominantly to the watershed zones of the cerebrum as cerebral autoregulation causes redistribution of blood flow to the hypermetabolically active deep gray matter structures. Along with brain maturation the vascular supply changes.

Ventriculopetal penetrating arteries extending inward from the surface of the brain supply the periventricular regions in the preterm brain. So hypoperfusion results in a periventricular border zone of white matter injury. In term, ventriculofugal pattern of vessels seen extending from lateral ventricles. The intervascular border zone moves peripherally to parasagittal location. So, subcortical white matter and parasagittal cortical injury predominates in term neonates.

Profound Or Severe Asphyxia Pattern Of Brain Injury:

Cerebral autoregulation is lost in severe hypoperfusion. The deep gray matter and the early or actively myelinating fibers with higher concentrations of neurotransmitter receptors are vulnerable to injure in profound form.

Severe asphyxia in term neonates causes injury to the posterior putamina, ventrolateral thalami, hippocampi and dorsal brainstem and occasionally the sensorimotor cortex.

HII IN PRETERM INFANTS:

Preterm newborns are born before 37 weeks of gestation and typically less than 1500g in weight. Approx. 50% of these newborns will exhibit some degree of white matter injury (19). Perinatal HII is more common in preterm newborns. The

prevalence of injury shows an inverse relationship to gestational age at birth. The superior cerebellar vermis, perirolantic cortex and posterior limb of internal capsule are not involved in preterm neonates as they normally myelinates near or at term. The unique physiology and cerebrovascular anatomy of the premature baby underlies the exquisite sensitivity of white matter to the abnormal milieu of preterm extra uterine life like ischemia and inflammation. These two upstream mechanisms can coexist and amplify their effects, leading to activation of two principal downstream mechanisms: excite-toxicity and free radical attack. Upstream mechanisms trigger generation of reactive oxygen and nitrogen species. The premature brain is intrinsically vulnerable to free radical attack due to immaturity of antioxidant enzyme systems and iron accumulation. Ischemia and inflammation trigger glutamate receptor mediated injury leading to maturation dependent cell death and loss of cellular processes [11]. In preterm born infants, elevated concentrations of inflammation related proteins in umbilical cord and neonatal blood are associated with neonatal cerebral white matter damage [20,21,22].

Patterns of injury in preterm includes profound or central, perinatal white matter damage, germinal matrix-intraventricular haemorrhage, periventricular hemorrhagic infarction and cerebellar injury.

Profound Hypoxic–ischemic Injury In Preterm Neonates:
Severe hypotension preferentially affects the early myelinated and metabolically active structures like thalami, basal ganglia (particularly posterior putamina), dorsal brainstem with relative sparing of cortex except pre and post central gyri. Relatively low involvement of the hippocampus, basal ganglia, perirolandic cortex and corticospinal tract are also observed. The early myelination of globus pallidus and thalamus by 24–25 weeks of gestation and late myelination of corpus striatum (caudate nucleus and putamen) and perirolandic cortex beyond 35–36 weeks of gestation explains the regional preference of injury [23,24]. These findings may be associated with GMH or PVL.

Reduced diffusivity (DWI hyperintensity, ADC hypointensity) and elevated lactate in magnetic resonant spectroscopy (MRS) are the first MRI finding to appear within 24 hrs in affected areas, followed by T2/T2 FLAIR hyperintensity by approx. 3rd day and T1 hyperintensity on approx. 4th day. Approximately after 7 days T2 hyperintensity signal decreases, DWI become normal (pseudonormalization) and decreased NAA in MRS. The involved basal ganglia shows cavitation and volume loss without gliosis.

Mild-to-moderate Hypoxic–ischemic Injury In Preterm Neonates:
The spectrum of brain injury in this group is broad and include germinal matrix haemorrhage, intraventricular haemorrhage(IVH)/periventricular hemorrhagic infarction (PVHI), perinatal white matter damage(PWMD) and cerebellar injury.

Germinal matrix (GM) forms the neuronal precursor zone. Larger number of cells aggregates at caudo-thalamic groove, superior margins of frontal horns, roof of fourth ventricle and granular cell layer of cerebellum. The prevalence of GMH is inversely related to gestational age and weight at birth. The prevalence of GMH in preterm neonates under 2000 g is approx. 25%. 90% of GMH occurs within the first 4 days of life and these bleeds less frequent after 34 weeks of gestation. Germinal matrix haemorrhage is of III grades, Grade I; Subependymal GMH typically involves caudothalamic groove with no or minimal intraventricular extension, Grade II; GMH with IVH filling < 50% of ventricular area, Grade III; GMH with IVH filling > 50% of ventricular area.

