""COMPARATIVE STUDY OF TRANSCRANIAL NEUROSONOGRAPHY AND CROSS-SECTIONAL IMAGING IN NEONATES WITH HYPOXIC ISCHEMIC ENCEPHALOPATHY"

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B.L.D.E. (DEEMED TO BE UNIVERSITY) VIJAYAPURA, KARNATAKA

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LIST OF ABBREVATIONS USED

ACA – Anterior cerebral artery

CT – Computer tomography

ICH - intra cerebral hemorrhage

GMH – Germinal matrix hemorrhage

IVH – Intraventricular hemorrhage

LSCS – Lower segment caesarean section

MCA – Middle cerebral artery

MRI – Magnetic resonance imaging

PCA – Posterior cerebral artery

PVHI – Periventricular hemorrhagic infarction

PVL Periventricular leucomalacia

USG – Ultrasonography

NSG - Neurosonography

TORCH - Toxoplasmosis, Rubella, Cytomegalo Virus and Herpes

VLBW - Very low birth weight

DWI Diffusion weighted imaging

ADC- Apparent Diffusion Coefficient

NICU- Neonatal intensive care unit

NVD - Normal Vaginal delivery

HIE - hypoxic ischemic encephalopathy

WMI – white matter ischemia

MRS - magnetic resonance spectroscopy

SNR – signal noise ratio

TR – time to repeat

TE – time to echo

FLAIR – fluid attenuated inversion recovery

FOV – field of view

BW – band width

PPM – parts per million

EEG – electroencephalography

SWI – susceptibility weighted imaging

GRE – gradient echo

NAA – N acetyl aspartate

SSFSE – single shot fast spin echo

HASTE - half-Fourier acquisition single-shot turbo spin-echo

PROPELLER -Periodically Rotated Overlapping Parallel Lines with Enhanced Reconstruction

WHO – world health organization

INTRODUCTION

Birth asphyxia is considered as one of the most common etiologies of cerebral palsy and severe neurologic deficits in paediatric population, occurring intwo to nine of every 1000 live births in developed countries. It is one of the major preventable cause of early neonatal mortality in India contributing to almost 20% of neonatal deaths in India and contributes to 1.2 million neonatal deaths every year and about 3 – 3.5 lakh infants die within first 3 days of life. The incidence of long-term complications depends on the severity of hypoxic-ischemic encephalopathy leading to mental retardation, learning disabilities and seizures. Infants with features of mild hypoxic-ischemic encephalopathy are mostly free from serious CNS complications.

Main pathophysiology includes lack of sufficient blood flow in conjunction with decreased oxygen content in the blood leads to loss of normal cerebral autoregulation and diffuse cerebral injury. The nature of the hypoxic brain injury depends upon the severity of hypotension, duration of hypoxia and the degree of brain maturation.

Imaging plays a crucial role in early diagnosis and timely intervention in severe birth asphyxia, thereby reducing the severity of complications in cases of neonatal brain injury. Accurate identification and characterization of the severity, extent, and

location of brain injury rely on the selection of appropriate neuroimaging modalities, including ultrasonography (US), computed tomography (CT), and magnetic resonance (MR) imaging. Newer diagnostic techniques such as diffusion-weighted MR imaging and MR spectroscopy provide further insight into HIE and for possible early therapeutic intervention[1].

Current study evaluates and compares transcranial neurosonography with CT / MRI in the neonates with Hypoxic ischemic encephalopathy.

AIMS & OBJECTIVES OF THE STUDY

1) To identify and evaluate the pattern of imaging findings in Hypoxic ischemic encephalopathy and its sequelae on Transcranial neurosonography and Cross-sectional Imaging (CT/ MRI).
2) To compare the imaging findings of Hypoxic ischemic encephalopathy on Transcranial neurosonography with Cross-sectional Imaging (CT / MRI).
3) To correlate the imaging findings with clinical severity.

PATHOPHYSIOLOGY OF HYPOXIC ISCHEMIC INJURY

Asphyxia is described as impairment in exchange of oxygen and carbon dioxide resulting in decreased blood oxygen content leading to hypoxia and increased carbon dioxide in blood causing hypercarbia , metabolic acidosis and decreased systemic blood pressure.

Hypotension and decreased cerebral blood flow leads to acidosis, release of inflammatory mediators, excitatory neurotransmitters and free radical formation.Hypoxia and hypercarbia cause dysregulation of normal cerebral blood flow in term neonates leading to pressure passive flow. In term neonates, constriction of blood vessel occurs when blood pressure increases and dilate when blood pressure decreases known as cerebral autoregulation to maintain cerebral blood flow. In preterm neonates, pressure passive flow is present even in absence of asphyxia or other disease.

Impaired cerebral blood flow may result from fetal cardiac and vascular compromise, either in utero or postnatally. Intrauterine asphyxia occurs when placental blood flow and gas exchange is interrupted. Fetal factors include fetomaternal hemorrhage, fetal thrombosis, and fetal bradycardia. Inadequate placental perfusion due to maternal hypotension, preeclampsia, chronic vascular disease, abruptio placenta.

Impaired maternal oxygenation asthma, pulmonary embolism, pneumonia, carbon monoxide poisoning, severe anemia or disrupted umbilical circulation due to tight nuchal cord and cord prolapse. Postnatal asphyxia results from underlying severe respiratory distress syndrome caused by hyaline membrane disease, pneumonia, meconium aspiration syndrome or congenital heart anomalies that cause neonatal pulmonary failure or hypotension.

Combination of decreased blood pressure and pressure passive flow result in cerebral hypoperfusion leading to hypoxic ischemic injury in term neonate and germinal matrix haemorrhage or white matter injury in preterm neonate.

Hypoxia also reduces cardiac contractility, altered capillary permeability. Reperfusion of weakened capillaries of premature neonates can cause rupture of vessels and results in germinal matrix haemorrhage (most common location, as reperfusion is maximum in germinal matrix), intraventricular and intracranial haemorrhage.

Patterns of hypoxic ischemic brain injury in neonate

Hypoxic ischemic injury consequences depend on severity of hypotension, maturity of the brain and duration of the hypoxic event.

A hypoxic-anoxic cerebral event that lasts for more than 10 minutes is considered to induce parenchymal changes, and the extent of injury increases with prolonged duration of the insult.

Severity of hypotension

When cerebral blood flow is mildly or moderately compromised, the blood flow is shunted to posterior circulation in order to maintain adequate perfusion to brainstem, cerebellum and basal ganglia[16]. As a result, the damage is limited to cerebral cortex, intervascular border zones or watershed areas of cortex and white matter of bilateral cerebral hemispheres.

In complete or near total compromise of blood flow, the shunting of blood is no longer adequate to save deep cerebral structures.

In profound hypotension, the location of injury is initially deep cerebral nuclei like thalami, basal ganglia, posterior brainstem and metabolically active regions of cerebral cortex including sensorimotor regions.

Neurotransmitters like glutamate, NMDA receptors, TNF- alpha, TGF-beta are postulated as main causes of hypoxic ischemic injury which are more in myelinated areas of cerebral parenchyma[82.]

Maturity of brain

Mild to moderate hypotension in preterm neonates causes periventricular and deep white matter injury sparing subcortical white matter and cerebral cortex. In term neonates, mild to moderate hypotension results in injury of watershed zones of the cerebral cortex and underlying subcortical, periventricular white matter.This change of injury pattern is attributed to ischaemic changes in intervascular boundary zones termed as watershed zones.

Ventriculofugal blood vessels are relatively less in preterm neonates as all blood supply to white matter comes from vessels coursing from surface of brain[66,67]. Preterm vessels have limited vasodilatory capacity. Preterm periventricular white matter is the site of oligodendrocytes proliferation that plays a crucial role in myelination and are highly vulnerable for hypoxic ischaemic damage leading to reduced GLU R 2 expression[17].

In term babies (38 -42 postconceptional weeks) & post term (43- 46 weeks) reduced GLU-R2 expression is seen in neocortex, thus cortex is more prone for hypoxic injury in mild to moderate ischemia.In profound hypoxia, injury is seen in most metabolically active regions of brain like thalami and brainstem in early third trimester. Brainstem, thalami, basal ganglia, prerolandic cortex are involved in late 3rd trimester.

Duration of the injury

Severity of hypoxic injury depends on the duration of injury, selective neuronal necrosis is demonstrated immediately after 10 minutes of perfusion arrest, basal ganglia injury can be seen at 8-10 min and entire cerebral parenchyma injury after 15-20 min[68].

There are four patterns of brain injury:

1) Mild to moderate hypotension in preterm infants.

2) Severe hypotension in preterm infants.

3) Mild to moderate hypotension in term infants.

4) Severe hypotension in term infants.

Hypoxic injury in preterm neonate

With increased survival of premature babies (less than 37 weeks) the neonates are at risk of severe neurodevelopmental impairment. Few other conditions also can result in hypoxic cerebral injury in premature neonates. The two most widely accepted pathologic mechanisms include impaired cerebrovascular auto regulation with subsequent ischemic injury and feto-maternal infection.

In mild to moderate hypoxic ischemic injury, several types of brain injuries can occur which includes white matter injury, germinal matrix hemorrhage, intraventricular hemorrhage and periventricular hemorrhagic infarction. GMH occurs secondary to rupture of thin walled capillaries in germinal matrix most commonly seen along the walls of lateral ventricles and extend into ventricles causing hydrocephalus. White matter injury is seen due to necrosis. Venous infarction occurs due to venous thrombosis and infarction. Rarely, cerebellar atrophy can be present.

White matter injury and germinal matrix hemorrhage are the most common findings in extremely preterm neonates. Large intracerebral hemorrhages and extensive periventricular cystic lesions in white matter can be easily identified with transcranial neurosonography. MRI is more sensitive in identifying small foci of intraventricular hemorrhage and cerebellar hemorrhages, early focal lesions in the periventricular white matter, cerebellum and large diffuse noncavitatory white matter injury. Additional imaging sequences like diffusion-weighted imaging, diffusion tensor imaging and susceptibility weighted imaging helps in recognition and prediction of neuromotor outcome[30].

Periventricular and intraventricular hemorrhage

Cerebral hypoperfusion followed by reperfusion with increased venous pressure in the weakened capillaries may rupture, resulting in hemorrhage. Most commonly involving ventricular, subventricular locations called germinal matrix, which is highly active between 8 weeks to 28 weeks of gestational age, as it helps in production of neurons and glial cells. Last areas of GM to involute are the regions around anterior tips of the frontal horns, near posterior aspect of caudate head called as ganglionic

eminence, external granular layers of cerebellar hemispheres and choroid plexus also bleed in premature neonates when seen in association with GMH.

Grading of germinal matrix hemorrhage in premature neonates[36]

Grade 1 :germinal matrix hemorrhage with no or minimal intraventricular haemorrhage.

Grade 2 :bleed extending from subependymal germinal zones to ventricles without ventricular dilatation.

Grade 3 :germinal matrix hemorrhage with intraventricular haemorrhage associated with ventricular dilatation.

Grade 4 :periventricular hemorrhagic infarction (PVHI).

PVHI once thought considered as extension of GMH to adjacent cerebral parenchyma. Currently thought of sequelae to cerebral venous infarctions (69).Bilateral PVHI, Grade 3 /4 GMH are mostly associated with poor neurological outcome[20].

Imaging in Germinal matrix hemorrhage

Transcranial ultrasonography shows GMH as the areas of increased echogenicity. In coronal planes, these areas are seen as well defined areas of increased echogenicity along the subependymal regions adjacent to ventricular walls. Most common location is caudothalamic groove, inferior to the floor of posterior part of frontal horn of lateral ventricle. Sagittal plane helps to differentiate GMH from echogenic choroid plexus. MRI spin echo, gradient echo or susceptibility weighted sequences are best imaging sequences for evaluating hemorrhage, seen as hypointense on T1WI , markedly hypointense on T2WI , T2*WI in acute phase (3days) becomes hyperintense on T1WI and low on T2WI , T2*WI over next 3 to 7days , between 7 to 14 days gradually increased signal intensity seen on T2WI slowly turning into CSF intensity on T1WI over next several months.

Both MRI & transcranial neurosonogram shows cerebellar hemorrhage better demonstrated in posterior fontanellar view or mastoid views on ultrasound. It does not result in hydrocephalus. However carries poor prognosis.

IVH in acute presentation is difficult to differentiate from choroid plexus. On power Doppler, vascularity can be seen in choroid plexus, over few weeks hemorrhage becomes organised and less echogenic seen as a sonolucent mass in lateral ventricle commonly in body or atrium of lateral ventricle.

In acute phase, IVH will cause ventriculomegaly due to blockage of CSF pathway and can lead to obliterative arachnoiditis most commonly occurs in basal cisterns. Ventricular dilatation can be demonstrated on USG or MRI[21].

PVHI is ischemic cerebral parenchymal injury with hemorrhage which occurs in periventricular white matter adjacent to lateral ventricle. Location and the size of the infarct depends on occluded vein. On neurosonography,PVHI can be seen as globular or crescent shaped area of hyper and hypoechogenicity depending on the time of injury.

MRI shows regions of hemorrhage as dark on T2WI surrounded by non-hemorrhagic venous infarct as high intensity on T2WI and demonstrate blooming on Gradient recovery sequence.

Cerebral sinovenous thrombosis (CSVT) is rare and can occur in 0.41 per 100,000 liveborn neonates, usually presents as thalamic hemorrhage with superior sagittal sinus thrombosis[92].

White matter injury in premature neonate

White matter injury of premature is also called as periventricular leukomalacia (PVL).

Hypoxic injury can affect any part of white matter. Most common locations include posterior periventricular white matter adjacent to lateral aspect of trigone of lateral ventricles, frontal periventricular white matter adjacent to foramen of monroe. As sonography is the primary modality to screen premature babies, cavitatory WMI is more common.

Volpe[70] described three types of white matter injury

1. Diffuse white injury

2. Focal / Multifocal noncavitatory white matter injury

3. Focal /Multifocal cavitatory white matter injury.

MRI is considered as the best imaging modality more over transcranial neurosonography in detecting noncavitatory WMI.

Imaging features of white matter injury in premature neonate

By cranial USG using high frequency linear array transducer (up to 10- 12 MHz) WMI can be demonstrated as increased echogenicity in periventricular white matter .However, cerebral edema will also cause increased echogenicity but resolves in subsequent imaging.

Occasionally, specular reflections from normal axon bundles also mimic WMI. However, normal appearance on neurosonography cannot rule out WMI. Periventricular flare should be equal or more echogenic than the choroid plexus or it should be heterogeneous with foci of hemorrhage and necrosis with subsequent cavity formation on follow up scans to confidently diagnose WMI. Timing of cavitatory changes varies and depends on the extent and severity of the injury typically occurs 2 to 4 weeks after injury[23].

WMI graded by characteristic of periventricular white matter by de vries[89].

Grade I **Periventricular / deep white matter areas of increased echogenicity present for 7 days or more (prolonged periventricular flare)**

Grade II **Increased echogenicity that evolve into small localized fronto parietal cysts.**

Grade III **Periventricular areas of increased echogenicity evolve into extensive periventricular cystic lesions involving fronto-parietal and occipital white matter**

MRI evaluation of white matter injury

Noncavitatory white matter injury is more common than cavitatory WMI[19] and is better seen on MRI as small foci of hyperintensity on T1WI mild hypointensity on T2WI. Neurosonography can be normal in these cases. On DWI, restricted diffusion can be seen up to one week. On follow up scans, larger areas of white matter hyperintensities may undergo necrosis, develop T1 hypointensity and T2 hyperintensity.

Over a time they cavitate and coalesce by astrogliosis with diminished white matter volume leading to exvacuo dilatation of ventricles along the site of gliosis and thinning of corpus callosum ultimately associated with poor neurodeveleopmental outcome.

Differential diagnosis for white matter injury to premature neonate include ventriculitis, inborn errors of metabolism, hydrocephalus.

Cerebellar injury in premature neonate

8-20 % of preterm neonates with hypoxic ischemic injury cases show focal / diffuse cerebellar injury[3,47]. It is usually due to hemorrhage and can be better demonstrated through posterior or mastoid fontanelle on ultrasonography.Cerebellar injury isusually associated with supratentorial bleeds. MRI shows high incidence of cerebellar hemorrhage (20 %). On follow up of these cases, focal / diffuse cerebellar atrophy can be seen due to selective neuronal necrosis or diaschisis due to cerebral white matter injury.

Profound hypotension in premature neonates

Outcome of profound hypotension carries very poor prognosis.Hypoxic injury predominantly involves deep gray matter nuclei and brainstem nuclei since these areas are the most mature and have highest metabolic rates. Although GMH and Periventricular WMI can occur as well, more specifically involved areas are dorsal brainstem, anterior cerebellar vermis, thalami, lentiform nuclei, precentral and post central gyri.

Sonography on the first day may be normal, by day two hyperechogenicity may be seen in basal ganglia and thalami.

MRI is acquired on the first 24 to 48 hrs shows restricted diffusion on DWI in dorsal midbrain and thalami, T2WI hyperintensities are seen in thalami. Preterm with > 20 min hypotension may suffer from severe hypoxic injury involving entire cerebral parechyma. By day 2 -3, T1WI shows faint diffuse hyperintensity in injured regions. Imaging at 1 to 3 weeks after injury shows white matter T2WI hyperintensities with small foci of T1WI hyperintensities scattered throughout centrum semiovale.Chronic phase will show changes of cerebral atrophy in the form of small shrunken thalami , small brainstem , small or absent basal ganglia, reduced cerebral white matter volume.

Hypoxic ischemic injury in term neonate

Mild to moderate hypotension in term neonate results in ischemic injury to cerebral white matter and cortex predominantly involving intervascular boundary zones (watershed zones).

Watershed regions are seen between regions supplied by ACA, MCA and those between MCA and PCA. These neonates usually presents with seizures, hypotension, weakness and spasticity.

On neurosonography with high frequency linear transducer, hypoxic injury may show increased echogenicity of periventricular white matter reflecting edema in subcortical watershed regions. In severe cases, cortex is also involved resulting in blurring of cortical – white matter interface with effacement of sulci and ventricles.

On CT scan in acute phase it is often difficult to detect these changes as fronto-parietal white matter is normally of low attenuation due to high water content in new born and beam hardening artefact from calvarium that may obscure cortex.

On MRI, DWI and proton spectroscopy are most sensitive during first 24 -48 hrs. DWI is better for evaluating the pattern of injury and helps in prognosis of hypoxic injury. DWI shows extensive involvement of cortex and subcortical white matter in intervascular watershed zones .It is essential to correlate ADC values as DWI has T2 component within and new born babies have high T2 relaxation time. Basal ganglia and posterior fossa structures are relatively spared. DWI performed in first few hours

or within 24 hrs. May be normal or can show pseudo-normalization after 7 days and can underestimate the injury (prior to secondary energy failure). ADC values should always be correlated with visual analysis of both conventional as well as diffusion-weighed images for maximum detection of pathological tissue, and the timing of the scan needs to be taken into consideration when interpreting the results[87].

In acute phase, proton MRS shows elevated lactate and reduced NAA in cerebral cortex notably in watershed zones and in subcortical zones more than deep gray matter. Proton spectroscopy remains abnormal with lactate levels peaking at 3rd to 5th day of injury with low NAA levels[72].

On T2WI, affected cortex will be hyperintense starting by 24 hrs. of injury more visible with first echo of T2 (60 ms). On T1WI, edematous brain will appear as low intense areas in cortex and white matter. Loss of gray white matter distinction is mainly seen in severe injury.

As injury evolves MRS and DWI undergoes pseudo-normalization at 6 to 10 days and then show more chronic brain injury in the form of increased diffusivity and decreased NAA[72].

MRI helps in definitive diagnosis in perinatal hypoxia and is reliable for detecting periventricular white matter injury thus helps in predicting outcome[89]. Patterns of brain injury are usually determined by the nature, timing and the severity of the hypoxic insult. However, imaging appearances are influenced by sequences used and duration of injury at the time of performing a scan. Thinning of cortex and cystic degeneration of white matter is seen by 3 to 4 weeks. Cavitatory areas shrink to glial scar causing ex-vacuo dilatation of lateral ventricles particularly in trigones and occipital horns leading to cerebral atrophy and poor neurological outcome. MRI helps in early detection of white matter injury[81,91].

Profound hypotension in term neonates

Different patterns of injury are seen with profound hypotension when compared to mild to moderate injury. These neonates show injury to lateral thalami, posterior putamen , sub-thalamic nuclei , hippocampi , corticospinal tracts which are metabolically more active with more blood supply and high degree of synapse

construction. Some patients show injury to lateral geniculate nucleus and optic radiation. Cortex is spared except perirolandic area. Most severe injury is involvement of brainstem nuclei and most of them die within first few days. Profound injury patients have lower 1 min Apgar score < 3 or may have postnatal cardiorespiratory arrest.

