NEUROSONOGRAM IN CRITICALLY ILL NEONATES IN NEONATAL INTENSIVE CARE UNIT IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE HOSPITAL SALEM

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GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE HOSPITAL

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DECLARATION

I, DR.P.PRABHU MBBS., solemnly declare that the Dissertation titled **"NEUROSONOGRAM INCRITICALLY ILL NEONATES IN NEONATAL INTENSIVE CARE UNIT IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICALCOLLEGE HOSPITAL SALEM"** had been prepared by me under expert guidance and supervision of **Prof..Dr.P.SAMPATHKUMAR,MD.,DCH.,** Professor and HOD, Department of Paediatrics, Government mohan kumaramangalam medical college hospital, salem. The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the regulation for the award of M.D. Degree (Branch VII) in Paediatrics. It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

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Instances where selected sources appear:

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LISTOFABBREVIATIONS

AIDS	→	AcquiredImmunodeficiencySyndrome
APH	→	AntepartumHaemorrhage
BG	→	BasalGanglia
BGT	→	BasalGangliaThalamus
BGTH	→	BasalGangliaThalamicHyperechogenecity
CHD	→	CongenitalHeartDisease
CMV	→	Cytomegalovirus
CNS	→	CentralNervousSystem
CP	→	CerebralPalsy
CSF	→	CerebrospinalFluid
CT	→	ComputedTomography
CUS	→	CranialUltrasound
DAMA	→	DischargeAgainstMedicalAdvice
DOB	→	DateofBirth
DOPPLERHUS	→	DopplerHeadUltrasound
DWM	→	Dandy–WalkerMalformation
EDD	→	ExpectedDateofDelivery
EEG	→	Electroencephalograph
GA	→	GestationalAge
GBS	→	GroupBStreptococcus
GMH	→	GerminomatrixHaemorrhage

HC	→	HeadCircumferences
LMP	→	LastMenstrualPeriod
HIE	→	HypoxicIschemicEncephalopathy
HIV	→	HumanImmunodeficiencyVirus
HPI	→	HaemorrhagicParenchymalInfarction
HSV	→	HerpesSimplesVirus
ICH	→	IntracranialHaemorrhage
IEM	→	InbornErrorsofMetabolism
IUD	→	IntrauterineDeath
IUGR	→	IntrauterineGrowthImaging
IVH	→	IntraventricularHaemorrhage
LBW	→	LowBirthWeight
MOD	→	ModeofDelivery
MRI	→	MagneticResonanceImaging
NBR	→	NewBornReflexes
NICU	→	NeonatalIntensiveCareUnit
NTD	→	NeuralTubeDefects
PHH	→	Post-haemorrhagicHydrocephalus
PIH	→	PregnancyInducedHypertension
PROM	→	PrematureRuptureofMembranes
PVD	→	Post-hemorrhagicVentricularDilation
PVHI	→	PeriventricularHemorrhagicInfarction
PVL	→	PeriventricularLeukomalacia

RDS	→	RespiratoryDistressSyndrome
REC	→	Recovered
RI	→	ResistiveIndex
SAH	→	SubarachnoidHaemorrhage
STORCH	→	Syphilis,Toxoplasma,OtherAgents,Rubella, Cytomegalovirus,HerpesSimplex
TOB	→	TimeofBirth
TORCH	→	Toxoplasma,OtherAgents.Rubella,Cytomegalovirus,HerpesSimplex
US	→	Ultrasound
V	→	Vertex
VLBW	→	VeryLowBirthWeight
VPShunt	→	VentriculoperitonealShunt
WM	→	WhiteMatter

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ABSTRACT

INTRODUCTION

Of the late due to many developments in modern neonatal intensive care, the survival of the cririically ill neonates has greatly improved. Neurosonogram is the most common and easily repeatable imaging technique for the neonatal brain showing brain development and the most frequently occurring forms of cerebral injury in the preterms and terms.. This study is to assess the importance of cranial ultrasound as a investigatory modality for critically ill neonates .To find out the morphology of various cerebral lesions and to correlate neurosonogram finding with the clinical findings.