Grade IV GMH, called as PVHI occurs secondary to hemorrhagic ventricular distension, venous ischemia and hemorrhagic venous infarct. It occurs with a prevalence of 15% in newborns with IVH. Eighty to ninety percent of PVHI manifest within 96 hrs of life. In case of bilateral PVHI, heritable microangiopathy due to mutations in the gene COL4A1 on Ch 13q34 to be considered.

Depends on the stage of hematoma the signal intensities varies, hyperacute hematoma (< 24 hrs, T1 iso and T2 hyperintense), acute hematoma (1-3 days, T1 iso and T2 hypointense), early subacute hematoma (3 days-1 week, T1 iso and T2 hyperintense), late subacute (1-2 week T1 hypo and T2 hyperintense) and chronic hematoma (>14 days, T1 and T2 hypointense). Hemorrhage associated hydrocephalus with debri level in dependent part of ventricles also seen.

Delayed MR shows particularly in GRE and SWI images haemorrhagic debris within the periventricular infarction, the ipsilateral ventricular system and in subarachoid spaces (superficial siderosis). Infarct core shows diffusion abnormalities as described above.

Periventricular white matter damage also known as periventricular leukomalacia is inversely related to gestational age at birth. This pattern of injury alone or in conjucation with maternal or fetal infectious or inflammatory conditions or inborn error of metabolism lead to damage of premyelinating oligodendrocytes in periventricular white matter and neurons of subplate (deep layer of cortex). Given the diffuse cerebral injury (neurons and axons) beyond just white matter less commonly used description is encephalopathy of prematurity. White matter injuries are more common adjacent to foramen of monro and lateral ventricle trigones.

There are three pattern of PWMD have been described diffuse, focal or multifocal-non cavitatory, focal or multifocal-cavitatory **(Figure 3)**. Cavitatory PWMD is the least common one. Lower extremity neurons are normally seen in close to periventricular region than upper extremity neurons. Therefore lower extremity neurons are more frequently affected leading to clinical presentation called spastic diplegia (*cerebral palsy*). Geneculocalcrine (motor and visual) pathways also seen in periventricular region.

In first week of life PWMD shows T2/T2 FLAIR hyperintensity, diffusion restriction, lactate peak in MRS, fractional anisotrophy and radial diffusivity in diffuse tensor imaging. Reduced fractional anisotrophy for corrected gestational age is an important poor prognostic indicator of cognitive and neurocognitive development. Ti hyperintensity (cogulative necrosis) and T2 hyperintensity seen in involved areas. Blooming on SWI and GRE images seen which is more common in extreme prematurity. Late MRS shows (>2 weeks) decreased NAA and lactate washout.

End stage PVL shows reduced white matter volume and thinning of corpus callosum. In affected areas the cortex nearly touches the lateral ventricles, which are enlarged and irregular margins.

Cerebellar injury is underappreciated in preterm. Commonly manifests as reduced cerebellar volume. It reflects transsynaptic degeneration of cerebellum and tracts due to supratentorial neurons loss and/or disturbed signalling between the overlying leptomeninges and the underlying developing cerebellum.

HII IN TERM NEONATES:
Term newborns are those who completed 37 weeks or above. As mentioned previous areas with highly metabolic demands are more vulnerable to injury.

SEVERE HII IN TERM NEONATES:

In term neonates HII predominantly affects actively myelinating areas with high NMDA receptors. The deep gray matter (posterior putamina, ventrolateral thalami), hippocampi, dorsal brainstem, optic radiation, superior vermis and perirolandic cortical regions. Other at risk regions are subthalamic nuclei and corticospinal tracts may also involves deep white matter, para sagittal and other watershed areas.

Spin echo MR in HII often normal in first 8-10 hrs. DWI is the most sensitive sequence in first 24 hrs will reveal diffusion restriction and corresponding decrease in (apparent diffusion coefficient) ADC values. Diffusion abnormality peaks at 5th day then "pseudonormalize" by one week. Normal DWI does not exclude HII especially during first 6-18 hrs after insult and in pseudonormalization.

The posterior half of the PLIC is myelinated at birth and has high SI on T1-weighted images in normal full-term-born infants [25,26]. The loss of this normal high SI in the PLIC in infants with HIE, probably indicating a delay in myelination or injury to previously myelinated tracts[27]. At 2-3 days of life T1 shortening (hyperintensity) in basal ganglia and in thalami or decrease in T1 shortening of posterior limb of internal capsule is the marker of profound HII **(Figure 2)**. T1 shortening in posterior putamina than posterior limb of internal capsule is a poor prognosticator. This loss of high SI in the PLIC, though sometimes subtle, is associated with unfavorable outcome [28]. The pathological T1 hyperintensity is due to release of calcium, manganese and myelin lipids from infarcted tissue. T1 shortening may persist for month(s).