On Sonography, hyperechogenicity is noted in bilateral thalami, globus palidi, putamen, perirolandic cortex. Thalamic hyperechogenicity is usually associated with poor outcome. With severe injury, hyperechogenecity can be demonstrated in white matter, cortex and basal ganglia. Cortex becomes edematous with loss of gray white matter distinction after respiratory distress. Early cerebral edema can present as diffusely hyperechoic parenchyma with effaced lateral ventricles. On Transcranial Doppler, the resistive index of vessels can be seen raised (normal is 0.7)[11].

CT shows hypoattenuation of affected areas, thalamic hypo density can be seen in first few days and indicates bad prognosis[73].MRI is normal if performed on day 1.Only proton MRS will show elevated lactate within hrs and eventually normalized by 6-8 hrs. Persistent raised lactate levels with low NAA seen after 24 -48 hrs can be seen[72].

DWI shows restricted diffusion in lateral thalami in 24 hrs. DWI will underestimate the damage if performed < 24 hr prior to secondary energy failure and pseudo-normalizes by 5 to 6 days[74].By 3 days, T1 and T2 imaging gets abnormal with T2WI showing hyper intensity of posterior thalami. Maximal abnormality is seen by 3 to 5 days with DWI. T1WI and T2WI shows abnormal signal in lateral thalami, posterior putamen, corticospinal tract and dorsal brainstem. On T2WI, loss of normal hypointensity of posterior limb of internal capsule can be seen which is bad prognostic sign for future neurodevelopment . On proton MR Spectroscopy, NAA will be reduced maximally by 3 to 4 days. By 7 to 10 days, sonography shows hyperechogenicity.

Extent of injury is determined by severity of hypotension and duration of the ischemic event. With most severe injury of entire cerebral cortex, all deep nuclei, cerebellar vermis and brainstem are involved.

Transcranial neurosonography

Neonatal neurosonography is considered as an initial imaging modality particularly in high risk and unstable premature babies in ICU setup. Advantages of sonography includes portability, affordability, speed and reproducibility, non-ionizing and no sedation is required. Fontanelle are the spaces between infant skull bones that are covered by a tough fibrous membrane. Anterior fontanelle is seen at the junction between frontal and parietal bone which remains open up to 9 to 18 months. Posterior fontanelle is junction between two parietal bones and occipital bone which is earlier to close and opens up to 3 months. Other fontanelle useful for imaging include mastoid fontanelle which is a membranous portion between parietal bone, petrous part of temporal bone, occipital bone which closes by 12 to 18 months and lambdoid fontanel[84]. Other additional viewing areas for neurosonography include temporal bone.

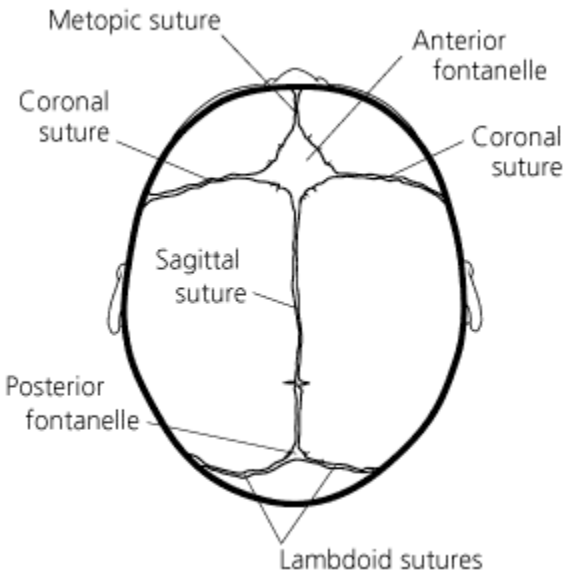


Figure 1: Normal skull sutures and fontanellae

Best results on ultrasound are obtained by using high frequency linear array transducers 7.5 MHZ or more. Transducer is used at a 120 degree sector angle with a small foot print. It is essential to maintain body temperature while performing neurosongraphy examination. Gentle handling of the babies required and a warm

sterile coupling gel should be used. Hand washing and cleaning transducer is very important to avoid the spread of infections[33].

Most brain sonographic examinations are done through anterior fontanellar windows in both coronal and sagittal planes. Coronal views are obtained by placing transducer transversely across anterior fontanella and atleast in 6 standard view planes should be examined. Six coronal views include (at the level of the both orbits, middle cerebral arteries, third and fourth ventricles, bodies of lateral ventricles and centrum semiovale) along with five sagittal-parasagittal views (midline section, at the level of ventricles and periventricular white matter areas) should be obtained through the anterior fontanelle[83].

Posterior fontanelle imaging is useful to evaluate occipital horns of lateral ventricles. Mastoid fontanelle is used for assessment of brain stem and posterior fossa pathologies. In mastoid view, Probe should be kept 1 cm behind the tragus. However, ultrasound has low sensitivity in evaluating early germinal matrix hemorrhage and white matter injury.

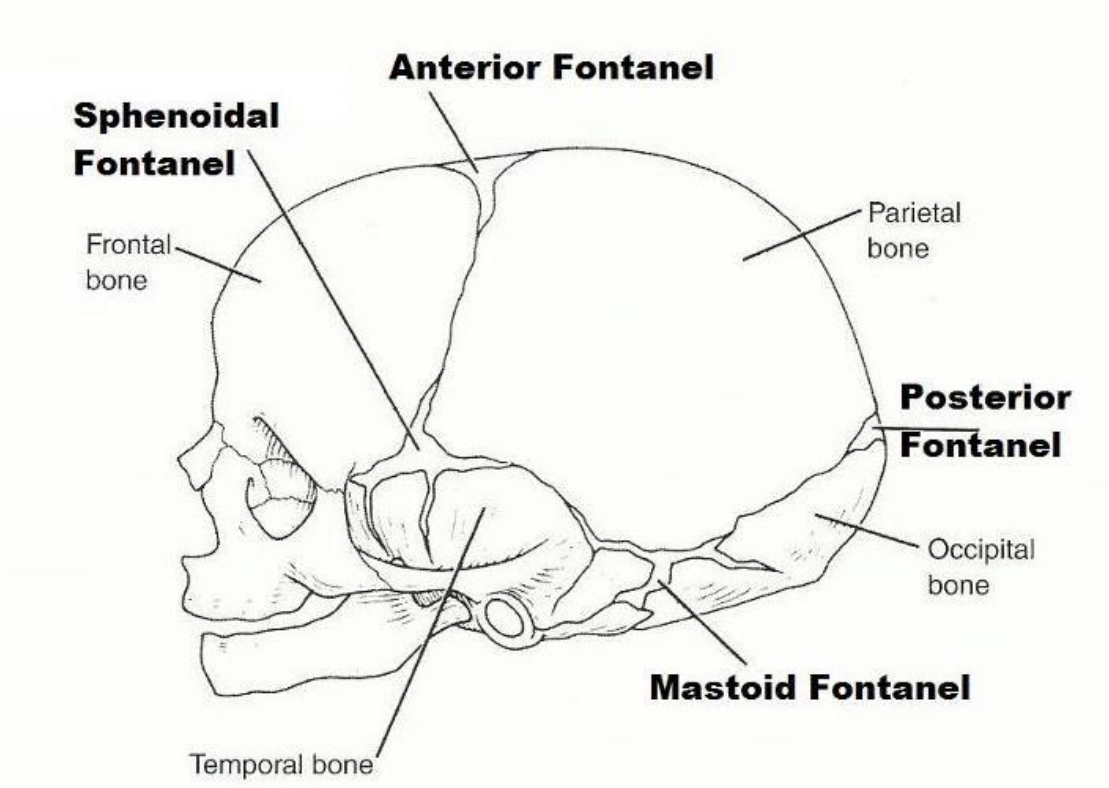


Figure 2: Approach to Transcranial neurosonography

Doppler imaging

Doppler imaging of circle of Willis and ICA, ACA, MCA, and cerebral venous sinuses should be used as a regular part in scanning protocol along with B-mode. Both ACA and MCA R.I are considered as significant predictors for neurological outcome[2].

Doppler ultrasound provides detailed evaluation of hemodynamics and predicts the changes of blood flow after respiratory distress[85]. Early cerebral edema can present as diffusely hyperechoic parenchyma with effaced lateral ventricles. Anterior and Middle cerebral arteries are evaluated on color Doppler. It is essential to evaluate flow and spectral tracing, resistive index of ACA, MCA. Normal RI values are 0.65 to 0.9. Cerebral venous sinus thrombosis can be evaluated as filling defect in superior sagittal sinus.

Computed tomography

Multidetector CT is a rapid mode of screening .CT is considered as the least sensitive imaging modality. This is due to the high protein concentration in cerebrospinal fluid (CSF) and high water content within brain[86]. It is considered as an intermediate between neurosonography and MR imaging .However, it is considered as the best and rapid imaging modality in evaluating hemorrhage without sedation. Neonate can be easily monitored during the examination .Presence of ionizing radiation is the main disadvantage and is least useful in preterm neonates[72].

MRI brain in neonate

The most sensitive and specific imaging technique for examining infants with suspected hypoxic-ischemic brain injury is MR imaging and is useful in identification of sequelae of an ischemic injury in neonates within the first few hours and days following the ischemic event. MRI also helps to exclude other causes of encephalopathy such as cerebral hemorrhage, infarction, intracranial neoplasms and congenital brain malformations. Newer imaging techniques like GRE, DWI and MRS are more accurate in diagnosing acute brain injury.

FLAIR sequence is essentially useful for demonstrating periventricular cystic leukomalacia and gliosis. Gradient recalled echo-T2*-weighted sequence or susceptibility weighted imaging is particularly sensitive for detecting intracerebral hemorrhage and distinguishing it from astrogliosis. DWI sequence demonstrates cytotoxic edema (due to hypoxic cerebral injury) in acute phase i.e., within 1- 8 days before the signal intensity changes that are evident on conventional T1 or T2W images.

However, the main limitation of DWI is, if performed within first 24 h of HI injury it may give false negative result. MR Spectroscopy performed within first 24 h after birth in a full-term neonate is highly sensitive to detect the severity of HI brain injury than DWI and can predict adverse outcome better.

Advantages of MR

The main advantages of magnetic resonance imaging (MRI) scan include:

1) It is a non-invasive technique, producing high quality, diagnostically

interpretable images with good spatial resolution.

2) Non- ionizing radiation, as they do not involve exposure to radiation and can be safely used in people who might be particularly vulnerable to the effects of radiation, such as neonates and pregnant women.

3) Better soft tissue resolution and discrimination in any imaging plane.

4) Provide both morphological and functional information.

5) Excellent depiction of anatomy and pathology.

6) High spatial & temporal resolution.

7) Depiction of vascular anatomy without use of contrast.

8) Chemical analysis of tissue (MR spectroscopy).

9) Multiplanar acquisition

Disadvantages of MRI:

The main disadvantages of magnetic resonance imaging (MRI) scans include:

1) MRI scanners are very expensive and limited access
2) Needs sedation in neonates and is difficult in ventilator supported infants
3) Patient being put in an enclosed space and the loud noises that are made by the magnets should be avoided in claustrophobic patients.
4) MRI can be associated with movement and produce less clear images making unsuitable in unstable patients.
5) Orthopedic hardware (screws, plates, artificial joints) in the area of a scan can cause artefacts and severe distortion in the images.
6) People with kidney disease or on dialysis are likely to develop Nephrogenic Systemic Fibrosis (NSF) from the MRI contrast after injection of gadolinium.

CLINICAL MANIFESTATIONS

The birth asphyxia neonate may have low Apgar scores (slow heart rate, poor respiratory effort, abnormal color, decreased level of alertness, abnormal muscle tone, and a weak or absent cry) at delivery and low pH in the cord blood, apnea and seizures with abnormal electroencephalography (EEG). The Sarnat and Sarnat staging system is useful in classifying the degree of encephalopathy.

Level of consciousness	Stage 1 (mild) Hyperalert	Stage 2 (moderate) Lethargic/obtunded	Stage 3 (severe) Stuporous
Neuromuscular control			
Muscular tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch	Overactive	Overactive	Decreased/absent
Segmental myoclonus	Present	Present	Absent
Complex reflexes			
Suck	Weak	Weak/absent	Absent
Moro	Strong	Weak	Absent
Oculovestibular	Normal	Overactive	Weak/absent
Tonic neck	Slight	Strong	Absent
Autonomic function			
Pupils	Mydriasis	Miosis	Variable
Heart rate	Tachycardia	Bradycardia	Variable
Bronchial/salivary secretions	Sparse	Profuse	Variable
Gastrointestinal motility	Normal/decreased	Increased/diarrhea	Variable
Seizures	None	Common/focal or multifocal	Uncommon
EEG	Normal/decreased	Early low voltage continuous delta and theta, later periodic, seizues focal 1–1.5 Hz spike-wave	Early periodic pattern with isopotential phases, later isopotential
Duration	<24 h	2–14 days	Hours–weeks

Table 1: Sarnat and Sarnat clinical staging

REVIEW OF LITERATURE

Wei shen and jia hua et al (2015) in their study - comparison of trancranial ultrasound and cranial MRI in evaluation of brain injuries from neonatal asphyxia, 30 asphyxia full term neonates were selected. Results were analysed and compared brain edema was observed in severe injury cases with ultrasound as major finding. Doppler showed RI changes with less than 0.5 or more 0.9 in severe asphyxia cases. DWI was more sensitive modality in picking up early post asphyxia cerebral edema than conventional MRI[10].

Babiker et al (2006) conducted a study on comparing cranial magnetic resonance imaging (MRI) and cranial sonography (US) in 150 neonates with suspected HIE. Magnetic resonance imaging findings were normal in 44 patients (29%); 18% of patients showed only basal ganglia brightness, 10.6% showed intracerebral hemorrhage, and 63% of patients showed additional diagnostic details. Cranial US was normal in 75 patients (50%) and showed increased periventricular echogenicity in 32%, intraventricular hemorrhage in 9%, and additional diagnostic details in 13% and concluded that US is a worthwhile modality for the diagnosis of HIE but early MRI findings will provide additional information in many cases in the detection of cerebral intraventricular hemorrhage[6].

Epelman et al (2010) in their study neonatal encephalopathy prospective comparison of head evaluation of hie in preterm neonates using transcranial ultrasound.US with MRI, a total of 76 cases were included MRI was considered positive in 53 cases diagnostic accuracy of USG was 95 % and concluded that USG is still regarded as screening tool in neonates , more attention should be paid for proper technique . MRI shows disease more extensively[11].

A study conducted by Kamal and Hassan et.al (2019) on comparison of transcranial ultrasound and MRI in hypoxic ischemic injury neonates in 36 neonates showed MRI imaging is most sensitive modality in detecting different MR patterns in newborn with birth asphyxia. MRI is considered for early assessment and early predictor of neurological sequelae in neonates with HIE[94].

Basavaraj patil et al (2015) in their study clinicoradiological correlation in birth asphyxia evaluated 37 neonates they concluded that sensitivity of USG in detecting

findings in HIE II is 4.7 % and in stage III 18.7 % while for MRI it was 52.3 % and 87.5 % respectively[60].

Ramachandran S et al (2017) conducted a study to evaluate the role of MRI in infants with suspected hypoxic ischemic encephalopathy and prognosticating neurological outcome in 50 neonates at end of one year and concluded that the sensitivity of MRI was 72% and specificity was 71% and Concluded that MRI is a useful modality to assess early changes in HIE and it can prognosticate clinical outcome[95].

Genadi et al (2016) in their study magnetic resonance imaging versus trancranial ultrasound in early identification of cerebral injuries in neonatal encephalopathy , 38 newborns with neonatal encephalopathy are evaluated, brain MRI was positive in 33 cases with hypoxia as etiology . Diagnostic accuracy of TCUS was 78 % compared to MRI. Concluded that USG is a good screening tool in detecting lesions however MRI is mandatory as it can detect precisely the extent of brain injury compared with TCUS alone[16].

Maalouf and Duggan et al (2001) conducted a comparative study of cranial ultrasound and MRI in preterm neonates with hypoxia and evaluated 62 neonates, Ultrasound has demonstrated the same MRI findings in GMH. Other MRI findings of small petechial hemorrhages, diffuse extensive signal injury are less predicted on Ultrasound and concluded that normal cerebral white matter echogenicity on ultrasound is not a good predictor of normal white matter signal on MRI[15].

Intrapiromkul et al (2013) in their study for evaluating the accuracy of head ultrasound in the detection of intracranial hemorrhage in preterm neonates: On Comparison with brain MRI and susceptibility-weighted imaging evaluated 12 neonates with germinal matrix and found USG had high sensitivity (100%) and specificity (93.3%) in detecting grade III GMH using SWI as a reference, but poor sensitivity (0%) in the detection of intraventricular hemorrhage with normal-sized ventricles (grade II GMH). US was not sensitive in detecting either small cerebellar or extra-axial hemorrhage[21].

Pinkesh, Jagruti et al (2018) conducted a study of Transcranial Ultrasound for Detection of Hypoxic Brain Injury in 50 Neonates with history of birth asphyxia and evaluated by bedside transcranial ultrasound. Out of 50 patients 41 patients were

positively identified by transcranial ultrasound which further confirmed clinically and radiologically and concluded that Bedside transcranial ultrasound appears to be very sensitive and promising in early detection and management of the hypoxic brain injury and proves to be valuable screening tool for hypoxic brain injury[35].

MATERIALS AND METHODS

METHOD OF COLLECTION OF DATA:

The study will be conducted on a minimum of 54 sample patients with complaints of low APGAR score, delayed cry and clinical features of birth asphyxia who are referred to Department of Radio diagnosis, Shri B.M. Patil Medical College Hospital and Research Center during the period of Nov 2019 to June 2021.

Before evaluating a patient, informed consent will be obtained from the guardian.

Method of collection of data (including sampling procedure, if any)

· Patients with eligible inclusion criteria are considered and underwent following method of procedures.

· 54 cases are intended to be taken up within the study period.

INCLUSION CRITERIA:

-Newborn (preterm and term babies) with perinatal insult and feto-maternal high risk factors.

-Hemodynamically stable neonates with seizures, bulging anterior fontanelle, fetal distress and low APGAR score.

EXCLUSION CRITERIA:

-Hemodynamically unstable neonates with septic shock and moribund status. -Neonates on ventilatory support.

-Neonates born with congenital anomalies, congenital infections and proven cases of metabolic diseases are excluded.

CONSENT:

Informed consent was taken from all parents/guardians who were selected on the basis of history and clinical criteria.

TECHNIQUES OF DATA COLLECTION:

All patients clinically suspected of birth asphyxia underwent transcranial neurosonography and Magnetic resonance imaging.

TRANSCRANIAL ULTRASONOGRAPHY PROTOCOL

All neonates included in the study will undergo Transcranial ultrasonography evaluating on B-mode and Color Doppler imaging with high frequency probes (3 - 12 MHz) using anterior fontanella, mastoid and temporal window views. Most of the images are obtained in sagittal and coronal views. The Ultrasound machines which are used in the study are PHILIPS HD11-XE and GE VIVID T8. The neonates with imaging findings of birth asphyxia on ultrasound are subjected to either CT / MRI for comparison and further correlated with the clinical findings. Emergency resuscitation equipment, infusion pump and endotracheal tubes are kept handy.

MRI PROTOCOL:

The neonate is sedated using pedicloryl syrup or short acting benzodiazepines under paediatrician supervision. Oxygen is provided for unstable neonates, with careful monitoring of vitals, baby is placed in supine position .GE SIGNA 1.5 TESLA MRI machine is used for this study and the sequence planning protocol includes-

- T1 FSE axial,
- T2-FLAIR axial,
- T2 FSE axial,
- DWI and ADC maps,
- GRE sequence.

The section thickness of 3 -6 millimeter. Only plain MRI studies was conducted without using intravenous contrast.

In my study, all the neonates underwent transcranial neurosonogram and these neonates are further subjected to MRI for further evaluation.

SOURCE OF DATA

Data for the study will be collected from neonates presenting with clinically suspected cases of birth asphyxia attending the OPD or admitted in NICU and PICU Paediatric wards who are referred to The Department of Radiodiagnosis department of B.L.D.E.U's Shri. B.M. Patil Medical College Hospital and Research Centre, Vijayapur.

PERIOD OF COLLECTION OF DATA

The study was done on patients, who visited the Department of Radio Diagnosis during the period from NOVEMBER 2019 to JUNE 2021 with prior consent.

STUDY DESIGN:

A Hospital based Cross-sectional study.