METHODS

An observational clinical study conducted at Government Mohan Kumaramangalam Medical College and Hospital, Salem involving 100 critically ill neonates admitted neonatal intensive care unit were subjected to neurosonography. Perinatal details were recorded and clinical examination with appropriate investigations was done. CUS was done and morphology of various findings was studied and recorded. Clinical correlation with CUS findings and follow up was done.

RESULTS

On cranial ultrasound, 37% of all neonates had abnormal findings. Of the all neonates with term gestation having abnormal findings on Neurosonogram, 60% had cerebral edema, 35% showed periventricular flare and 15% had periventricular leucomalacia. Correlation between Neurosonogram findings of neonates with sepsis, birth trauma, seizures and prematurity was not statistically significant. There was statistically significant correlation between various findings on Neurosonogram and clinical outcome of the neonate. Most of neonates died had abnormal cranial ultrasound.

CONCLUSION

High efficacy of Neurosonogram in identifying incidence of brain damage and its evolution on regular follow up guides clinical decisions and prognosis. This is particularly important in the expectation of potential preventive, protective, and rehabilitative strategies for the management of critically ill newborn infants.This study concludes cranial ultrasound is critical as an investigatory modality in NICU and effectively documents morphology of brain damage.

KEYWORDS

Critically ill neonates; Neurosonogram; NICU; Outcome

INTRODUCTION

Neurosonogram is an important diagnostic modality in developing neonatology for viewing normal and abnormal variations in neonatal brain. In general, sutures and fontanels of all neonates are open and these fontanalles can be used as acoustic windows to view into thebrain.[1]

Any neonate, irrespective of their birth weight or gestational age, who has the high chance of morbidity or mortality, due to fetal, maternal or any abnormalities or due to complicated pregnancy, within first four week after delivery is considered to be high risk neonate.[2] Neurosonogram is used in assessing neurological outcome of these critically ill neonates.

Patient friendly, simple, non-invasive and can be used eveninstantly after birth. It can be used frequently and whenever necessary, and thereby facilitates imaging of brain maturation and development of brain lesions, and also useful to evaluate timing of braindamage.[1]

Recent advances in ultrasonography has made significant improvement in image quality. Inventions of additional acoustic windows have significantly improved the diagnostic capacity of neurosonogram. Imaging the posterior and the mastoid fontanelle, is used to detect lesions structural malformations in hind brain and adjacent sub cortical white

matter, imaging through temporal window has improved views on the mesencephalon and brainstem.[3]

Cranial ultrasound can be used at bedside for imaging neonatal brain. Nowadays cranial ultrasound is used more commonly in identifying congenital and acquired lesions of the perinatal brain and patterns of brain injury in all asphyxiated neonates. It identifies most of the brain lesions , infections and structural abnormalities in preterm and full term newborns.[1]

Cranial ultrasound is used in early identification of the conditions leading to neonatal encephalopathy which causes seizures in the neonate and also used in monitoring changes occurring in course of hypoxic-ischemic brain lesions.

In critically ill neonates and in newborns with serious cerebral abnormalities, cranial ultrasound plays an important role in deciding whether to continue or withdraw intensive treatment. In surviving newborns, it is helpful to improve treatment during the neonatal period and also on further follow up.[4]

Appropriate transducers are used at appropriate time so that imaging quality will be good and better viewing can be made like in the case of preterm births where serial imagings are done at regular intervals until term age and most of the diagnoses will be detected, which is highly

useful in prognosis and follow up of the neonate. Serial cranial ultrasound examinations are used to assess the inception of injury and differentiate further development of lesions.[1]

The basic criteria for using Cranial ultrasound screening is first to understand that whether it is suitable for neonates without neurological symptoms ,and in neonates with brain pathology and for neonates with stable brain abnormalities. In cases of any cerebral and neurological abnormalities present, the intensity and frequency of using neurosonogram may be decided depending upon the clinical picture and the lesions.[1]

Most of the NICU's perform serial cranial ultrasound examinations in the course of hospitalization for premature infants and follow-up examination is done at time of discharge. All these are done to document the presence of any haemorrhagic lesions, which are useful in planning choice of treatment that might reduce further risk of haemorrhage, and to prevent further neuronal damages.[5]

Early imaging of the brain can detect lesion such as IVH, HPI and cystic PVL. Recent modalities are targeted more at detecting subtle white matter changes, assessing brain growth and maturation, so that these are used in predicting neurodevelopment outcome.