T2/T2 FLAIR hyperintensity begins within 24 hrs persist for 3-4 days then transitions to hypointense. MRS shows elevated lactate at 1.3 ppm, which may elevated as early as 4 hrs. A lactate:NAA ratio > 0.5 indicates serious neurological injury. Elevation of gluatamine-gluatamate peak resonating at 2.3 indicates acute brain injury. Should be aware of lactate mimics, lactate doblet resonance at 1.15 from methyl protons of propane-1,2, idol vehicle of sedation agent phenobarbital. Ulegyria is shrunken cortex with flattened, mushroom shaped gyri and cystic encephalomalacia in involved areas are chronic findings of hypoxic ischemic injury.

LESS SEVERE HII IN TERM:

Less severe pattern usually spars brainstem, cerebellum and deep gray nuclei. Prolonged partial asphyxia usually causes injury in watershed zones **(Figure 5)**. Para sagittal cortex and deep white matter regions are usually involved. Depends on the day of injury findings appears in different sequences as described in severe pattern above. Late finding is multicystic border zone encephalomalacia.

HII MIMICS:

Neonatal hypoglycemic, on imaging it can manifest on MRI as bilateral areas of increased signal on both T2 and FLAIR affecting the posterior limb of the internal capsule,cerebral cortex particularly in parieto-occipital and insular cortex, hippocampus and basal ganglia. Bithalamic symmetrical T1 shortening in metabolic disorders like kernicterus also to be considered. Should be aware of lactate mimics, lactate doublet resonance at 1.15 from methyl protons of propane-1,2, idol vehicle of sedation agent phenobarbital. Proper clinical history is essential to reach proper diagnosis.

CLINICAL FEATURES AND NEUROLOGICAL OUTCOMES OF HII:

Neurodevelopmental outcome can be predicted depends upon the pattern of injury. Mostly clinical history and perinatal events may give clues to the pattern of injury.

HII involving basal ganglia and thalamic region usually needs resuscitation at birth, severe encephalopathy, and clinical seizures. BGT pattern is usually associated with cerebral palsy and cognitive deficits. Abnormal PLIC is associated with abnormal motor outcome [23] redominant cognitive, visual, language, behavioural and seizure problems at a median age of 2 years usually associated with watershed pattern [24,25].

Most common outcome associated with periventricular leukomalacia are motor and visual impairment. Minimal PVL may or may not associate with impairments. Most of the children with severe PVL usually associated with impairments like cognitive, visual, motor and seizures [30].

Shamic et al.[31] proposed a validated clinical MRI injury scoring system in neonatal hypoxic-ischemic encephalopathy. They gave score in different regions: (a) subcortical: caudate nucleus, globus pallidus and putamen, thalamus and the posterior limb of the internal capsule, (b) white matter, (c) cortex, (d) cerebellum and (e) brainstem.

The MRI injury was graded as none, mild, moderate or severe. The result was higher MRI injury grades were significantly associated with worse outcomes in the cognitive, motor and language domains of the Bayley-III.

AIMS AND OBJECTIVES:

Aims of this study are to see the different pattern/distribution of injury in preterm and term born patients.

MATERIAL AND METHODS:

This retrospective study was conducted at Department of radiology, Superspeciality Hospital, Gajra Raja Medical College, Gwalior, Madhya Pradesh during the period of February 2019 to February 2020. All patients with findings of hypoxic ischemic injury were followed up retrospectively for history like gestational age at delivery, mode of delivery, prolonged labour, maternal or fetal infection, intensive care unit admission, seizures, focal neurological deficits, developmental delay etc.

RESULT AND DISCUSSION:

This was a retrospective study of 100 patients. There were 58 (58%) males and 42 (42%) females and a male to female ratio of 1.3:1. Most of the children were born first order 68% and born second order or more were 32%. A 48% of patients were belonged to the age less than one month and 52% were belonged to age between one month to 2.5 years. The youngest patient of the study was 7 days old newborn and oldest was 2.5 years. The patients of age less than one month had history of perinatal complications, intensive care unit admission and focal neurological deficits.

Delayed development of milestones was the most common presenting complaints in patient age between 1 month to 2.5 years. The socioeconomic status of our study population were; 63% in lower class, 28% in upper lower class, 6% in lower middle class and 3% in upper middle class.