SAMPLE SIZE

With anticipated sensitivity and specificity of TCUS in correlation with MRI 80% and 67% resp [ref] , at 95% confidence level, with prevalence 80% and precision of 0.12 the sample size calculated is 54 per group Using formula.

$$N=\frac{Z^2P(1-p)}{\Delta^2}$$

N will be (a+c) if we use sensitivity as p

N= (a+c)/Prevalence

STATISTICAL ANALYSIS

- Numerical variables will be presented as Mean ±SD, and categorical variables will be presented as frequency(%) and diagrams
- Comparison of numerical variables between groups will be found using unpaired t test/Mann whitney U test , and categorical variables by Chi square or Fisher's Exact test.
- Diagnostic tests will be performed using Sensitivity, specificity, Positive predictive Negative predictive values and Accuracy.

Statistical methods used

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean±standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables.

The formula for the chi-square statistic used in the chi square test is:

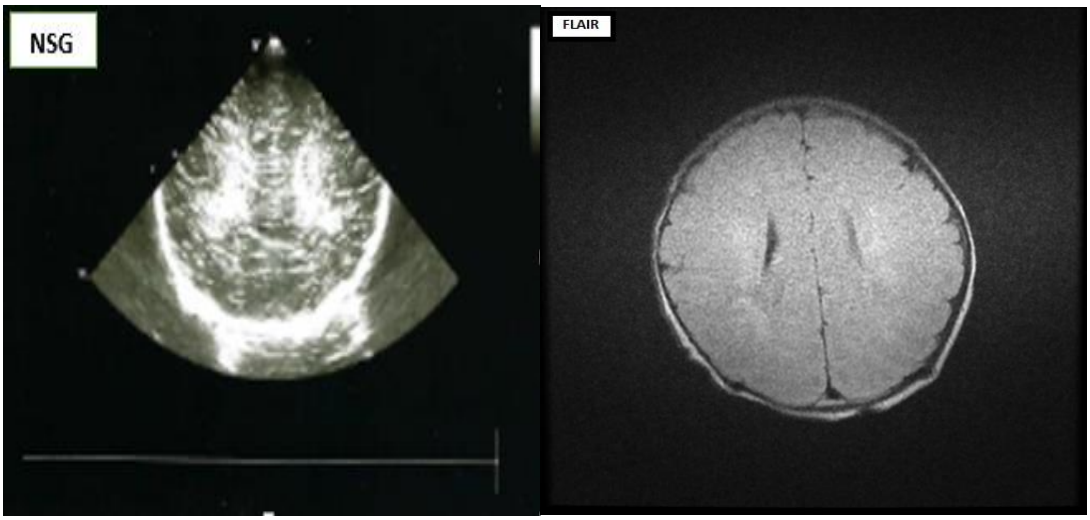
$$\chi_c^2 = \sum \frac{(O_i - E_i)^2}{E_i}$$

The subscript "c" are the degrees of freedom. "O" is observed value and E is expected value. C= (number of rows-1)*(number of columns-1)

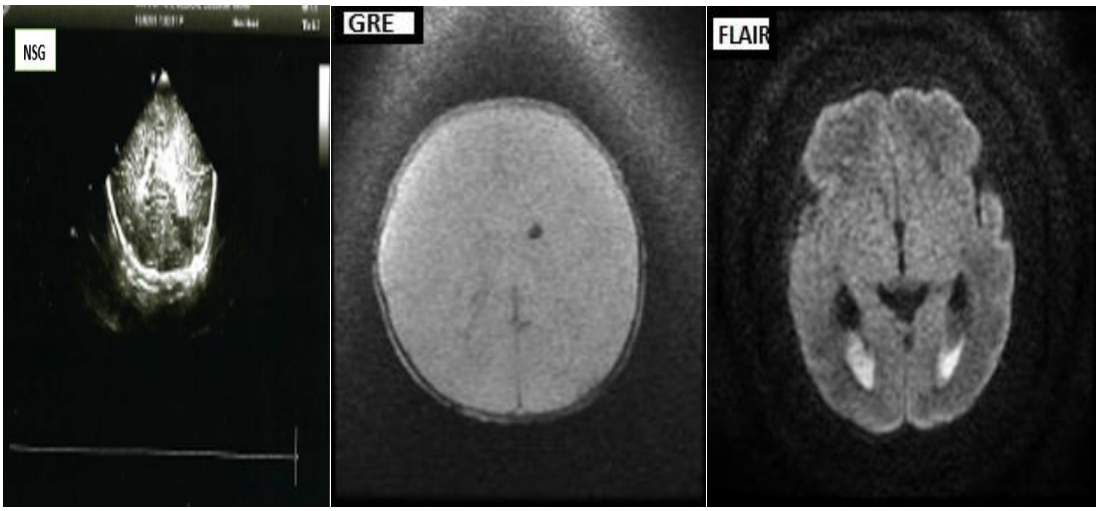
sensitivity or true positive rate (TPR)
eqv. with hit rate, recall
$$TPR = TP/P = TP/(TP + FN)$$
specificity (SPC) or true negative rate
$$SPC = TN/N = TN/(FP + TN)$$
precision or positive predictive value (PPV)
$$PPV = TP/(TP + FP)$$
negative predictive value (NPV)
$$NPV = TN/(TN + FN)$$

If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data was analyzed using SPSS software v.23 (IBM Statistics, Chicago, USA) and Microsoft office 2007.

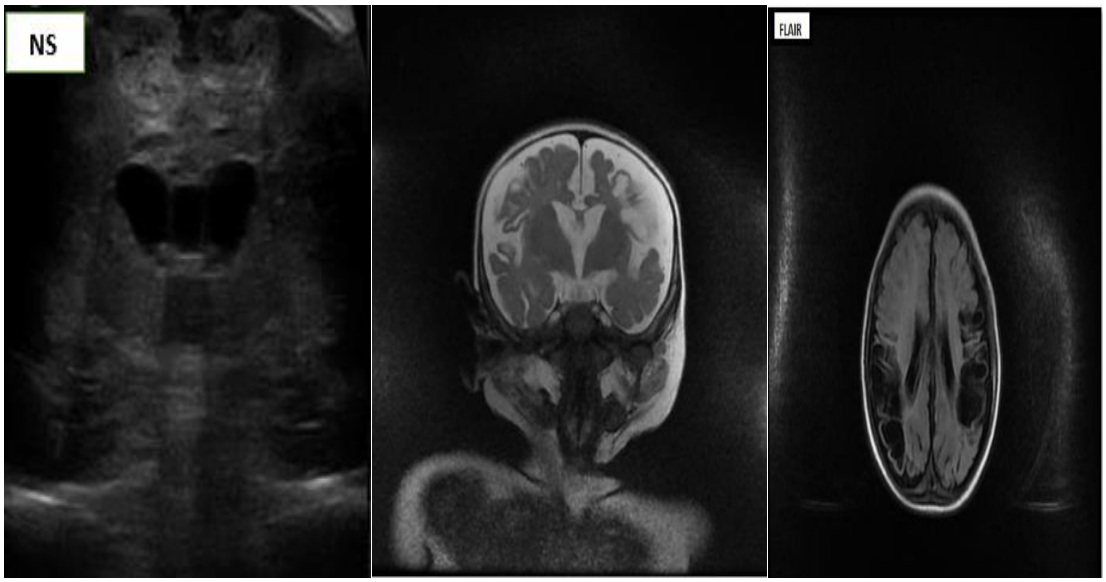
IMAGING GALLERY



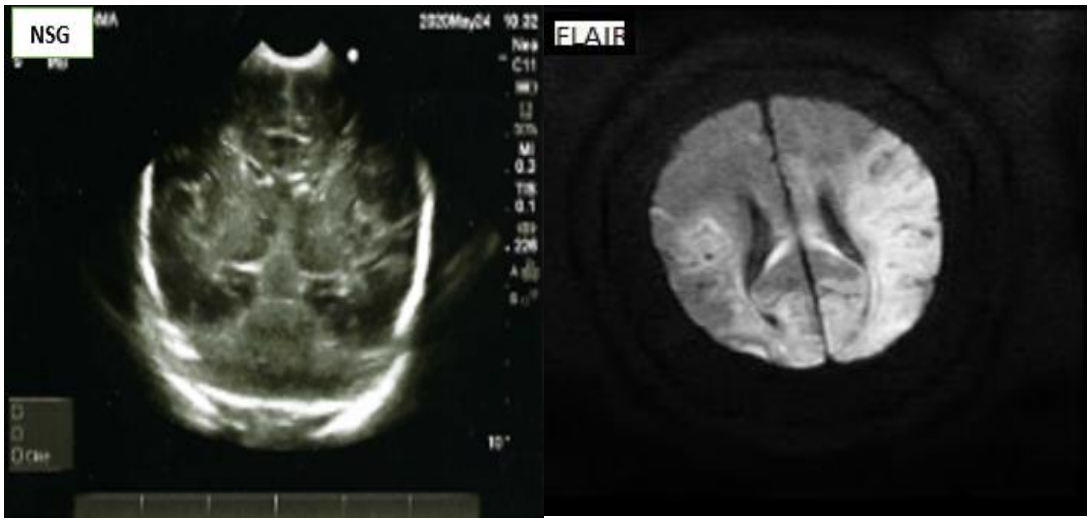
Case 1: Transcranial neurosonography showing bilateral periventricular white matter echogenicity with corresponding FLAIR hyperintensities on MRI Brain suggesting Periventricular white matter injury.



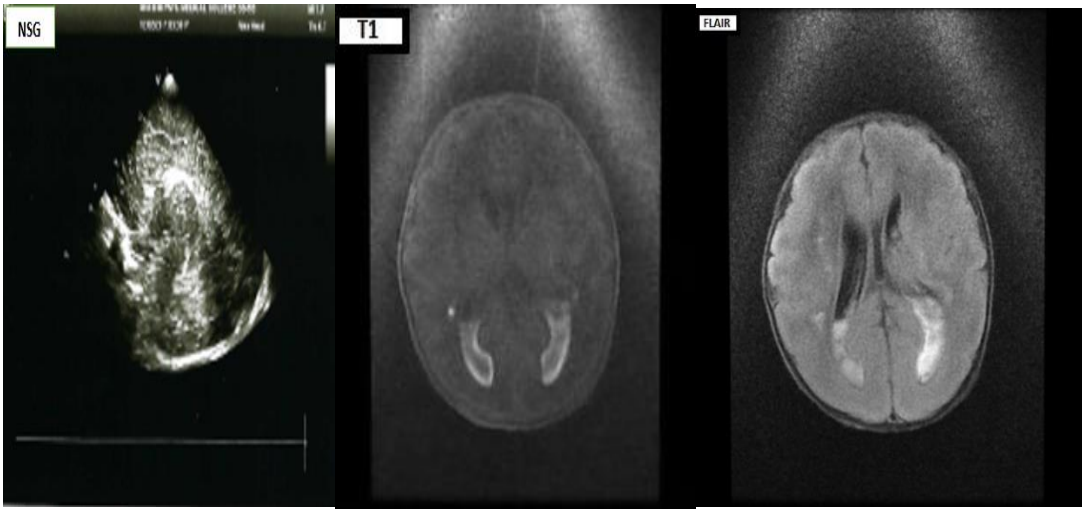
Case 2:Transcranial neurosonography showing left germinal matrix hemorrhage with intraventricular extension with corresponding blooming on GRE with FLAIR hyperintensities in the occipital horns of bilateral lateral ventricles on MRI Brain suggesting Grade II Germinal matrix hemorrhage.



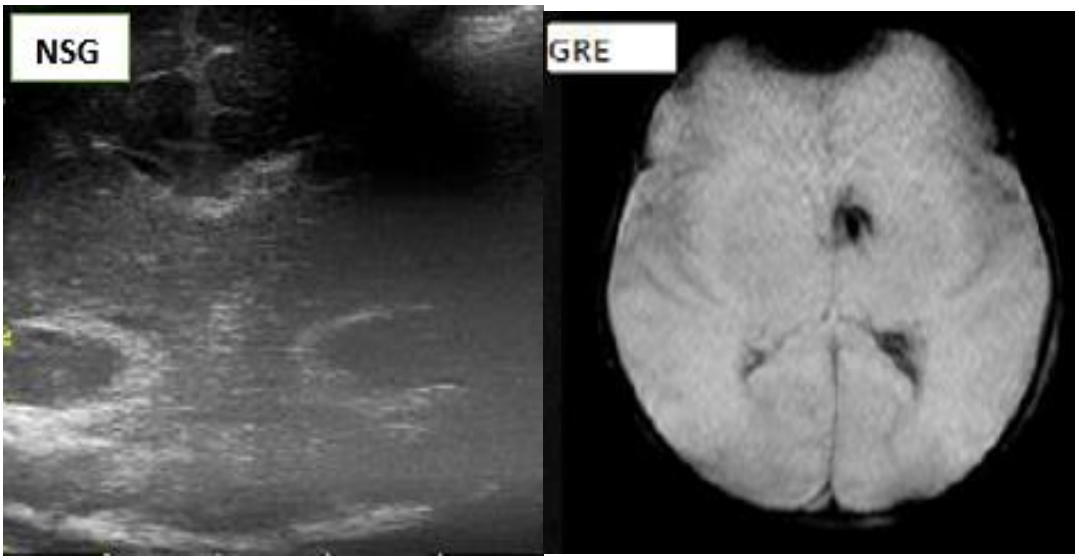
Case 3: Transcranial neurosonography showing multiple white matter cystic areas with corresponding cavitatory white matter disease changes on coronal T2 and axial FLAIR images suggesting severe periventricular white matter disease with leukomalacia (PVWM IV).



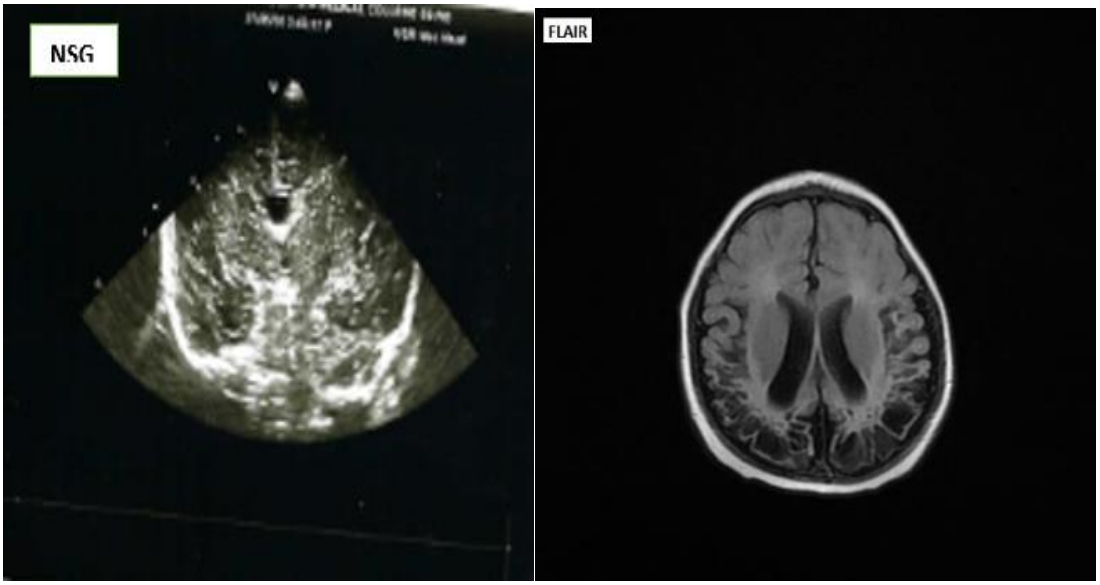
Case 4: Transcranial neurosonography showing diffuse cerebral edema with few hypoechoic areas in frontotemporo-parietal region corresponding to hyperintensities on axial FLAIR images and restricted diffusion on DWI suggesting severe periventricular white matter disease (Global ischemic injury).



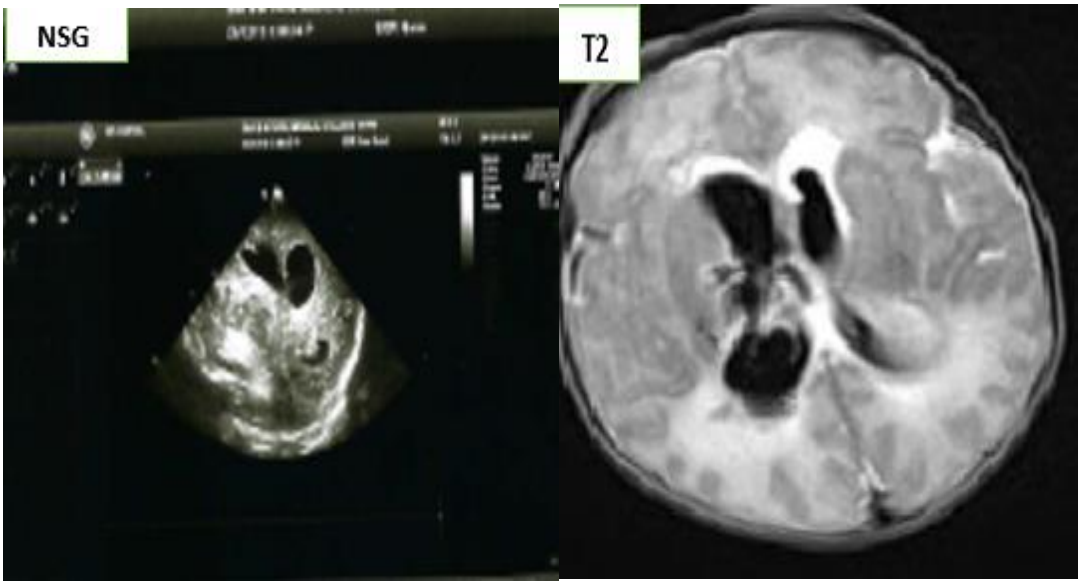
Case 5:Transcranial neurosonography showing germinal matrix hemorrhage with intraventricular extension and corresponding T1/ FLAIR hyperintensities in the occipital horns of bilateral lateral ventricles on MRI Brain suggesting Grade II Germinal matrix hemorrhage.



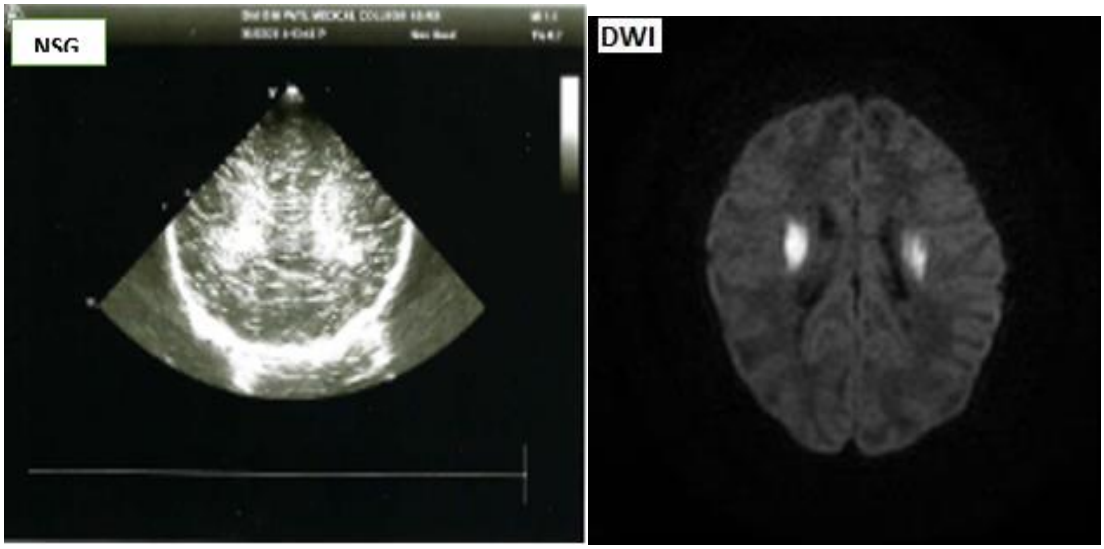
Case 6:Transcranial neurosonography showing left germinal matrix hemorrhage and corresponding blooming on GRE is noted in MRI Brain suggesting Grade I Germinal matrix hemorrhage.