Appropriate timing of neurosonogram plays vital for correct

prediction in neurodevelopment outcome. Many studies have discussed that in 40–50% of preterm babies with CP, lesions can be detected on neurosonogram as early as possible. However, if only one or two sonological examination is made, the detection of cystic PVL, the most predictive sonological marker for CP, is less reliable.[6]

In term infants neurosonogram plays asignificant role in the diagnosis of lesions in newborns presenting with hypoxic ischemic encephalopathy (HIE) , focal abnormality in the basal ganglia and thalami (BGT), stroke and other focal lesions which showsigns of metabolic and congenital disorders.[7]

Neurosonogram plays crucial role in assessing severity and neurodevelopment outcome of babies with HIE. In cases of apparently mild or suspected brain injury, it is advisable to undergo serial neurosonogram evaluations until stabilisation of abnormalities that has occurred.[7]

Meningitis and other fulminant brain infections can have a very rapid course which should be intensively monitored with serial neurosonogram examinations.

Diagnosis made by using neurosonogram, depends on the use of apt ultrasound machine for neonates with suitable settings and probes,

also the knowledge and experience of the examiner.[10] Recently developed machines and probes use varieties of acoustic windows and adequate scanning protocols to give high-definition images that are diagnostically accurate.[10]

AIMS AND OBJECTIVES

- To know the importance of neurosonogram as an investigatory modality for critically ill neonates.

- To detect morphological changes occurring in various cerebral lesions

- To relate cranial ultrasound finding with the clinical findings and factors causing such brain lesions

REVIEW OF LITERATURE

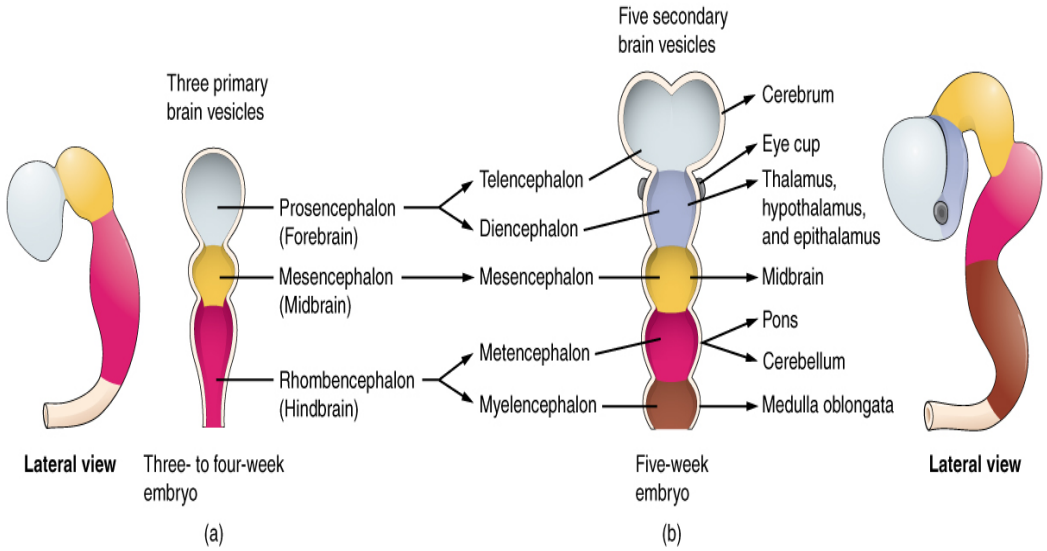
EMBRYOLOGY AND SONOLOGICAL ANATOMY OF BRAIN

Central nervous system development in humans is very complex.[11-14]usually brain starts to develop by dorsal induction at approximately 14 days after fertilization with development of the neural plate on dorsal part of the embryo. The transformation of the neural plate to a tube starts at the day 21. The fusion starts in the midpoint and spreads cranially and caudally with the cranial end fusing on day 24 and caudal end on day 26.[15]

In the start of the 4thweek, the cranial end of the neural tube endures expansion, flexion and fusion of the neural pore to give rise to three primary brain vesicles: the forebrain or prosencephalon, the mid brain or mesencephalon, and the hind brain or rombencephalon. The prosencephalon gives rise to telencephalon and diencephalon. The telencephalon will develop into the cerebral hemispheres with their lateral ventricles and third ventricle, the diencephalon develop into thalamus, hypothalamus and the neurohypophysis of the pituitary gland. The mesencephalon will grow into midbrain. The rhombencephalon gives rise to myelencephalon and metencephalon. The myelencephalon forms the medulla oblongata and the metencephalon forms the pons and

the cerebellum.