In our study 78% of patients were associated with maternal diseases. Most common associated maternal disease was oligohydramnios seen in 57.6% of patients and next common was maternal anaemia in 32%. Out of 100 patients 73 (73%) were born through lower segment caesarean section and 27% through vaginal delivery. Among vaginal delivery 92.5% were assisted delivery (vacuum extraction, forceps). Scalp collection was seen in 24% patients after delivery and all of them were born through assisted vaginal delivery. 1% of patients were having extra axial collection (subdural hematoma).

The term born patients of our study was 72% and preterm born 28%. All the patients were having delayed cry at birth and admitted in Neonatal Intensive Care Unit. Of them 66%

were having low APGAR score at 1 minute and 5 minutes, 34% were having low APGAR score at 1 minute and improved at 5 minutes. **Ibrahim Aliyu et.al** [32] reviewed records of 142 asphyxiated patients in which most of the cases were having 1 minute APGAR score of 4-5 (62.0%) and 5th minute score was 6 or more (45.8%). They concluded the study that, APGAR score still remains most important tool in resuscitation and monitoring asphyxiated paediatric patients. Clinically grading of HIE in our study population as follows; Grade I in 56%, Grade II in 30% and Grade III in 14%.

In our study most common location involved in term born patients was watershed areas (36.1%) and most of them (89%) were clinically diagnosed as Grade I or Grade II HIE. With respect to the location of involvement, spectrum of MRI findings in term born patients were; cortical (8.3%), watershed areas (36.1%), periventricular white matter (34.7%), basalganglia and thalami (16.7%), brainstem (1.4%), cerebellum (2.8%), germinal matrix and intraventricular (0%) and diffuse involvement (0%). Findings were mentioned in table no.1

TABLE-1:

SPECTRUM OF MRI FINDINGS IN TERM (N=72)		
Location	**No.of cases**	**Percentatage**
Cortex	6	8.3%
Watershed areas	26	36.1%
Periventricular white matter	25	34.7%
Basal ganglia and thalami	12	16.7%
GMH/IVH	0	0%
Brainstem	1	1.4%
Cerebellum	2	2.8%
Diffuse	0	0%

Most common location involved in preterm patients of our study was watershed areas (32.2%), followed by basal ganglia and thalami (25%). With respect to the location of involvement, spectrum of MRI findings in preterm born patients were; watershed areas (32.1%), basalganglia and thalami (25%), periventricular white matter (17.9%), germinal matrix and intraventricular (10.7%), cortical (7.1%), diffuse involvement (7.1%), brainstem (0%) and cerebellum (0%). Findings were mentioned in table no.2.

TABLE-2:

SPECTRUM OF MRI FINDINGS IN PRETERM (N=28)0.5		
Location	**No.of cases**	**Percentage**
Cortex	2	7.1%
Watershed areas	9	32.2%
Periventricular white matter	5	17.9%
Basal ganglia and thalami	7	25%
GMH/IVH	3	10.7%
Brainstem	0	0%
Cerebellum	0	0%
Diffuse	2	7.1%

Ulegyria seen in 43% of study population. Corpus callosal thinning (56%) predominantly in its posterior part and microcephaly (21%) were associated with HIE in our study. These associated findings are may be secondary to brain parenchymal volume loss. Intra or extra axial and scalp collections seen in 25% study population, all of them were associated with difficult and assisted delivery. 3% of cases were associated with congenital anomalies. Most common congenital anomaly was corpus callosal agenesis seen in 2 patients and Chiari 1 malformation in 1 patient. HIE with intracranial infections seen in 6% cases. HIE associated significant findings mentioned in table.no 3.

TABLE-3:

OTHER SIGNIFICANT FINDINGS(N=100)	
Ulegyria	43%
Microcephaly	21%
Corpus callosum thinning	56%
Collection(intra/extra axial/scalp)	25%
Congenital anomalies	3%
Infection	6%

CONCLUSION:

Hypoxic ischemic encephalopathy is one of the most important causes of perinatal mortality. It involves in different locations of neonatal brain. Most common location involved in term and preterm born patients of our study is watershed areas.Most common associated imaging findings were corpus callosum thinning followed by ulegyria. Other significant associated imaging findings are microcephaly, collections, congenital anomalies and infections.

Financial Support And Sponsorship:
Nil.

Conflicts Of Interest:
There are no conflicts of interest.

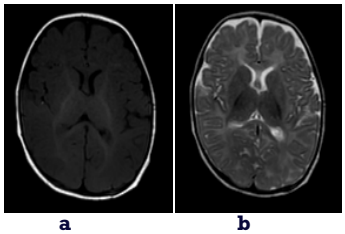

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Figure 1 : (a, b): Axial magnetic resonance imaging (MRI) of a 8 day old full-term normal neonate at the level of internal capsule. (a) T1 weighted image (WI) shows normal increased signal intensity (SI) of the posterior limb of internal capsule relative to the basal ganglia and thalamus; (b) Corresponding T2WI shows normal hypointense signal of the posterior limb of internal capsule.