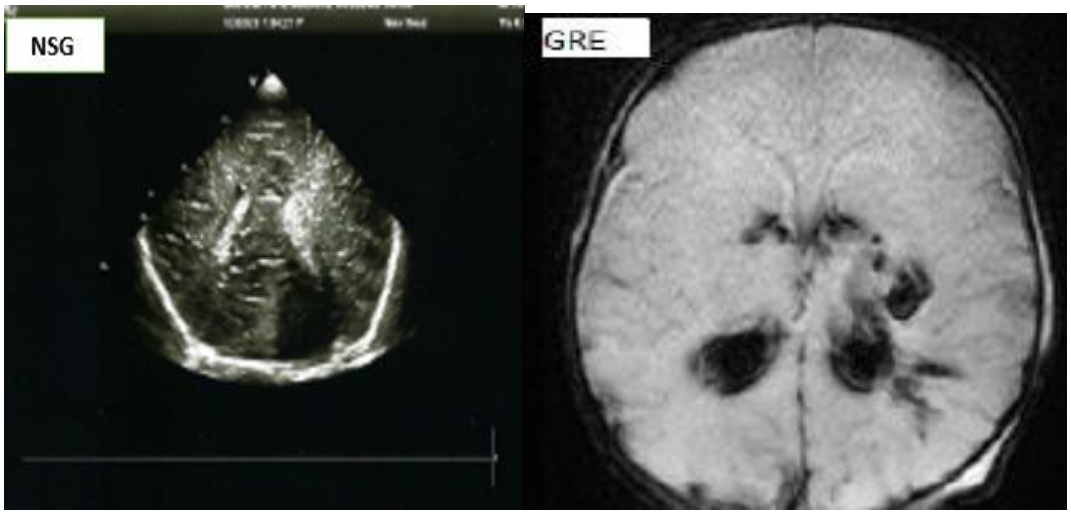
Case 7: Transcranial neurosonography showing multiple white matter cystic areas with corresponding cavitatory white matter disease changes on axial FLAIR images suggesting severe periventricular white matter disease with leukomalacia (PVWM IV).



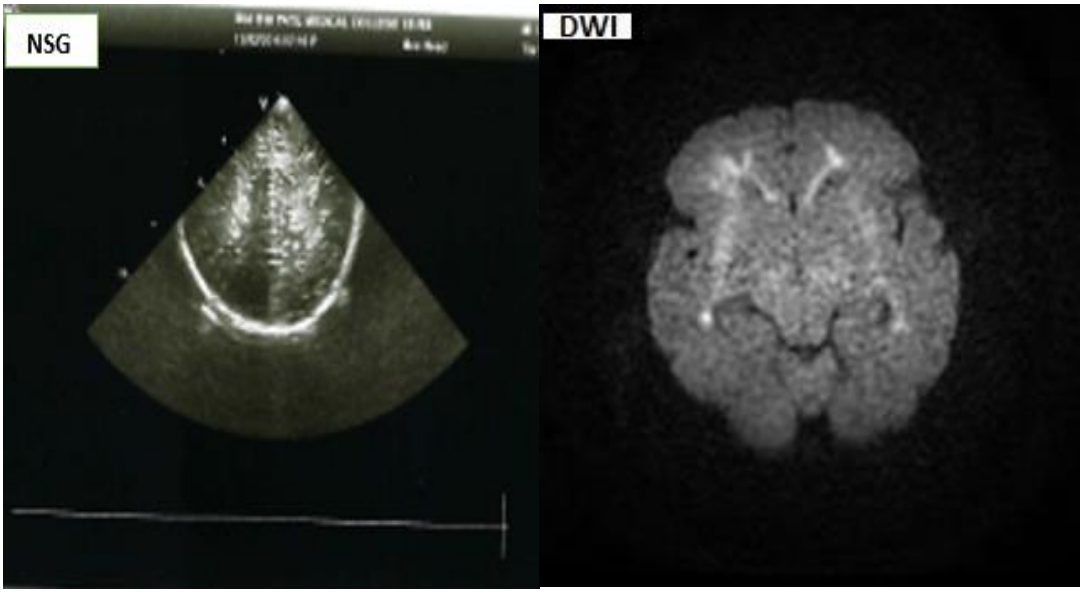
Case 8:Transcranial neurosonography showing right germinal matrix hemorrhage with intraventricular extension and mild ventricular dilatation with corresponding blooming on GRE with FLAIR hyperintensities in the occipital horns of bilateral lateral ventricles on MRI Brain suggesting Grade III Germinal matrix hemorrhage.



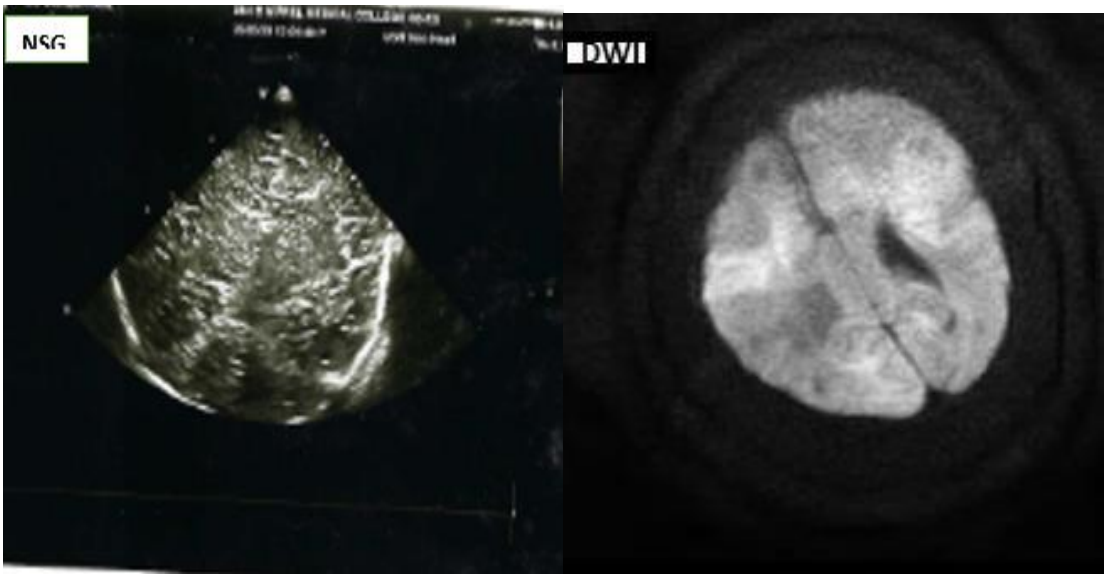
Case 9: Transcranial neurosonography showing periventricular echogenic areas in fronto-parietal region corresponding to areas of restricted diffusion on DWI suggesting severe periventricular white matter disease (Periventricular white matter injury).



Case 10:Transcranial neurosonography showing left germinal matrix hemorrhage with intraventricular extension and intraparenchymal echogenic areas with corresponding blooming on GRE on MRI Brain suggesting Grade IV Germinal matrix hemorrhage.



Case 11: Transcranial neurosonography showing periventricular echogenic areas in fronto-parietal region corresponding to areas of restricted diffusion on DWI in watershed areas and bilateral capsuloganglionic regions suggesting severe periventricular white matter disease (Periventricular white matter injury).



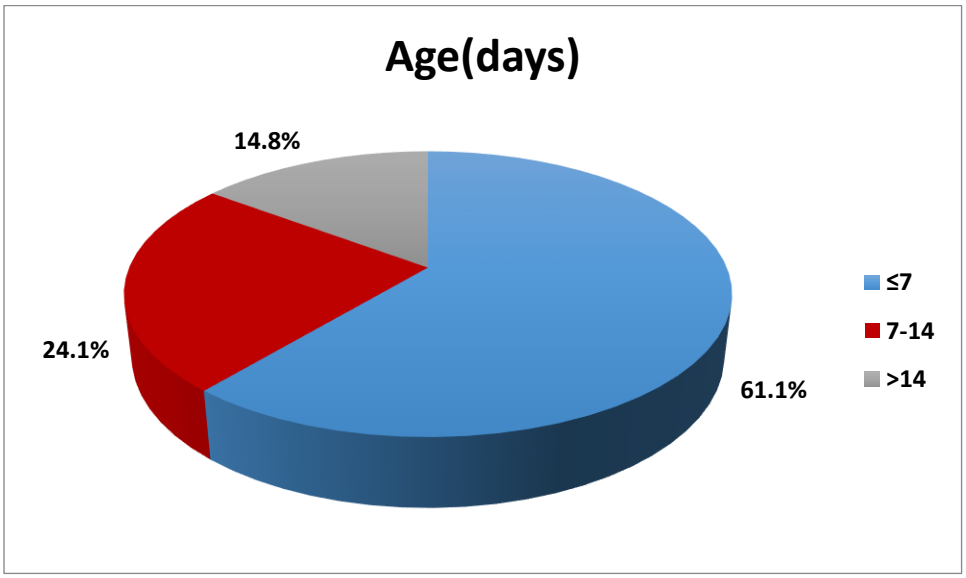
Case 12: Transcranial neurosonography showing periventricular echogenic areas in fronto-parietal region corresponding to areas of restricted diffusion on DWI in watershed areas suggesting severe periventricular white matter disease (Periventricular white matter injury).

RESULTS OF STATISTICS AND ANALYSIS

Table 1: Distribution of Cases according to Age

Age(days)	N	Percent
≤7	33	61.1
7-14	13	24.1
>14	8	14.8
Total	54	100

Figure 1: Distribution of Cases according to Age

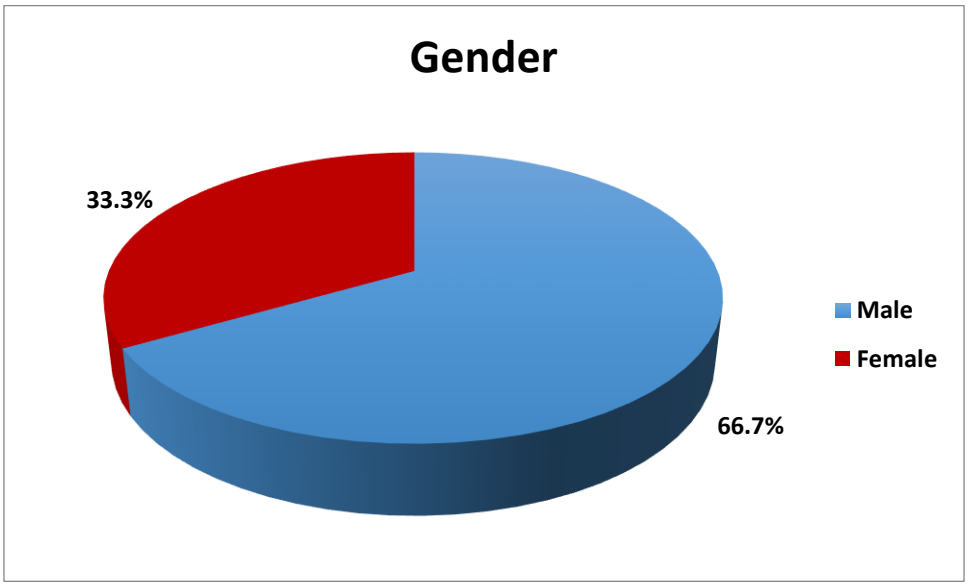


In the present study, majority of cases were presented within 7 days of post natal life.

Table 2: Distribution of Cases according to Gender

Gender	N	Percent
Male	36	66.7
Female	18	33.3
Total	54	100

Figure 2: Distribution of Cases according to Gender

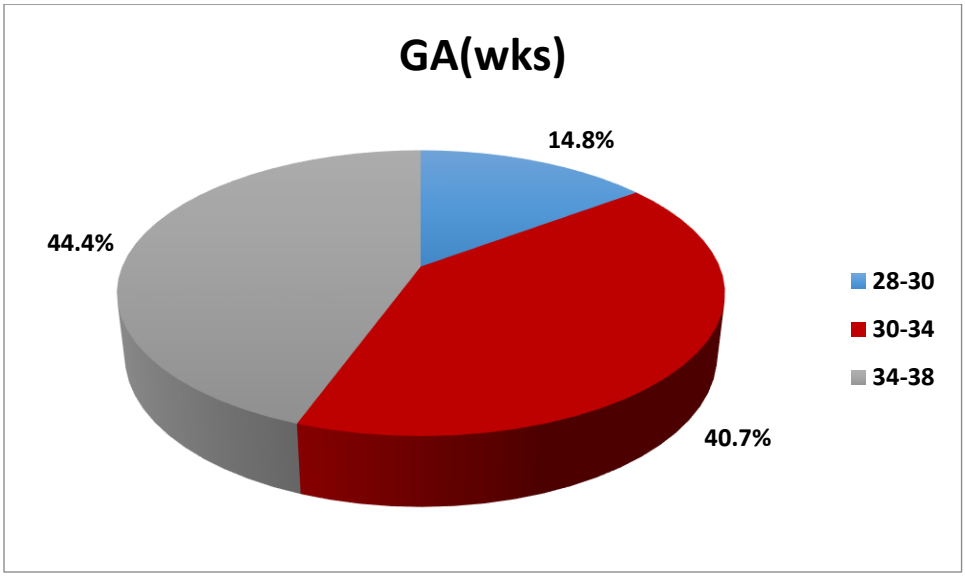


In the present study, majority of cases were male neonates accounting for 67 % and female neonates were 33 %.

Table 3: Distribution of Cases according to GA

GA(wks)	N	Percent
28-30	8	14.8
30-34	22	40.7
34-38	24	44.4
Total	54	100

Figure 3: Distribution of Cases according to GA

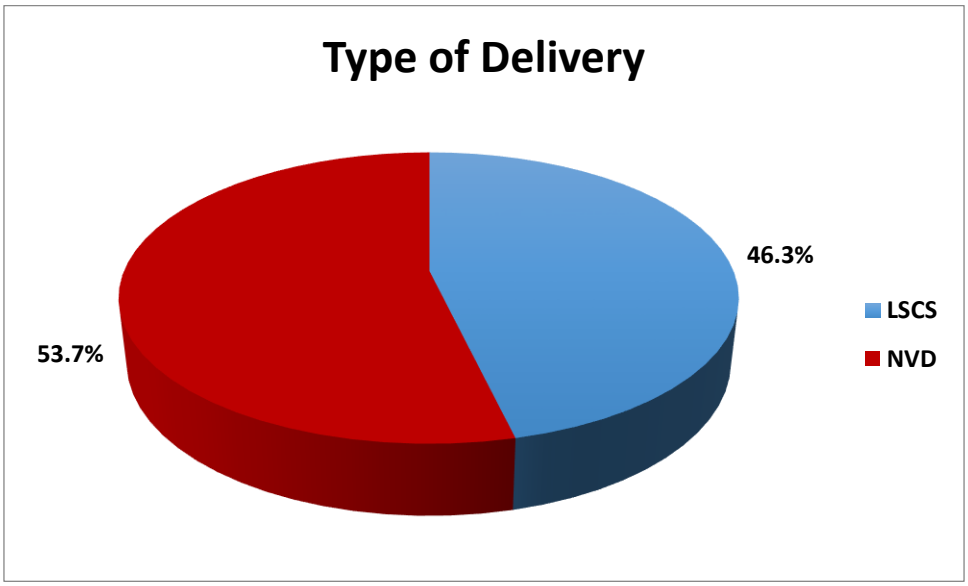


In the present study, majority of cases are presented at 34 – 38 weeks of gestational age accounting for 44 % .

Table 4: Distribution of Cases according to Type of Delivery

Type of Delivery	N	Percent
LSCS	25	46.3
NVD	29	53.7
Total	54	100

Figure 4: Distribution of Cases according to Type of Delivery

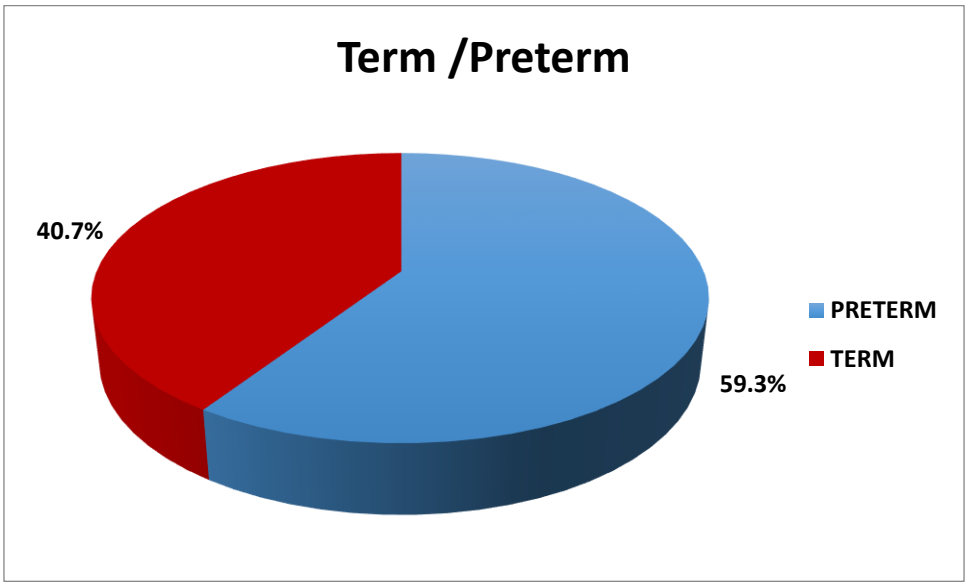


In the present study, 46 % of cases were delivered by LSCS and 54 % by NVD.

Table 5: Distribution of Cases according to Term /Preterm

Term /Preterm	N	Percent
Preterm	32	59.3
Term	22	40.7
Total	54	100

Figure 5: Distribution of Cases according to Term /Preterm

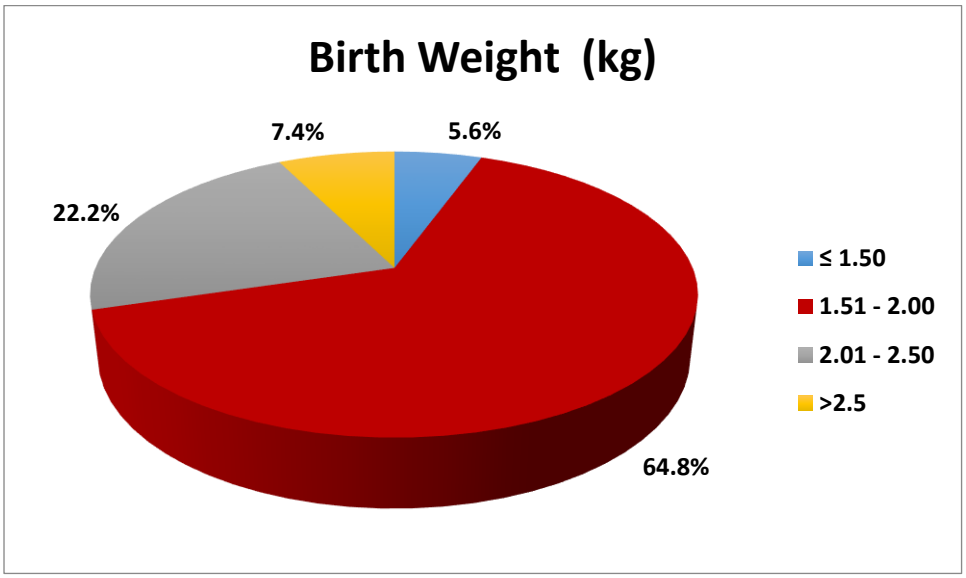


In the present study, majority of cases were term neonates accounting for 41 % and preterm neonates were 59 %.

Table 6: Distribution of Cases according to Birth Weight

Birth Weight (kg)	N	Percent
≤ 1.50	3	5.6
1.51 - 2.00	35	64.8
2.01 - 2.50	12	22.2
>2.5	4	7.4
Total	54	100

Figure 6: Distribution of Cases according to Birth Weight

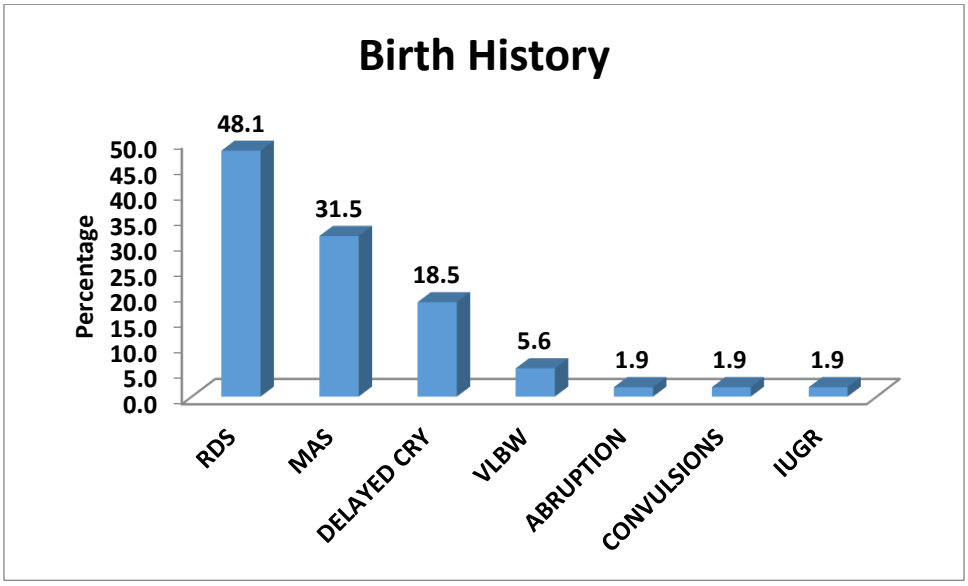


In the present study, majority of cases were around 1.51 – 2 kg accounting for 65 %.