During the late foetal and perinatal period and during early infancy, major maturational processes and development of the brain take place. Because of this constant maturation, the preterm brain in particular is very susceptible to aberrant development and damage. Patterns of perinatal brain injury be determined by not only on the origin of the injury but also on the post-conceptional age of the foetus or infant at the time of the event(s).

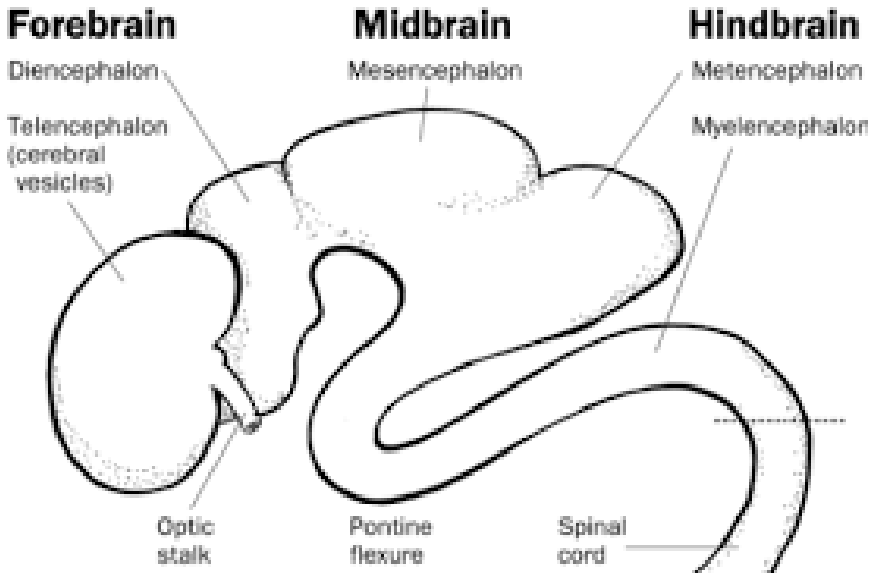


Maturational phenomena gives us very specific neurososonogram features, and images change with ongoing evolution. Maturational processes include a major rise in volume, weight, and surface area of the brain; gyration; cell migration; germinal matrix involution; and myelination.[16]

Gyration starts in the second trimester of pregnancy, continues in

an well-ordered, anticipated way, and is accomplished around term age when the brain surface has an almost mature appearance. In extremely preterm infants (GA 24–26 weeks), the brain surface is still very smooth Normally, the posterior parts of the brain show the fastest development, and the anterior parts of the brain gyrate much later.[16]This can be followed by neurosonogram which also helps to assess the GA of the infant from the ultrasound images.

Like gyration, myelination is a very well-ordered and predictable process. Generally the central parts of the brain myelinate before the peripheral parts, and the posterior parts before the anterior. Myelination, cell migration, and germinal matrix involution result in white matter changes, which are shown on neurosonogram.[16]



In normal preterm infants, we may identify subtle areas of symmetrical increased echogenicity, primarily in the frontal regions. These areas of amplified echogenicity characterize glial cell migration and should be distinguished from pathological periventricular flares.