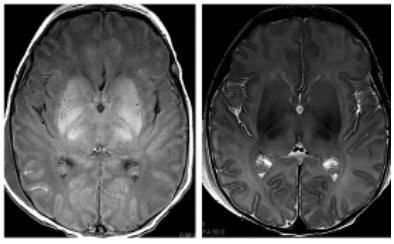

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Figure 2: Basal ganglia–thalamus pattern of HII in a 7-days old infant with seizures. (a) Axial T1-weighted MR images shows abnormal severe high signal intensity in bilateral lentiform nuclei and ventrolateral thalami. (b) Axial T2-weighted MR images shows marked hypointensity in the bilateral posterolateral putamen and ventrolateral thalamus.

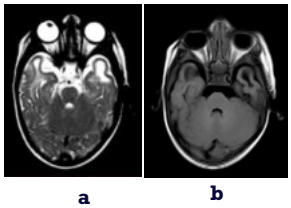

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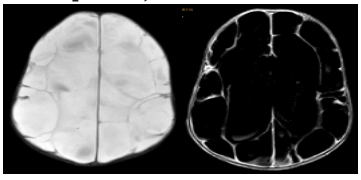
Figure 3: Axial T2 WI (a) and T2 FLAIR (b) MR images of three months old preterm born child with delayed development of milestones shows cystic areas in bilateral basitemporal lobes (periventricular pattern).



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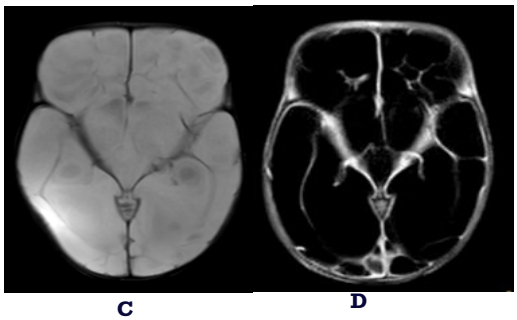
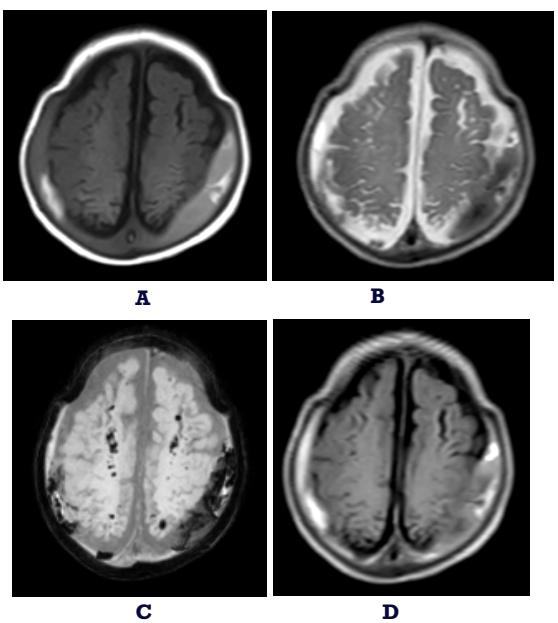


Figure 4: Axial T2 WI (a), axial T2 FLAIR (b), axial T2 WI (c), axial T2 FLAIR (d) at the level of basal ganglia, sagittal T2 WI (e), axial T2 WI (f) and T2 FLAIR (g) MR images of a 2 years old preterm born child with global developmental delay shows multiple cystic areas in bilateral supra tentorial brain parenchyma with no remaining brain parenchyma, involvement of deep grey matter nuclei in bilateral basal ganglia and marked thinning of corpus callosum. Brainstem and bilateral cerebellar hemispheres are normal.



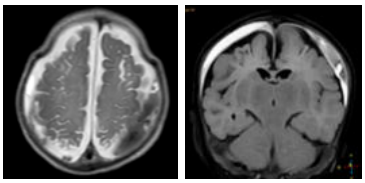


Figure 5: Axial T1 WI (a), axial T2 WI (b), axial T2* (SWI) (c), axial T2 FLAIR (d), axial T2 WI (e) and coronal T2 FLAIR (f) MR images of 10 months old preterm born patient shows mixed intensity bilateral subdural hematoma, multiple blooming foci in bilateral frontoparietal white matter, cystic areas in bilateral frontoparietal lobes (in watershed areas) and ulegyria pattern.

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