Table 7: Distribution of Cases according to Birth History

Birth History	N	Percent
RDS	26	48.1
MAS	17	31.5
DELAYED CRY	10	18.5
VLBW	3	5.6
ABRUPTION	1	1.9
CONVULSIONS	1	1.9
IUGR	1	1.9

Figure 7: Distribution of Cases according to Birth History



In the present study, majority of cases presented with respiratory distress accounting for 48 %

Table 8: Distribution of Cases according to APGAR Score

APGAR Score	N	Percent
2	11	20.4
3	14	25.9
4	12	22.2
5	11	20.4
6	2	3.7
7	4	7.4
Total	54	100

Figure 8: Distribution of Cases according to APGAR Score

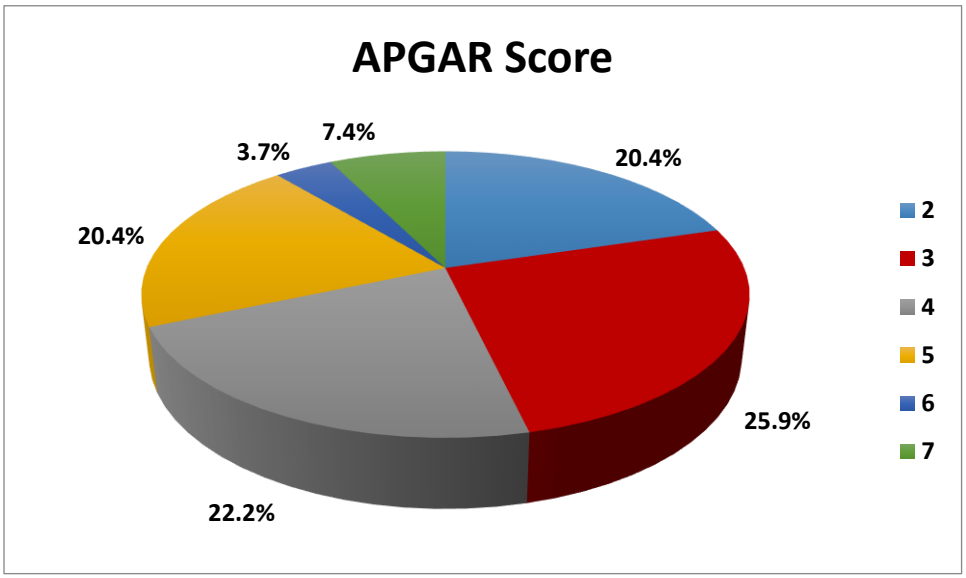
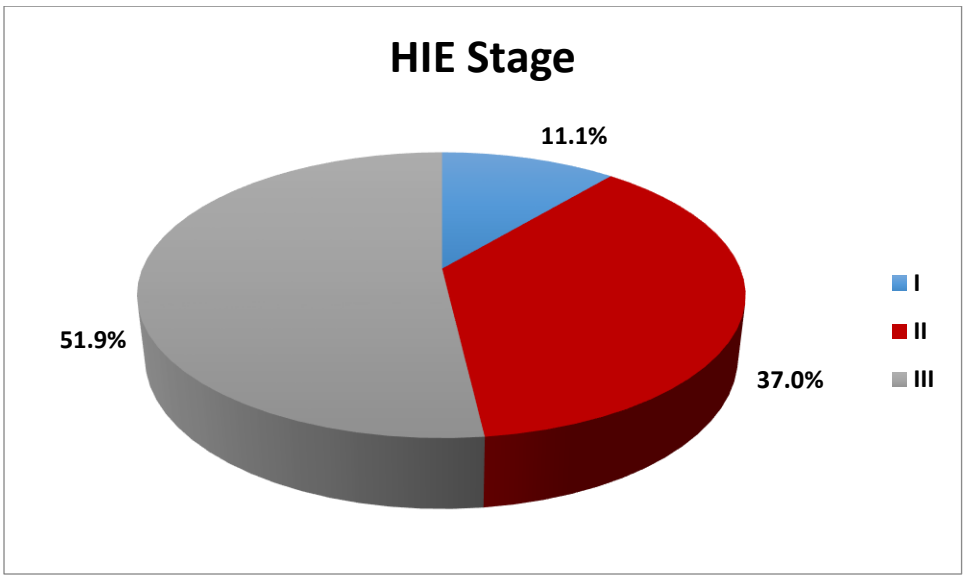


Table 9: Distribution of Cases according to HIE Stage

HIE Stage	N	Percent
I	6	11.1
II	20	37
III	28	51.9
Total	54	100

Figure 9: Distribution of Cases according to HIE Stage

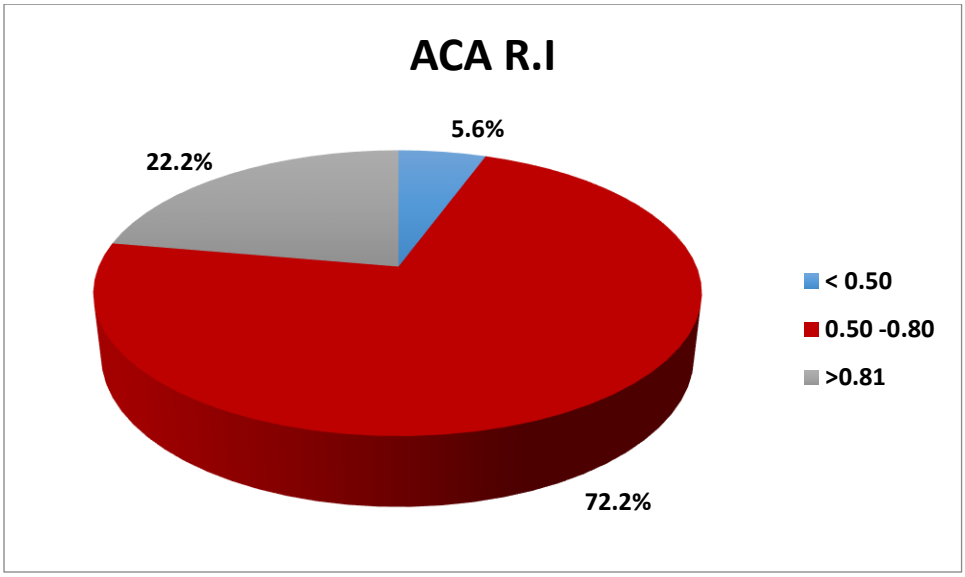


Out of 54 cases, HIE stage III cases comprised majority of the present study group – 28 cases (52%) followed by stage II (37%) , stage I (11%)

Table 10: Distribution of Cases according to ACA R.I

ACA R.I	N	Percent
< 0.50	3	5.6
0.50 -0.80	39	72.2
>0.81	12	22.2
Total	**54**	**100**

Figure 10: Distribution of Cases according to ACA R.I

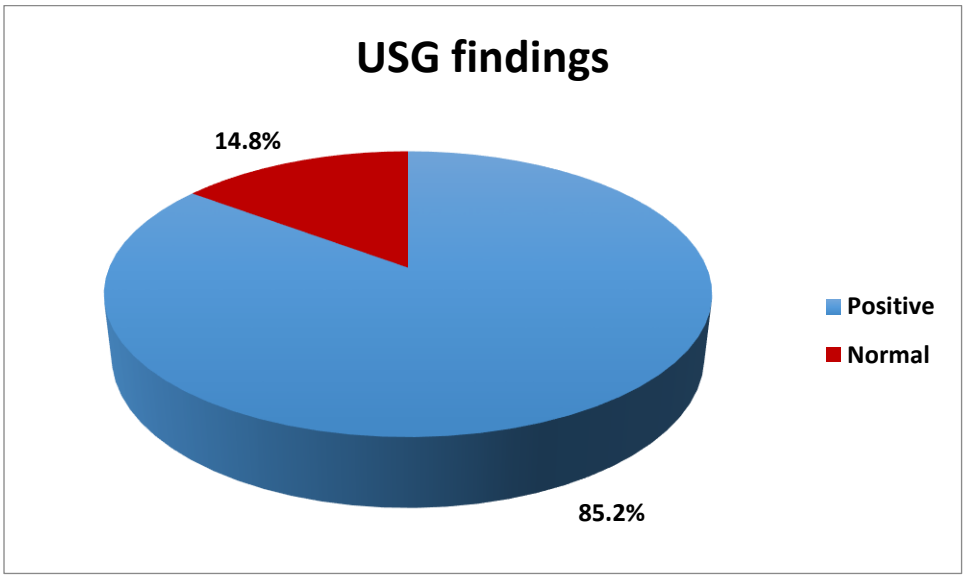


Out of 54 cases, Anterior cerebral artery showing R.I value of 0.5 – 0.8 comprised 72 %, and <0.50 and >0.81 comprised 28 %.

Table 11: Distribution of Cases according to USG findings

USG findings	N	Percent
Positive	46	85.2
Normal	8	14.8
Total	54	100

Figure 11: Distribution of Cases according to USG findings

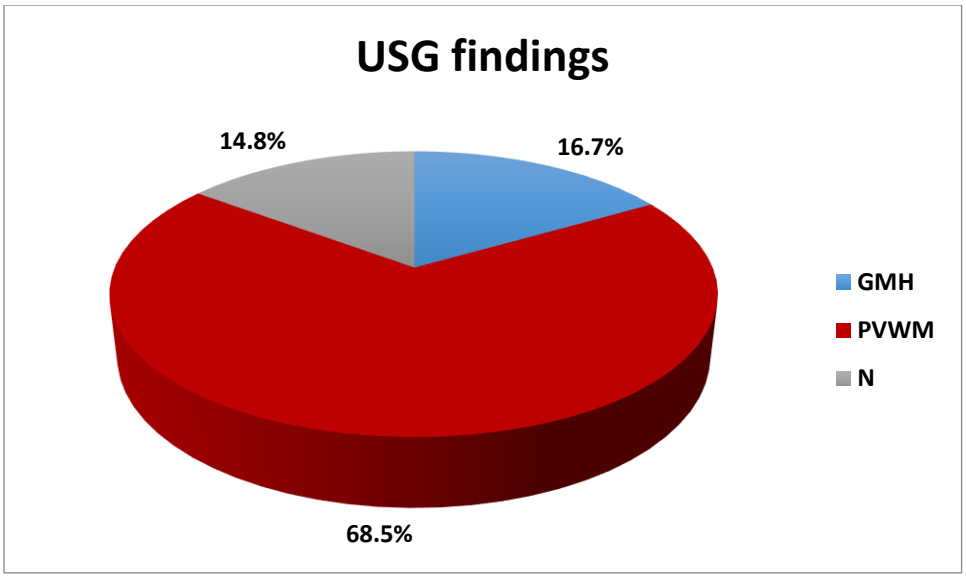


Out of 54 cases, Transcranial ultrasound was positive in 46 cases (86%) and normal were 8 cases (15 %).

Table 12: Distribution of Cases according to USG findings

USG findings	N	Percent
GMH	9	16.7
PVWM	37	68.5
N	8	14.8
Total	54	100.0

Figure 12: Distribution of Cases according to USG findings

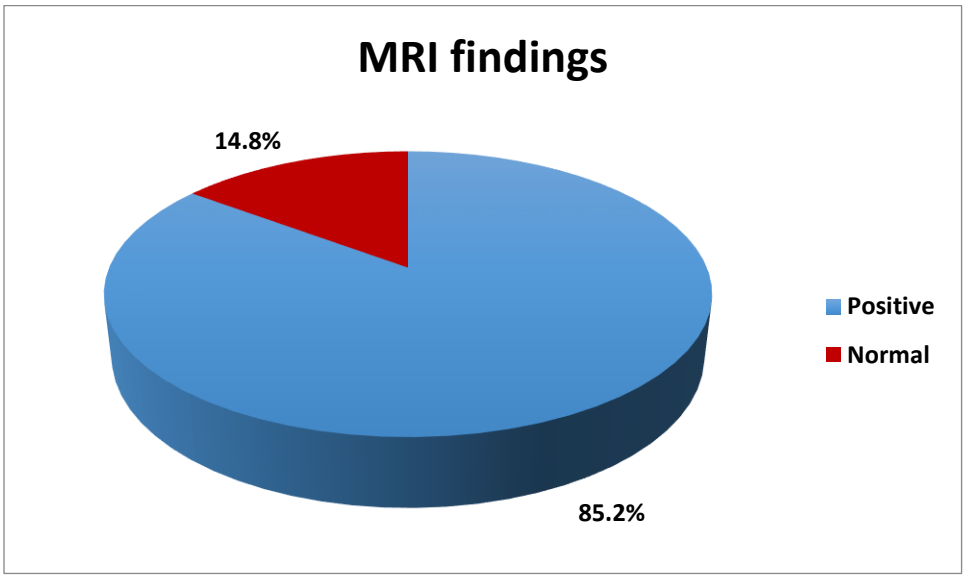


Out of 54 cases, on transcranial ultrasound PVWM cases comprised 37 cases (68.5%), GMH cases comprised 9 cases (16.7 %).

Table 13: Distribution of Cases according to MRI findings

MRI findings	N	Percent
Positive	46	85.2
Normal	8	14.8
Total	54	100

Figure 13: Distribution of Cases according to MRI findings

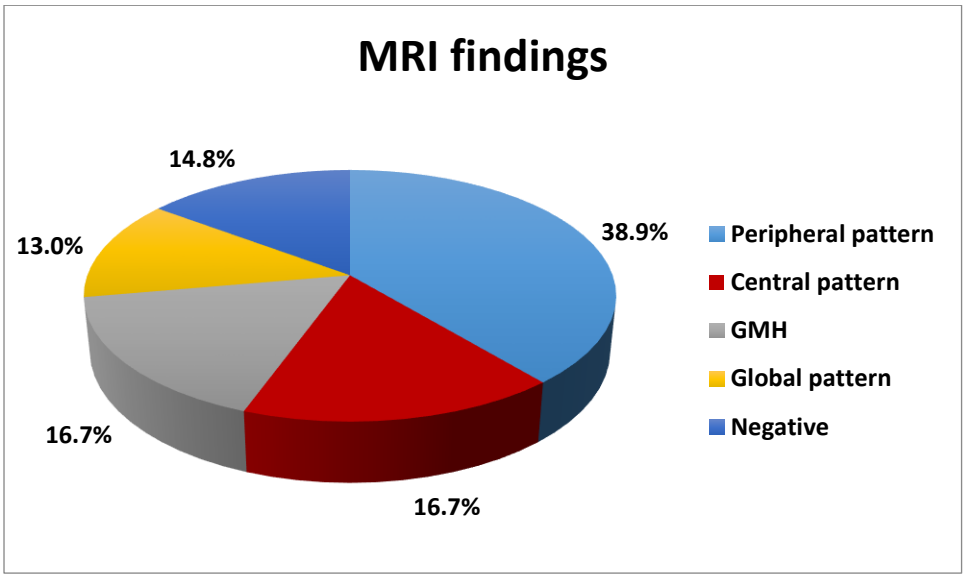


Out of 54 cases, MRI showed signs of HIE in 46 cases (85%) and was normal in 8 cases (15%).

Table 14: Distribution of Cases according to MRI findings

MRI findings	N	Percent
Peripheral pattern	21	38.9
Central pattern	9	16.7
GMH	9	16.7
Global pattern	7	13
Negative	8	14.8
Total	54	100

Figure 14: Distribution of Cases according to MRI findings

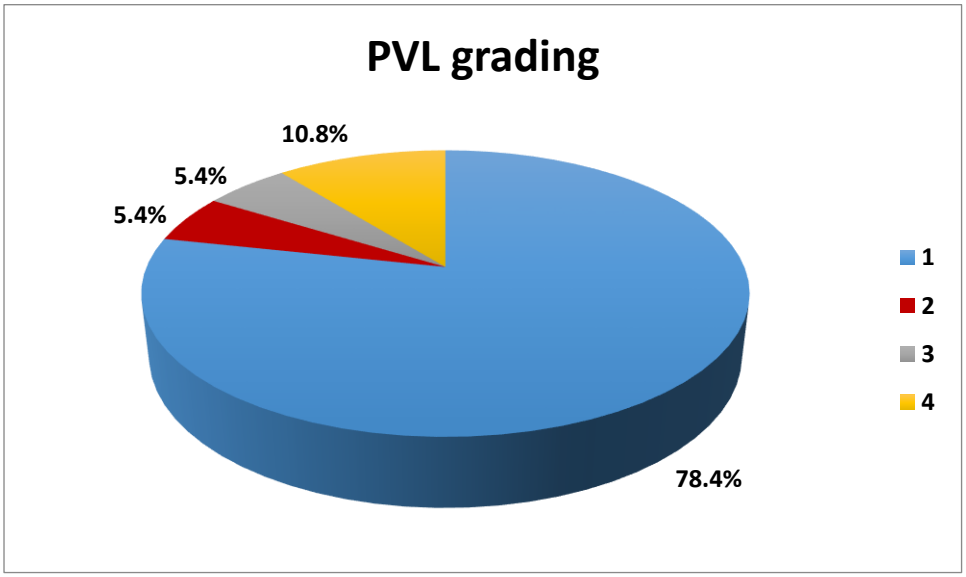


Out of 54 cases, on MRI peripheral pattern of HIE cases comprised 21 cases (38.9%), central pattern and GMH cases comprised 9 cases (16.7 %) each, global hypoxic injury is seen in 7 cases (13 %).

Table 15: Distribution of Cases according to PVL grading

PVL grading	N	Percent
1	29	78.4
2	2	5.4
3	2	5.4
4	4	10.8
Total	37	100.0

Figure 15: Distribution of Cases according to PVL grading

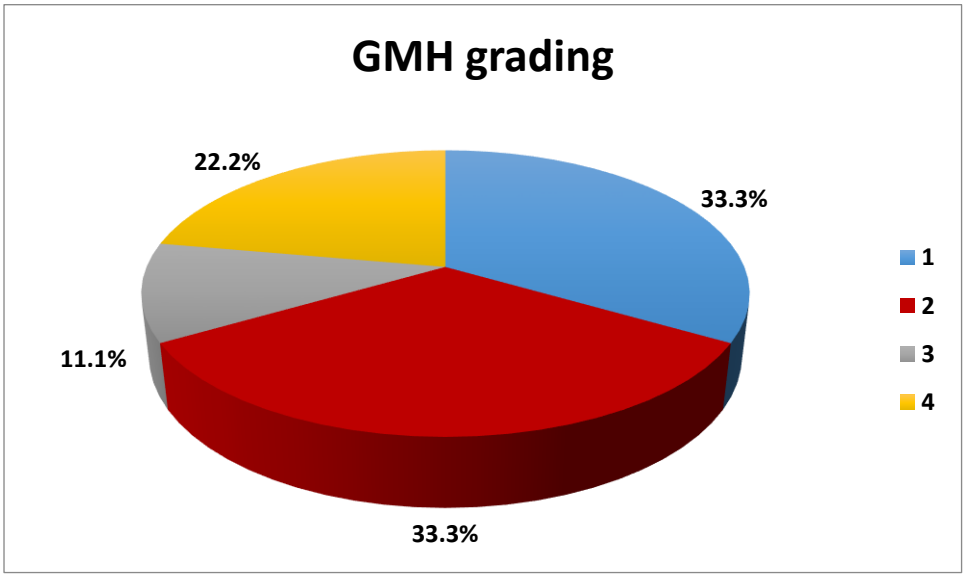


Out of 37 positive cases, PVL grade 1 is seen in 29 cases (78 %), grade 2 in 2 cases (5 %) and grade 3 in 2 cases (5 %) and grade 4 in 4 cases (11%).

Table 16: Distribution of Cases according to GMH grading

GMH grading	N	Percent
1	3	33.3
2	3	33.3
3	1	11.1
4	2	22.2
Total	9	100.0

Figure 16: Distribution of Cases according to GMH grading



Out of 9 positive cases, grade 1 is seen in 3 cases (33.3%), grade 2 in 3 cases (33.3 %), grade 3 in 1 case (11.1%) and grade 4 in 2 cases (22.2%).

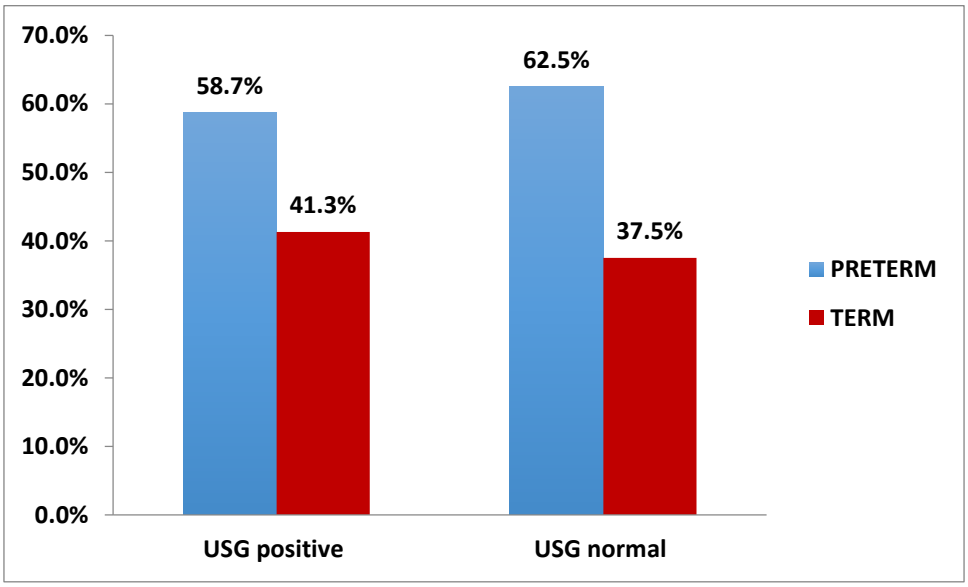
Table 17: Association of Positive cases and GA and HIE stage

	Total		**Preterm**		**Term**		**HIE stage I**		**HIE stage II**		**HIE stage III**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Total cases	54	100%	32	100%	22	100%	6	100%	20	100%	28	100%
Total USG positive cases	46	85.2%	27	84.4%	19	86.4%	5	83.3%	16	80.0%	25	89.3%
Total MRI positive cases	46	85.2%	28	87.5%	18	81.8%	4	66.7%	15	75.0%	27	96.4%
USG Positive MRI Positive	40	74.1%	24	75.0%	16	72.7%	4	66.7%	12	60.0%	24	85.7%
USG Positive MRI Normal	6	11.1%	3	9.4%	3	13.6%	1	16.7%	4	20.0%	1	3.6%
USG Normal MRI Positive	6	11.1%	4	12.5%	2	9.1%	0	0.0%	3	15.0%	3	10.7%
USG Normal MRI Normal	2	3.7%	1	3.1%	1	4.5%	1	16.7%	1	5.0%	0	0.0%

Table 18: Distribution of USG positive cases according to GA

Term /Preterm	USG positive		USG normal		p value
	N	%	N	%	
Preterm	27	58.7%	5	62.5%	0.840
Term	19	41.3%	3	37.5%	
Total	46	100.0%	8	100.0%	

Figure 18: Distribution of USG positive cases according to GA

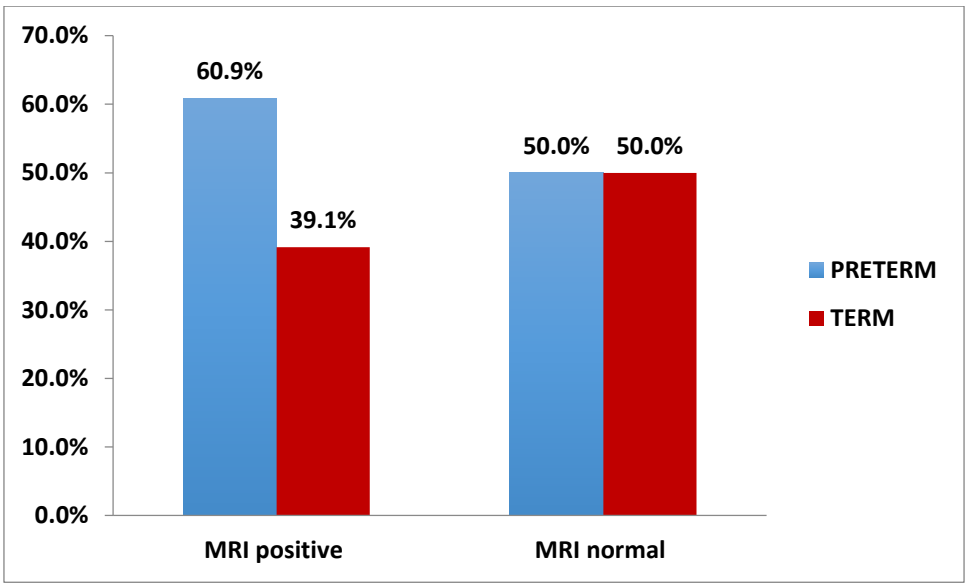


Out of 46 positive cases, total preterm positive are 27 cases (58.7%) and term positive are 19 cases (41.3%).

Table 19: Distribution of MRIpositive cases according to GA

Term /Preterm	MRI positive		MRI normal		p value
	N	%	N	%	
Preterm	28	60.9%	4	50.0%	0.564
Term	18	39.1%	4	50.0%	
Total	46	100.0%	8	100.0%	

Figure 19: Distribution of MRIpositive cases according to GA



Out of 46 positive findings cases, total preterm positive are 28 cases (60.9%) and term positive are 18 cases (39.1%).

Table 20: Distribution of MRIpositive cases according to GA among USG Normal cases

Term /Preterm	MRI positive		MRI normal		p value
	N	%	N	%	
Preterm	4	80.0%	1	20.0%	0.673
Term	2	66.7%	1	33.3%	
Total	6	75.0%	2	25.0%	

Figure 20: Distribution of MRIpositive cases according to GA among USG Normal cases

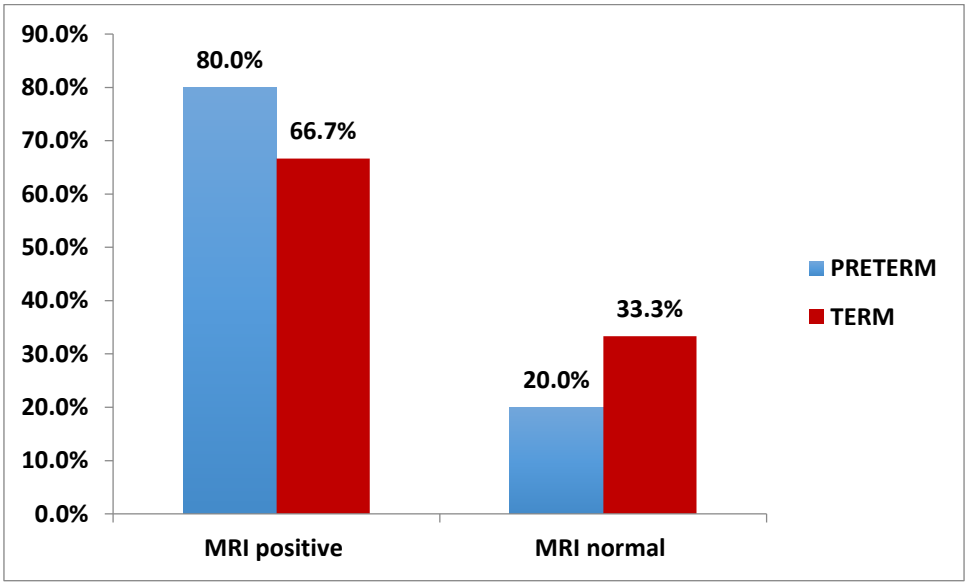


Table 21: Distribution of MRIpositive cases according to GA among USG positive cases

Term /Preterm	MRI positive		MRI normal		p value
	N	%	N	%	
Preterm	24	88.9%	3	11.1%	0.643
Term	16	84.2%	3	15.8%	
Total	40	87.0%	6	13.0%	

Figure 21: Distribution of MRIpositive cases according to GA among USG positive cases

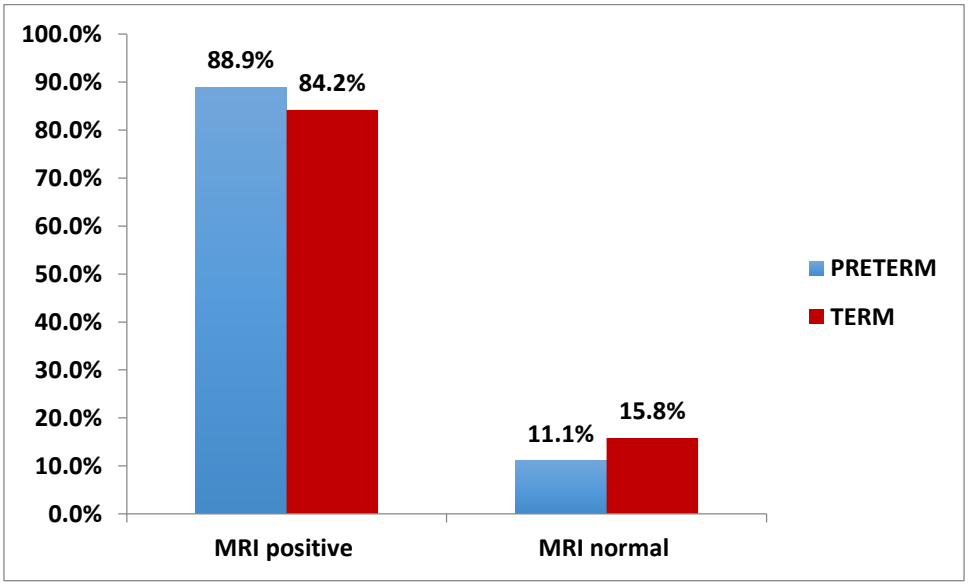
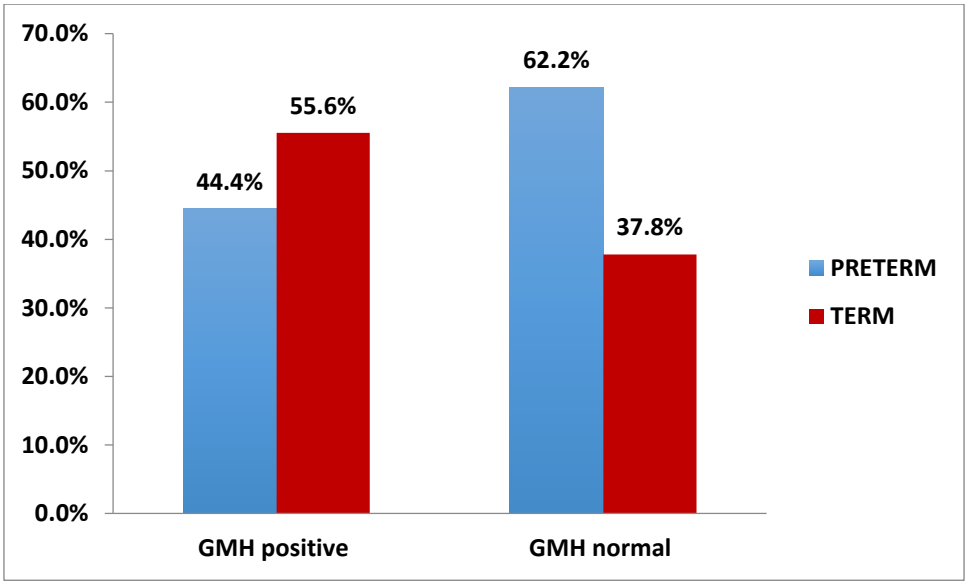


Table 22: Distribution of GMHpositive cases according to GA

Term /Preterm	GMH positive		GMH normal		p value
	N	%	N	%	
Preterm	4	44.4%	28	62.2%	0.322
Term	5	55.6%	17	37.8%	
Total	9	100.0%	45	100.0%	

Figure 22: Distribution of GMHpositive cases according to GA

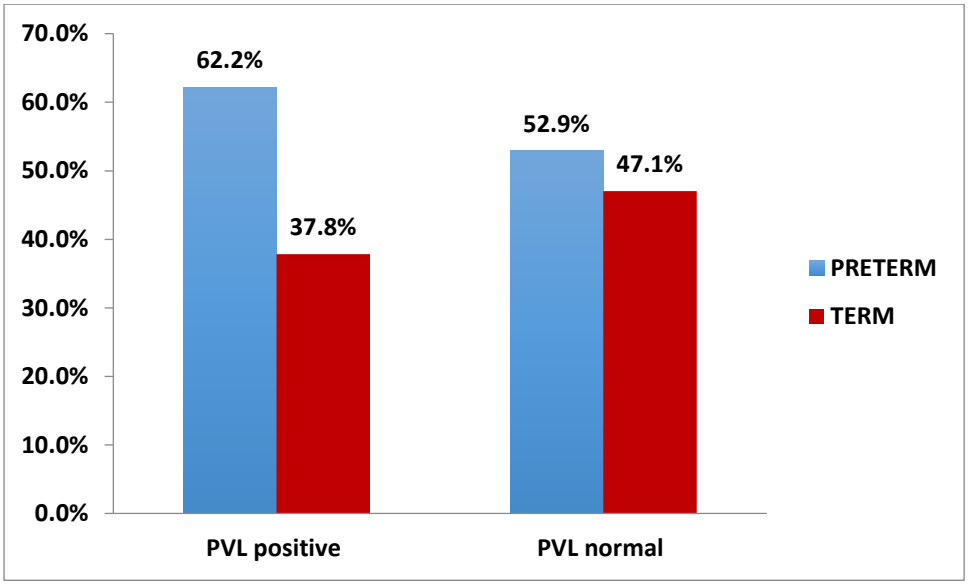


Out of 9 cases showing GMH findings, preterm cases showing positive findings are 4 cases (44.4%) and term cases with positive findings are 5 cases(55.6%).

Table 23: Distribution of PVLpositive cases according to GA

Term /Preterm	PVL positive		PVL normal		p value
	N	%	N	%	
Preterm	23	62.2%	9	52.9%	0.522
Term	14	37.8%	8	47.1%	
Total	37	100.0%	17	100.0%	

Figure 23: Distribution of PVLpositive cases according to GA



Out of 37 cases showing PVL findings, preterm cases showing positive findings are 23 cases (62.2%) and term cases with positive findings are 14 cases (37.8%).

Table 24: Distribution of PVLpositive cases according to GAamong GMH positive cases

Term /Preterm	PVL positive		PVL normal		p value
	N	%	N	%	
Preterm	0	0.0%	4	44.4%	-
Term	0	0.0%	5	55.6%	
Total	0	0.0%	9	100.0%	

Figure 24: Distribution of PVLpositive cases according to GAamong GMH positive cases

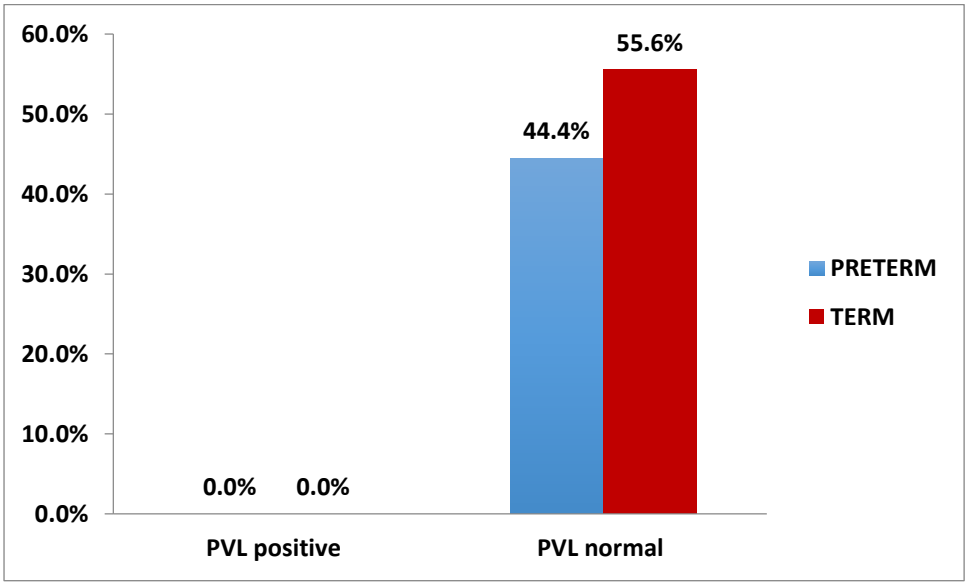


Table 25: Distribution of MRIpositive cases according to HIE Stage

HIE Stage	MRI positive		MRI normal		p value
	N	%	N	%	
I	4	8.7%	2	25.0%	0.048(sig)
II	15	32.6%	5	62.5%	
III	27	58.7%	1	12.5%	
Total	46	100.0%	8	100.0%	

Figure 25: Distribution of MRIpositive cases according to HIE Stage

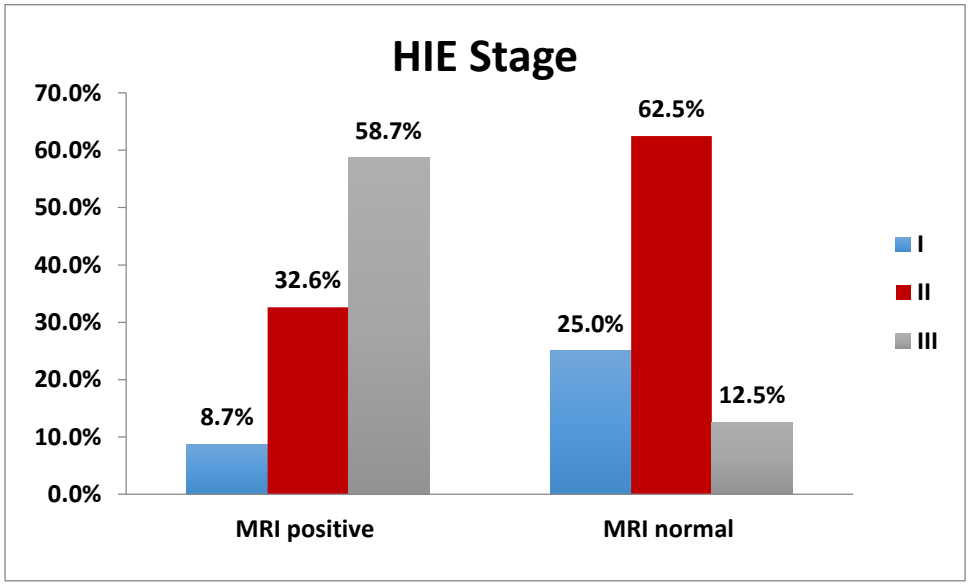


Table 26: Distribution of USG positive cases according to HIE Stage

HIE Stage	USG positive		USG normal		p value
	N	%	N	%	
I	5	10.9%	1	12.5%	0.665
II	16	34.8%	4	50.0%	
III	25	54.3%	3	37.5%	
Total	46	100.0%	8	100.0%	

Figure 26: Distribution of USG positive cases according to HIE Stage

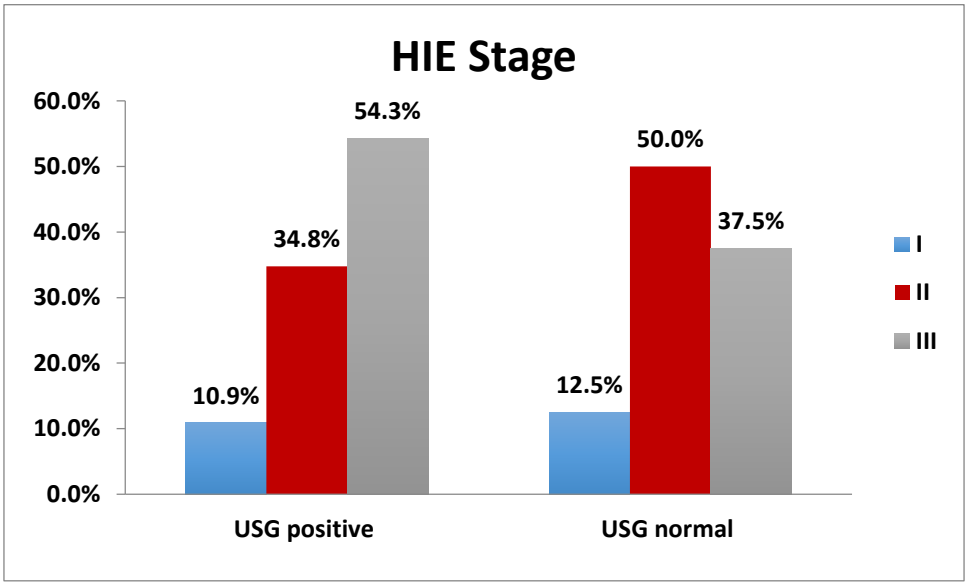


Table 27: Association of USG and MRIpositive cases

USG	MRI positive		MRI normal		p value
	N	%	N	%	
Positive	40	87.0%	6	75.0%	0.380
Normal	6	13.0%	2	25.0%	
Total	46	100.0%	8	100.0%	

Figure 27: Association of USG and MRIpositive cases

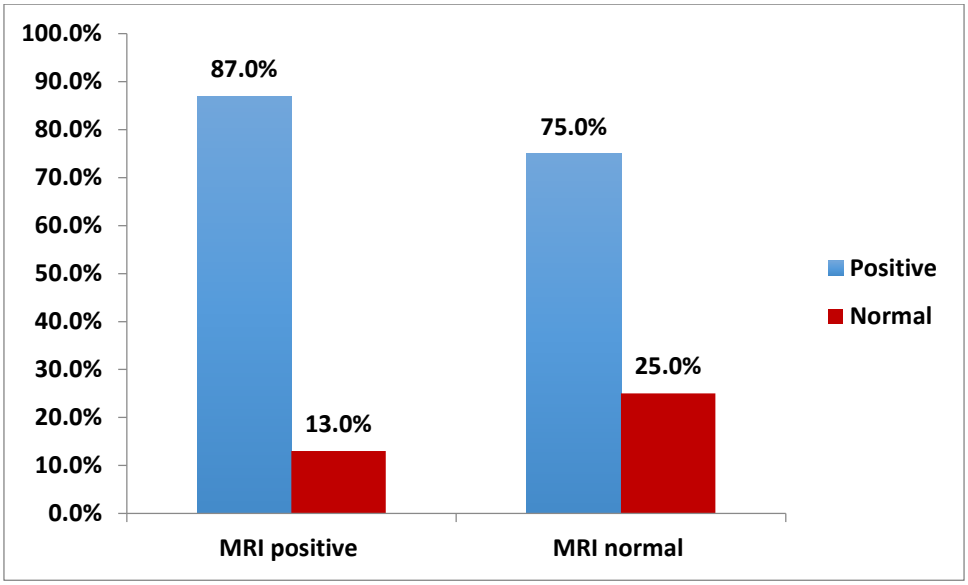
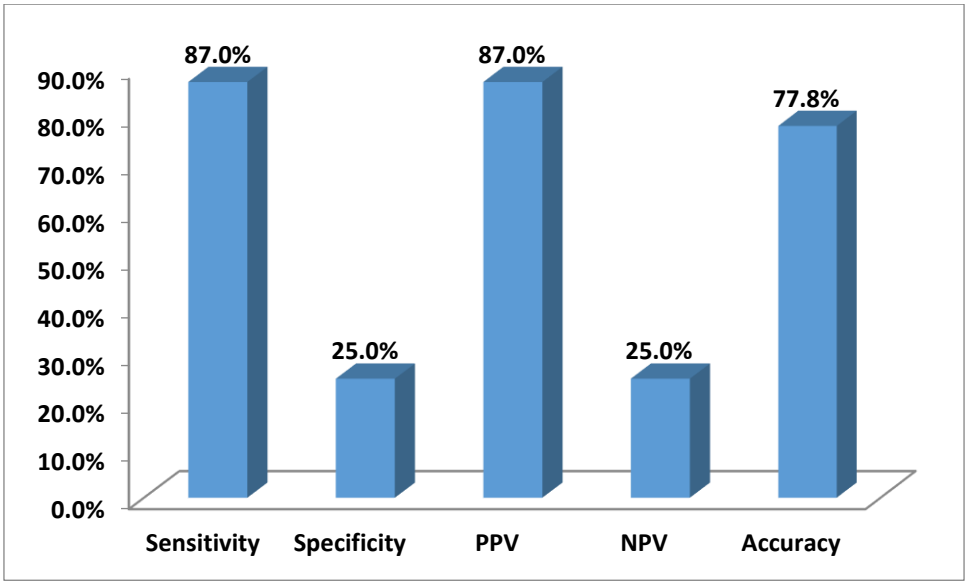


Table 28: Sensitivity analysis of USG compared to MRI

Sensitivity	87.0%
Specificity	25.0%
PPV	87.0%
NPV	25.0%
Accuracy	77.8%

Figure 28: Sensitivity analysis of USG compared to MRI



In the present study the sensitivity of transcranial neurosonogram is 87 % with positive predictive value of 87 % and accuracy of 77.8 % .

Table 29: Sensitivity analysis of USG compared to MRI among Term/Preterm neonates

	PRETERM	TERM
Sensitivity	85.7%	88.9%
Specificity	25.0%	25.0%
PPV	88.9%	84.2%
NPV	20.0%	33.3%
Accuracy	78.1%	77.3%

Figure 29: Sensitivity analysis of USG compared to MRI among Term/Preterm neonates

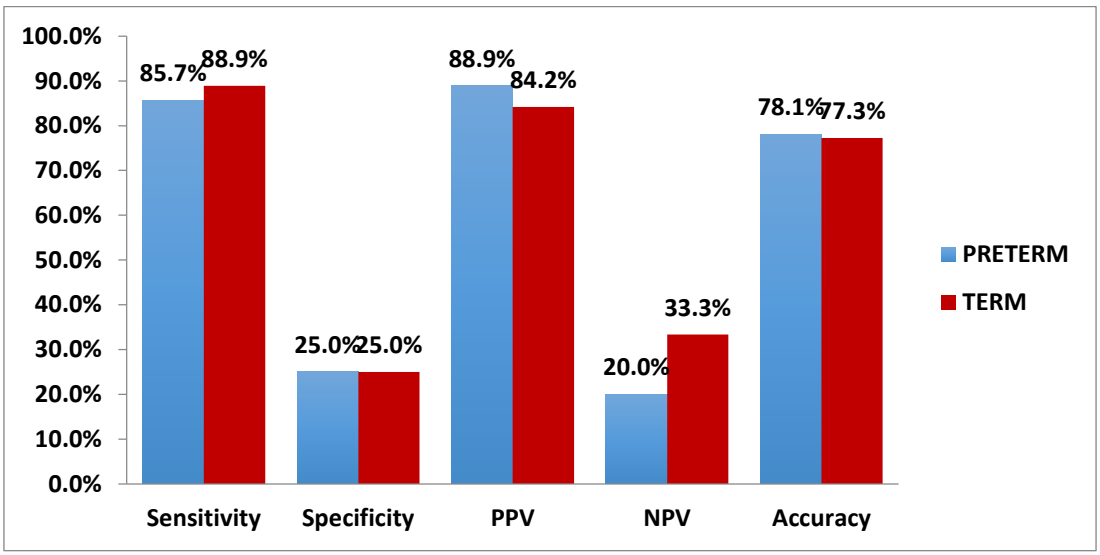


Table 30: Association of USG and MRIpositive cases among RD positive

USG	MRI positive		MRI normal		p value
	N	%	N	%	
Positive	15	93.8%	1	100.0%	0.797
Normal	1	6.3%	0	0.0%	
Total	16	100.0%	1	100.0%	

Figure 30: Association of USG and MRIpositive casesamong RD positive

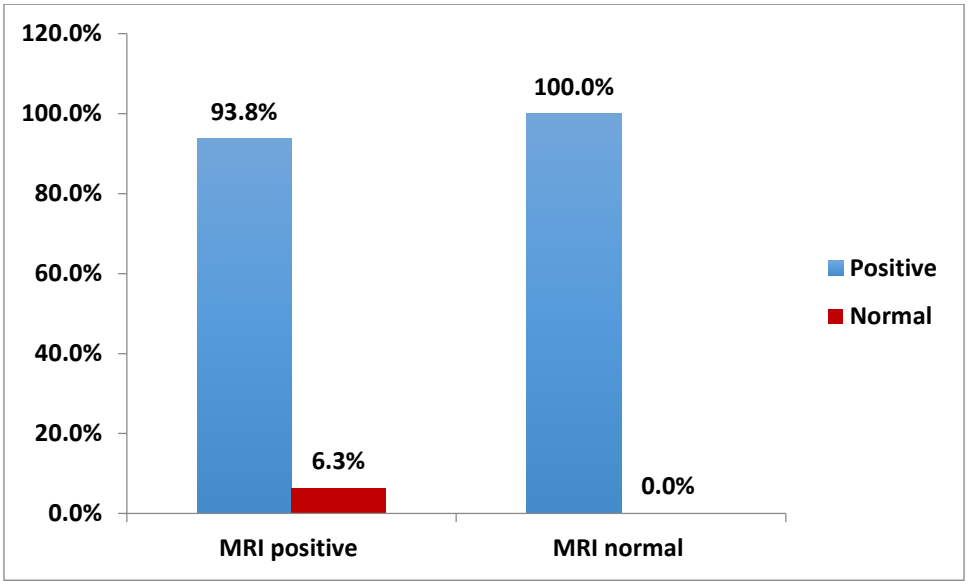
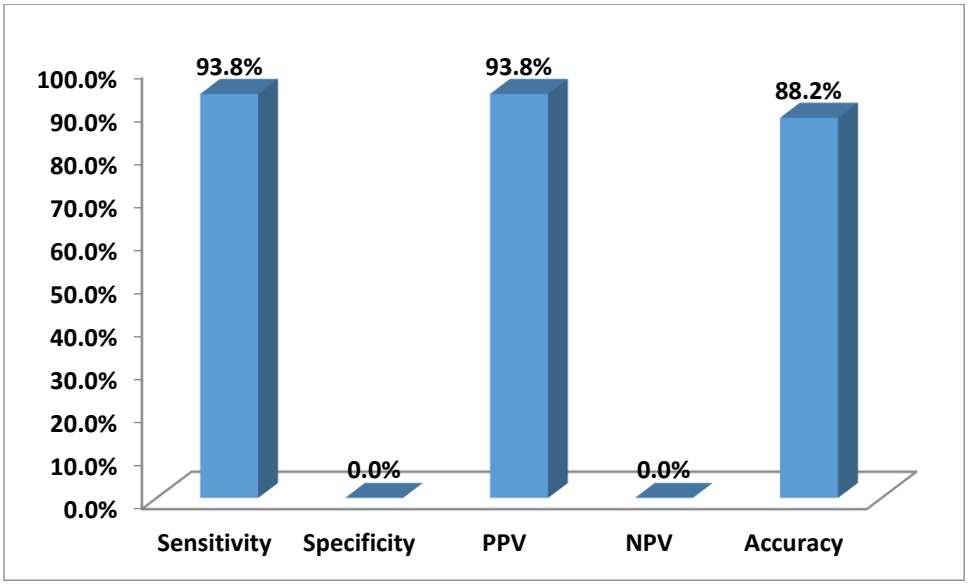


Table 31: Sensitivity analysis of USG compared to MRIamong RD positive

Sensitivity	93.8%
Specificity	0.0%
PPV	93.8%
NPV	0.0%
Accuracy	88.2%

Figure 31: Sensitivity analysis of USG compared to MRIamong RD positive



DISCUSSION

Hypoxic ischemic encephalopathy is an abnormal neurodevelopmental outcome in which the predominant pathogenic mechanism is impaired cerebral blood flow that may result in neonatal death or be manifested later as cerebral palsy, permanent neurodevelopmental handicaps including seizures, severe mental retardation and learning disabilities.

HIE is characterized by clinical and laboratory evidence of acute or subacute cerebral injury (i.e., cerebral hypoxia & metabolic acidosis), most often the exact timing & underlying cause remains unknown. WHO defines perinatal asphyxia as failure to initiate and sustain breathing at birth.

In the present study of 54 cases, all cases had undergone MR imaging as well as transcranial ultrasound study of the brain.

In the present study, majority of cases were preterm neonates were 59 % and term neonates accounting for 41 %.

Ramachandran et al.[95]in their study found that from total 50 patients, 82% were term babies and 18% were preterm babies. Our study disagreed with the above study, as we detected higher incidence of preterm babies compared to term babies. The incidence of HIE is higher among the preterm babies compared to its incidence among full term babies due to incomplete brain maturation in preterm babies.

In the present study out of 54 neonates with hypoxic ischemic encephalopathy 32 (59.3 %) were preterm neonates and 22 (40.7 %) were term neonates. Clinically these neonates were classified into mild (HIE I) moderate (HIE II) , severe (HIE III) grades according to sarnat and sarnat staging. In the present study majority cases belong to HIE stage III 28 (51.9 %). 20 cases were stage II (37 %) , 6 cases were (11.1 %) stage I.

In relation to clinical sarnat & sarnat staging, on ultrasound out of 6 HIE stage I cases 5 cases showed positive imaging findings (10.9 %) .Out of 20 HIE stage II cases, 16 cases had positive imaging findings (34.8 %), Of 28 HIE stage III cases 25 cases showed positive imaging findings (54.3 %).

Out of 54 cases, 46 cases had positive imaging findings for hypoxic ischemic encephalopathy (85 %), of which preterm neonates were 27 which accounts for 58.7

% of preterm neonates group and term neonates were 19 which accounts for 41.3 % of term neonates on ultrasound.

Commonest imaging finding in preterm neonates in my study was white matter injury (68.5 %) followed by Germinal matrix hemorrhage.

Most common imaging in term neonates with HIE in the present study was periventricular white matter injury presenting as watershed infarcts with peripheral pattern 21 cases (39 %), followed by central pattern of white mater injury 9 cases (6 %) and global pattern of injury in 7 cases (13%).

Out of periventricular white matter injury case, 23 cases showed DWI restriction on MRI as positive finding.

Out of 54 cases, Anterior cerebral artery showing R.I value of 0.5 – 0.8 comprised 72 %, and <0.50 and >0.81 comprised 28 %. Cut –off values of <0.5 and >0.8 were associated with bad prognosis.

In the present study out of 24 preterm neonates, 9 cases had GMH (41 %) GMH grade I was found in 3 cases (33 %) . GMH II was found in 3 cases(33 %) GMH III in 1 case (11 %) and GMH IV was found in 2 cases (22 %) .

On transcranial neurosonography, 9 cases of GMH was identified. MRI identified all cases of GMH. GRE sequence & T1W images are most sensitive for identifying GMH in the present study, which are seen as T1 hyperintensity and foci of blooming on GRE. In our study, Transcranial neurosonography detected all cases which were detected on MRI.

In the present study, out of 9 cases of GMH 4 cases are seen in preterm neonates (44.4 %) and 5 cases are seen in term neonates (55.6%), which is lower than the findings by **susan C . carson et al.**[31] who found it in 57 % cases and is almost equal to **jerome burrstein et al**[75] who found it in 44 %. **Kalyani et al.**[28] found it in 21.1 % cases which is lower than the present study.

Eman sh gendi et al.[16] compared MRI and transcranial ultrasound in identification of cerebral injuries In neonatal encephalopathy, out of 38 cases they found GMH 5% preterm neonates. Their TCUS sensitivity in detecting GMH was 100 % which is similar to our present study.

Jarunee . Frances et al.[21]in their study on 12 neonates Ultrasound had 100 % sensitive 93 % specific in detecting GMH III and ultrasound was found not sensitive (0) in detecting small GMH which is similar to present study.

Grade IV GMH also known as periventricular hemorrhagic infarction (PVHI) was found in 2 cases in present study (22.2 %). On MRI in both these cases hemorrhage was noted in intraparenchymal location.

In the present study, 37 neonate cases presented with white matter injury which accounts for 68.5 % of total cases and constitutes 23 cases of preterm neonates (62.2 %) and 14 cases of term neonates (37.8 %).

In the present study, on ultrasound white matter injury was detected as increased periventricular echogenicity of grade I in 29 cases (78.4%). grade II PVL in 2 cases (5.4 %), grade III in 2 cases (5.4%) and 4 cases with grade IV PVL (10.8 %).

Out of 37 cases with white matter injury, 5 cases were normal on trans cranial neurosonography. Fronto-parietal white matter showing peripheral pattern of injury was affected in majority of cases (21 cases), central pattern on injury was seen in 9 cases and 2 cases presented with global hypoxic injury.

E , Inder nigel et al.[27]in this study they compared cranial sonography MRI in premature neonates with white matter injury they included 96 preterm neonates 38 (40 %) had white matter injury , 18 % had cystic injury .In their study, fronto-parietal whitematter involved in majority of cases which is similar to the present study.

Steven P miller , Camilla et al.[46] in their study of 32 preterm neonates white matter injury seen in 51 % cases as scattered T1 hyperintense foci which was not detected on ultrasonography which is similar to present study as non cavitatory whitematter injury was not identified on transcranial neurosonography . Their sensitivity in detecting cystic white matter injury on ultrasound is 100 % with positive predictive value of 100 % which is similar to present study.

Jarunee ,frances et al.[21]in their study, they examined 12 preterm neonates with intracranial hemorrhage with a history of birth asphyxia on cranial ultrasound and compared with MRI and SWI. Their study found cases of cerebellar hemorrhage. Ultrasound detection rate for cerebellar hemorrhage in their study was 0 which is similar to present study.

Eman A h et al.[16]in their study evaluated 38 neonates with MRI and transcranial ultrasound with encephalopathy in their study 5 preterm neonates had basal ganglion injury (13 %) which is lower than present study.

Miller SP et al.[18]in their study pattern of injury in term neonatal encephalopathy it is a prospective study of 175 term neonates watershed infarct was the predominant pattern (78 cases) of on their study accounting for 45 % which higher than present study (39%).

Dr sanjay , kahlkadhar et al.[77] in their study evaluated 100 cases of perinatal hypoxia of which 64 cases were term neonates 46 of them showed abnormal imaging findings which is higher than present study 46/ 64 (40 %) . 36 neonates had cortical, periventricular white matter injury (60 %) which is higher than the present study.

Childs AM , cornette L et al.[17]in their study out of 25 term neonates 15 cases were abnormal (60 %) Out of 15 cases 10 cases were mild to moderate injury cases (66 %) 4 had subcortical infarcts and 6 had watershed infarcts which is higher than the present study.

Mohammad s babiker et al.[6] in their prospective study evaluated 150 term neonates with hypoxic ischemic encephalopathy, cranial ultrasound was positive in 75 cases (50 %) and increased periventricular echogenicity was found in 32 % cases of which watershed infarct was found 20 % cases which is lower than present study.

Monica epelman et al.[4] in their study evaluated 76 neonates with hypoxic ischemic encephalopathy with MRI and head USG in their study diagnostic accuracy of ultrasound for detecting watershed infarcts was 95 % which is higher than present study (68%).

Miller SP et al.[18] in their study of 173 term neonates basal ganglion / thalamic pattern of injury noted in 44 cases (25 %) which is higher than present study.

Rutherford et al. [5]in their study, cranial USG and MRI in HIE they studied over 40 term neonates 38 term neonates were abnormal. Basal ganglia injury was not identified on ultrasound which is similar to present study.

In the present study 5 term neonates (55.5 %) had germinal matrix hemorrhage. Neurosonography finding showed increased periventricular echogenicity. On MRI, small germinal matrix hemorrhage was noted in which was identified on neurosonography. **Suneja et al.** [52]in their prospective study on term neonates with

hypoxic ischemic encephalopathy identified 1.9 % term neonates having germinal matrix hemorrhage which is lower than the present study.

Basavaraj patel et al. [60]in their study- clinicoradiological correlation in birth asphyxia evaluated 37 neonates, out of which mild to moderate cases were 18 and severe cases (Stage III) were 19. In their study, USG was abnormal in 1 case of HIE 2 (4.7 %).MRI was abnormal in 11 cases (61.3 %) which is lower than the present study (75 %) . In stage III cases 14 cases (87 %) were having abnormal findings on MRI which is lower than present study (96.4 %).

Monica epelman et al. [11] in their study of 76 neonates with HIE, Ultrasound sensitivity was 95.7 % which is higher than present study (87 %)

Eman ash genedi et al. [16]in their study of 38 neonates, 16 were term neonates transcranial ultrasound sensitivity was 80.2 % which is lower than present study (87 %).

Mohmad S babiker et al. [6]in their study of 150 neonates with HIE, 50 % cases were normal on neurosonography, 29 % were normal on MRI. In their study, transcranial ultrasonography has sensitivity of 70 %, specificity of 100 % and positive predictive value was 100% with an accuracy of 76.8% which was slightly lower than the present study.

Wei shan and jia hua et al[10] in their study - compared trancranial ultrasound and cranial MRI in evaluation of brain injuries from neonatal asphyxia in 30 asphyxia full term neonates and observed brain edema in severe injury cases with ultrasound as major finding which was almost similar to our present study. ACA and MCA Doppler showed RI changes with less than 0.5 or more 0.9 in severe asphyxia cases.

DWI was more sensitive modality in picking up early post asphyxia cerebral edema than conventional MRI which was almost similar to our present study.

A study conducted by **Kamal and Hassan et.al**[94] on comparison of transcranial ultrasound and MRI in hypoxic ischemic injury neonates in 36 neonates showed MRI imaging is most sensitive modality in detecting different MR patterns in newborn with birth asphyxia which was similar to our present study.

Ramachandran S et al[95] conducted a study to evaluate the role of MRI in infants with suspected hypoxic ischemic encephalopathy and prognosticating neurological outcome in 50 neonates at end of one year and concluded that MRI is a useful modality to assess early changes in HIE and it can prognosticate clinical outcome which was similar to our present study.

Maalouf and Duggan et al[15] conducted a comparative study of cranial ultrasound and MRI in preterm neonates with hypoxia and evaluated 62 neonates, Ultrasound has demonstrated the same MRI findings in GMH. Other MRI findings of small petechial hemorrhages, diffuse extensive signal injury are less predicted on Ultrasound and concluded that normal white matter echogenicity on ultrasound is not a good predictor of normal white matter signal on MRI which was similar to our present study findings.

Pinkesh, Jagruti et al[35] conducted a study of Transcranial Ultrasound for Detection of Hypoxic Brain Injury in 50 Neonates with history of birth asphyxia and evaluated by bedside transcranial ultrasound. Out of 50 patients, 41 patients were positively identified by transcranial ultrasound which further confirmed clinically and radiologically and concluded that Bedside transcranial ultrasound appears to be very sensitive and promising in early detection and management of the hypoxic brain injury and proves to be valuable screening tool for hypoxic brain injury which was similar to our present study.

In the present study Transcranial neurosonography was normal and MRI was normal (true negative) in 2 cases among preterm and 1 cases of were term neonates.

Transcranial neurosonography was normal and MRI was abnormal (false negative) in 6 cases. Out of which 2 cases were term neonates all were having watershed infarct and 4 cases were preterm neonates showing 5 cases of periventricular noncavitatory white matter injury and 1 case of small focus of GMH.

Neurosonography was abnormal and MRI was also abnormal (true positive) in 40 cases. Out of which 16 were term neonates and 24 cases were preterm neonates.

Transcranial neurosonography was abnormal and MRI was normal (false positive) in 6 cases . Among which 3 cases were term and 3 cases were preterm neonates, their findings was increased periventricular white matter echogenicity.

In the present study, out of 22 term neonates 19 cases were having hypoxic ischemic encephalopathy findings. True positive cases were 16 in which periventricular white matter injury was identified on Neurosonography and imaging was correlated on MRI. False negative cases were 6 out of which 5 cases were having white matter injury and 1 case was showing GMH on MRI. False positive cases were 6 all of them are having increased echogenicity in periventricular regions on neurosonography. In the present study, out of 32 preterm neonates, 27 cases are having positive hypoxic ischemic encephalopathy findings. Periventricular leucomalcia was identified in all cases on USG which was correlated with MRI. Out of 9 GMH cases ,8 cases were identified on USG. Small grade IV GMH was missed in 1 case.

In the present study sensitivity of transcranial neurosonography was 87 % , specificity was 25 % , positive predictive value was 87 % ,negative predictive value was 25 % and accuracy was 77.8 % .

In term neonates sensitivity of transcranial neurosonography was 88.9 % ,specificity was 25 % ,positive predictive value was 84.2 %, negative predictive value was 33.3 % and accuracy was 77.3 % . In preterm neonates sensitivity was 85.7 % positive predictive value was 88.9 % ,and accuracy was 78.1 % .

SUMMARY

1. Perinatal asphyxia or hypoxic ischemic encephalopathy in neonate is defined as failure to initiate or sustain respiration at birth due to impaired cerebral autoregulation.

2. During the period of 19 months, 54 neonates with birth asphyxia were evaluated with transcranial neurosonography and MRI

3. Out of 54 neonates, preterm neonates were 32 (59.3 %) Term neonates were 22 (40.7 %)

4. 46 neonates had hypoxic ischemic encephalopathy imaging findings. Of which, 19 were term neonates and 27 were preterm neonates.

5. In preterm neonates, 61 % cases were having positive image findings for HIE and in term neonates 39 % cases were showing findings on MRI.

6. White matter injury was the predominant HIE injury (62.2 %) in 23 cases of preterm neonates and in 14 cases of term neonates (37.8%) followed by GMH (16.7 %).

7. Out of 9 positive cases of GMH, preterm cases showing positive findings are 4 cases (44.4%) and term cases with positive findings are 5 cases(55.6%).Grade 1 is seen in 3 cases (33.3%), grade 2 in 3 cases (33.3 %), grade 3 in 1 case (11.1%) and grade 4 in 2 cases (22.2%). Neurosonography identified all the cases of GMH which were detected on MRI.

8. Out of 37 cases showing PVL findings, preterm cases showing positive findings are 23 cases (62.2%) and term cases with positive findings are 14 cases (37.8%). MRI identified white matter injury which were missed in transcranial neurosonography.

9. About 62.2 % preterm neonates had white matter injury (23) in which 4 cases had noncavitatory white matter injury detected onMRI as T1WI hyperintense foci out of which 1 case of Grade I was missed on NSG. 2 cases presented with Grade II PVL on NSG.

2 cases as grade III PVL and 4 as grade IV PVL, these findings were almost correlated with NSG.

10. Basal ganglia infarcts were seen in 9 cases of birth asphyxia neonates (16.7 %). DWI detected the infarct as restriction and low on ADC. NSG was abnormal in 8 cases.
11. In term neonates, out of 22 cases 19 had positive imaging findings for HIE of mild to moderate injury with watershed infarct being the major finding. Most of the cases with mild moderate HIE had increased white matter echogenicity and periventricular flare on NSG.
12. Out of 54 cases, HIE stage III cases comprised majority of the present study group – 28 cases (52%) followed by stage II (37%), stage I (11%).
13. Preterm neonates corresponding to HIE I and HIE II, irrespective of clinical staging all had positive HIE imaging findings on MRI.

CONCLUSION

1. Transcranial neurosonography is sensitive in detecting and grading GMH and periventricular white matter injury.

2. Transcranial neurosonography is less sensitive in detecting basal ganglia injury and posterior fossa hemorrhages. However, it is sensitive in detecting white matter injury.

3. Inspite of having many advantages of MRI, Transcranial neurosonography is still considered as an important primary investigation of choice from prognostic point of view and in neonates who are on ventilator support and unfit for transport to MRI.

4. Transcranial neurosonography when combined with ACA Doppler can be initial modality of investigation and helps to determine the prognosis of Hypoxic ischemic changes which can be used as a prognostic indicator of hypoxic cerebral injury and neurological outcome.

5. MRI can be deffered in term neonates with stage - I HIE, if transcranial neurosonography is normal.

6. In term neonates, mild to moderate HIE results in watershed infarct and severe injury results in basal ganglion and thalamic injury. Neurosonography shows increased echogenicity in the affected areas.

7. MRI is superior in detecting mild to moderate and severe HIE injury in both Preterm and term neonates.

8. DWI is more sensitive in detecting early and severe HIE injuries than any other MRI sequences.

9. In preterm neonates, irrespective of their clinical staging they should be evaluated with neurosonography and possibly MRI if neonate is ambulatory.

10. In term neonate with HIE stage - III, neonate should be evaluated with Transcranial neurosonography and MRI.

LIMITATIONS

1) Low sample size.

2) Less number of HIE stage I cases.

3) Lack of availability of dedicated MRI head coil for neonates and proper sedation protocol.

4) Lack of follow up with MRI brain after successful resuscitation of neonates.

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22-11-2019

B.L.D.E. (DEEMED TO BE UNIVERSITY)

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The ethical committee of this college met on 13-11-2019 at 3-15 pm to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: : Comparative study of transcranial neurosonography and cross-sectional imaging in neonates with hypoxic ischemic encephalopathy.

Name of PG student: : Dr. P. Naga Bhavani, Department of Radiodiagnosis

Name of Guide/Co-investigator: Dr.Shivanand V Patil, Associate Professor Department of Radiodiagnosis

DR RAGHVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

B.L.D.E.(Deemed to be University)
SHRI B.M.PATIL MEDICAL COLLEGE,VIJAYAPUR-586103
INSTITUTIONAL ETHICAL COMMITTEE

Date : 13-11-2019

1. Name of UG/PG Students/Researcher: Dr. P. Naga Bhavani
2. Department : Radiodiagnosis
3. Title : Comparative Study Of Transcranial Neurosonography And Cross-Sectional Imaging In Neonates With Hypoxic Ischemic Encephalopathy.
4. Guide/Co-Guide/Principle Researcher: Dr.Shivanand V Patil, Associate Professor
5. Date of Admission (PG Only) :

Observation :

- There are no ethical issues.

I.E.C. Remarks : Ethical Clearance accorded/be Chairman after corrected revised version is submitted by stipulated time.
1. Any alternation in Synopsis protocol should be intimated to E.C. in writing for review & approval.
2. Any adverse effects to subject of the study should be intimated in writing to E.C.
3. If study is stopped or an included patient is out of study inform E.C. the same with reason.

Signature of the Committee Members :

1. Dr Raghavendra Kulkarni, Chairman
2. Dr Tejaswini Vallabha
3. Dr Akram Naikawadi
4. Dr P.B.Jaju
5. Dr Chandrashekhar Bhuyyar
6. Dr Pranesh Jahagirdar
7. Dr Manjunatha Aithala
8. Dr Satish Patil
9. Dr Mohammed Shannawaz

BLDEU'S SHRI B.M.PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA

COMPARATIVE STUDY OF TRANSCRANIAL NEUROSONOGRAPHY AND CROSS-SECTIONAL IMAGING IN NEONATES WITH HYPOXIC ISCHEMIC ENCEPHALOPATHY

CASE PROFORMA :

NAME OF THE BABY :

AGE :

SEX :

BIRTH WEIGHT :

TYPE OF DELIVERY AND BIRTH HISTORY :

CHIEF COMPLAINTS :

APGAR SCORE :

HIE CLINICAL STAGE :

USG FINDINGS :

CT FINDINGS :

MRI FINDINGS :

PATIENT CONSENT FORM

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further compensations.

Signature of Guardian Signature of patient

Signature of the guide Signature of postgraduate

STUDY SUBJECT CONSENT STATEMENT:

I/my ward confirm that Dr. P. Nagabhavani has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I/my ward have been explained all the above in detail in my own language and I understand the same. Therefore, I agree to give my consent to participate as a subject in this project.

(Participant) Date

(Witness to above signature) Date

S NO	DATE OF STUDY	IP NO	PATIENT NAME	AGE(DAYS	GEST.AGE	GENDER	BIRTH WEIGH	TYPE OF DELIVARY	TERM /PRETERM	BIRTH HISTORY	CHIEF COMPLAINTS	APGAR SCORE	HIE CLINICAL STAGE	USG FINDINGS	NORMAL
1	13.11.2019	4856	B/O AFATH	2	31	M	1.1	LSCS	PRETERM	RDS	MAS	4	III		PV FLARE
2	14.12.2019	4912	B/O NAGAMMA	3	37	M	2.2	NVD	TERM	MAS, RDS	BIRTH ASPHYXIA	5	II		N
3	15/02/2020	5667	BO SUJATHA PUJARI	3	38	M	2.1	LSCS	TERM	RDS	MAS	3	III		RT GMH
4	12.02.2020	5275	BO KALLAMMA	1	38	M	2.8	NVD	TERM	RDS	RDS	4	II		PV FLARE
5	08.02.2020	4608	BO LAXMI	1	33	M	2.6	LSCS	PRETERM	MAS, RDS	MAS	4	II		PV FLARE
6	08.02.2020	4495	BO ANITA	1	34	M	1.8	NVD	PRETERM	VLBW RDS	RDS	3	III		PV FLARE
7	26.12.2019	4936	BO SIDDAMMA	27	35	F	2.7	NVD	TERM	RDS	DELAYED CRY	2	III		PV FLARE
8	26.12.2019	4621	BO NAZNIN	20	38	M	2.8	NVD	TERM	RDS	CONVULSIONS	4	III		GMH,IVH,HYDROCEPH,IPH
9	07.11.2019	4172	BO SHEKAMMA	15	38	M	1.8	LSCS	TERM	IUGR	BIRTH ASPHYXIA	3	III		GMH,IVH,HYDROCEPH,IPH
10	07.12.2019	4728	BO ALTHANUSHEEN	1	28	M	1.1	NVD	PRETERM	RDS	BIRTH ASPHYXIA	4	II		PV FLARE
11	26.05.2020	4965	BO PRIYANKA	2	32	F	1.9	LSCS	PRETERM	ABRUPTIO	RDS	5	II		PV FLARE
12	26.05.2020	4838	BO MAIXABAI	1	33	F	2	LSCS	PRETERM	RDS	MAS	2	III		PV FLARE
13	05.04.2020	5162	BO RESHMA	3	34	F	2.2	LSCS	PRETERM	RDS	MAS	3	III		PVLM
14	27.05.2020	5326	BO KASTURIBAI	1	33	M	2	LSCS	PRETERM	MAS,DELAYED CRY	CONVULSIONS	5	II		PV FLARE
15	26.05.2020	5265	BO DRAKSHYANI	2	32	F	1.8	NVD	PRETERM	DELAYED CRY	MAS	2	III		PV FLARE
16	27.05.2020	5472	BO MAHABUBI	2	29	F	1.5	NVD	PRETERM	VLBW	RDS	3	III		GMH
17	22.05.2020	5486	BO PARVATI	1	32	M	1.7	LSCS	PRETERM	VLBW	RDS	4	II		GMH,IVH
18	19.05.2020	5674	BO SIDDAWWA	3	37	M	2.3	NVD	TERM	DELAYED CRY	RDS	2	III		GMH
19	26.05.2020	5569	BO GANGA	30	30	M	1.6	LSCS	PRETERM	MAS,DELAYED CRY	BIRTH ASPHYXIA	3	III		PV FLARE
21	13.06.2020	5987	BO RESHMA	2	37	M	1.9	NVD	TERM	MAS	MAS	5	II		PV FLARE
22	25.06.2020	6028	BO ANNAPURNA	12	31	M	1.8	NVD	PRETERM	RDS	RDS	4	II		N
23	29.06.2020	6094	BO BORAMMA	8	30	M	1.7	LSCS	PRETERM	MAS	CONVULSIONS	2	III		PV FLARE
20	01.07.2020	6127	BO DEVAMMA	5	37	M	2.1	LSCS	TERM	RDS	RDS	7	I		PV FLARE
24	06.07.2020	6199	BO MEENAXI	9	31	F	2	NVD	PRETERM	DELAYED CRY	CONVULSIONS	4	II		PV FLARE
24	08.07.2020	6269	BO NAGARATNA	6	38	F	2	NVD	TERM	MAS	BIRTH ASPHYXIA	5	II		PV FLARE
26	13.07.2020	6342	BO SHREEDEVI	10	33	M	1.9	NVD	PRETERM	DELAYED CRY	RDS	3	III		GMH,IVH,HYDROCEPH
27	21.07.2020	6428	BO VIJAYALAXMI	8	34	F	1.8	NVD	PRETERM	MAS	MAS	6	I		PV FLARE
28	24.07.2020	6302	BO JAYASHREE	7	37	F	2	LSCS	TERM	RDS	RDS	7	I		PV FLARE
29	28.07.2020	6380	BO ASHWINI	30	33	M	1.9	NVD	PRETERM	RDS	RDS	5	II		PV FLARE
30	31.07.2020	6372	BO BALU	60	37	M	2	LSCS	TERM	MAS	MAS	4	II		N
31	09.08.2020	6378	BO BHARATI	2	29	M	1.7	LSCS	PRETERM	RDS	RDS	4	II		PV FLARE
32	13.08.2020	6349	BO HONNAMBIKA	12	37	M	2.1	NVD	TERM	MAS	RDS	3	III		PV FLARE
33	18.08.2020	6376	BO MANJULA	4	33	F	1.9	NVD	PRETERM	DELAYED CRY	RDS	5	II		GMH,IVH
34	29.08.2020	6389	BO NASEEMA	1	38	F	2	LSCS	TERM	RDS	MAS	4	II		PV FLARE
35	01.09.2020	6399	BO NEELAMMA	12	30	F	1.7	NVD	PRETERM	CONVULSIONS	RDS	2	III		PV FLARE
36	03.09.2020	6490	BO POONAM	8	38	M	2.1	LSCS	TERM	RDS	RDS	3	III		PVLM
37	07.09.2020	6578	BO PRABHAVATI	12	37	M	1.9	LSCS	TERM	MAS	RDS	2	III		GMH,IVH
38	12.09.2020	10863	BO SAVITA BIRADAR	5	35	M	2	NVD	PRETERM	DELAYED CRY	RDS	7	I		N
39	19.09.2020	9942	BO SHAINAZ	5	31	M	1.7	NVD	PRETERM	RDS	MAS	3	III		PV FLARE
40	23.09.2020	3978	BO SHANTABAI	3	36	M	1.8	LSCS	TERM	RDS	DELAYED CRY	3	III		PVLM
41	25.09.2020	10468	BO SIDDAMMA	8	34	M	2.1	LSCS	PRETERM	DELAYED CRY	MAS	2	III		PVLM
42	28.09.2020	10764	BO SUDHA	5	37	M	2.2	NVD	TERM	RDS	MAS	7	I		PVLM
43	08.10.2020	4021	BO AIESHA	8	33	M	2	NVD	PRETERM	RDS	RDS	2	III		N
44	17.10.2020	4068	BO ANNAPURNA	4	30	F	1.9	NVD	PRETERM	MAS	DELAYED CRY	4	III		PVLM
45	22.10.2020	12949	BO BANGARAMMA	45	32	M	1.7	LSCS	PRETERM	MAS	CONVULSIONS	3	III		PV FLARE
46	28.10.2020	10792	BO BHAGYASHREE	4	38	M	2.1	LSCS	TERM	RDS	BIRTH ASPHYXIA	5	II		N
47	01.11.2020	11487	BO NEELOFER	14	37	F	1.9	NVD	TERM	MAS	RDS	2	III		PV FLARE
48	21.11.2020	4057	BO SUPHIYA	3	30	M	1.7	LSCS	PRETERM	DELAYED CRY	MAS	2	III		N
49	04.12.2020	12344	BO VIJAYALAXMI	4	38	F	2	NVD	TERM	RDS	BIRTH ASPHYXIA	6	I		PV FLARE
50	19.12.2020	13452	BO REKHA	1	34	F	1.9	NVD	PRETERM	MAS	DELAYED CRY	5	II		PV FLARE
51	09.01.2021	16214	BO MALLIKA	5	37	M	2.1	LSCS	TERM	RDS	RDS	5	II		PV FLARE
52	23.01.2021	15432	BO MAULASAB	3	33	M	1.8	NVD	PRETERM	MAS	CONVULSIONS	3	III		N
53	06.02.2021	16712	B ROHINI	12	35	F	2	NVD	PRETERM	RDS	BIRTH ASPHYXIA	5	II		PV FLARE
54	10.02.2021	17521	B VINAYAK	23	34	M	2.1	LSCS	PRETERM	MAS	CONVULSIONS	3	III		PVLM

BG THALAMI	GMH GRADING	PVL GRADING	BRAINSTEM	ACA R.I	OTHERS	MRI FINDINGS	MRI N	MR BG HYPER	FLAIR	DW	HYDRO	BRAINSTEM	ISCHEMIA	GRE	OTHERS
		1		0.7			PVWM		HYPERINTENSE						
				0.6			PVWM		HYPERINTENSE						
	1			0.9			GMH								
		1		0.7			N								
		1		0.8			PVWM								
		1		0.7			PVWM								
		1		0.72			PVWM			FTPO CC					
	4			1.3			GMH,IVH,IPH				HYDRO				
	4			0.8			GMH,IVH,IPH				HYDRO				
		1		0.95			SEVERE HIE			SEVERE HIE		SEVERE HIE			
		1		0.75			N								
		1		0.53			PVWM								
		4		0.9			PVLM								
		1		0.94			PVWM								
		1		0.7			N								
	1			0.8			GMH								
	2			0.72			GMH,IVH								
	1			0.62			GMH								
		1		0.4			SEVERE HIE			SEVERE HIE		SEVERE HIE			
		1		0.9			SEVERE HIE			SEVERE HIE		SEVERE HIE			
		1		0.7			N								
		1		0.8			PVWM								
		1		0.6			PVWM			FTPO					
				0.7			PVWM								
		1		0.8			PVL								
	3			0.5			GMH,IVH				HYDRO				
				0.7			PVWM			SCWM FTPO					
		1		0.6			N								
		1		0.7			N								
				0.8			PVWM								
		1		0.9			SEVERE HIE			SEVERE HIE		SEVERE HIE			
		1		0.48			SEVERE HIE			SEVERE HIE		SEVERE HIE			
	2			0.5			GMH								
		1		0.69			PVWM								
		1		0.96			SEVERE HIE			SEVERE HIE		SEVERE HIE			
		4		0.7			PVWM			FTPO					
	2			0.49			GMH,IVH,IPH								
				0.68			N								
		1		0.72			PVWM								
		3		0.85			PVL			FTPO					
		3		0.7			PVL			FTPO					
		4		0.8			PVL								
				0.83			PVWM								
		2		0.7			PVWM								
		1		0.8			SEVERE HIE			SEVERE HIE		SEVERE HIE			
				0.73			N								
		1		0.98			SEVERE HIE			SEVERE HIE		SEVERE HIE			
				0.83			PVWM			FTPO RD CC					
		1		0.6			PVWM			FTPO RD CC					
		1		0.5			PVWM								
		1		0.57			SEVERE HIE			SEVERE HIE		SEVERE HIE			
		4		0.6			PVWM								
		1		0.58			PVL CYSTS								
		2		0.6			PVL CYSTS								