The germinal matrix is abundant, cellular and vascular “strip” of sub-ependymal tissue and on neurosonogram, the germinal matrix can be distinguished as small areas of high echogenicity, mostly only visible around the thalamo-caudatenotch.[16]

On cranial USG of preterm infants, the thalami and basal ganglia undergo changes. In very preterm infants these deep grey matter structures may show a diffuse, subtly increased echogenicity in comparison with surrounding tissue. This diffuse, subtle, echogenic “haze” over the basal ganglia and/or thalami can be a normal finding in very preterm neonates, whereas in term neonates, increased echogenicity in this same region often presents hypoxic-ischemic injury, with possible, sometimes serious, consequences for neurodevelopment.[16]

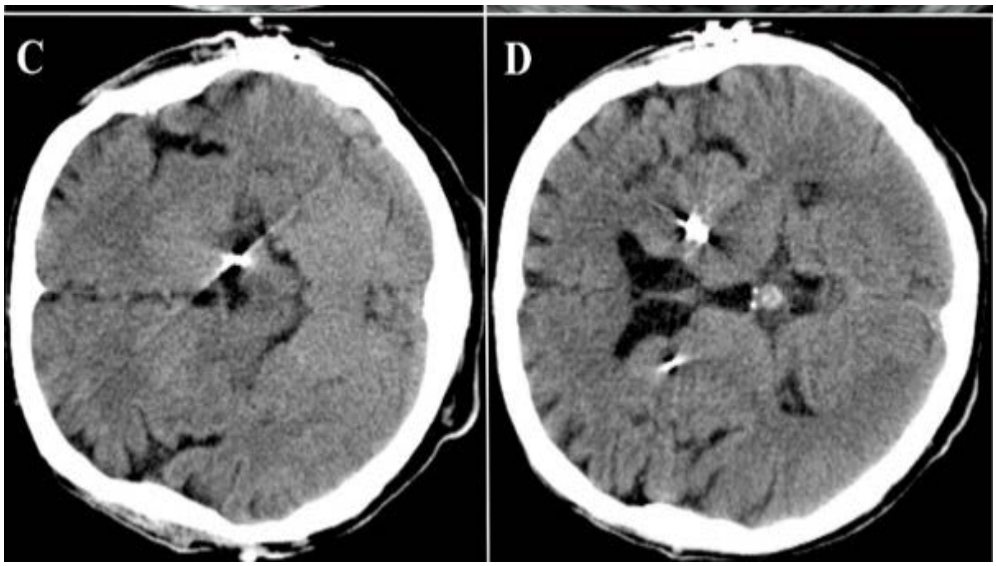
HISTORY

Brain was the first organ to be thoroughly examined by ultrasound, using A mode Echo Encephalography. A mode Encephalography was initiated in early 1950s to identify midline structure, shifts, and to obtain rough estimation of ventricular size. 2 D Echo encephalography came into act in 1960s with better visualisation of ventricular size and intracranial spatial relationship. This A-mode scanning and static gray scale imaging are now only of historical interest.

The linear array units with large transducers have now been substituted by sector and linear, high frequency, small head transducers that easily fit over the fontanells. Transducers come in many different frequencies. Increasing frequencies improves resolution but decreases penetration. Therefore higher frequency transducers are used in neonatal brain examinations.

The resolution of images since Leksell's use of ultrasound in 1956 (A-mode), and the pioneering work of Kossoff and colleagues[18], and Garrett and colleagues[19]on ultrasound of the normal and hydrocephalic neonatal brain have markedly improved with the real time gray scale imaging.[20,21,22]Initially, anterior and temporal fontanell of the skull were used as windows, later in 1980 Dewbury,Aluwikaee and Babcock first stated using the mastoid fontanel as the bone free window.Transfontanel ultrasound has become extensively used method and is considered as the

primary imaging procedure for neonatal and infantile brain.The addition of color and pulsed wave Doppler has added to the versatility of ultrasound imaging through the cranium.[23,24]Newer advances in recording images from the static conventional photographic film, video tape to the newest of digital recording on the hard disc with the capability of post-processing the images has added a new aspect to neonatal brain ultrasound investigation. As a result of current progress in ultrasonography, image quality is high now-a-days, provided optimal settings and procedures are applied.



When we did review of literature Pape et al in 1978 first started using real time ultrasound scanner for detection of IVH in preterm neonates.[25]Later in 1982 Volpe JJ established that haemorrhagic leukomalacia can be diagnosed reliably by real-timeultrasound. [26]Few

other researchers found that cranial ultrasound abnormalities, especially a white-matter echolucency, predict future disability.[27]

In 1995 Soni JP supported the view that neurosonogram is a sensitive and specific technique for the detection of ICH , its types and for the detection and monitoring of complications of ICH observed in high risk term neonates.[28]

In 1997 Ancel PY in his EPIPAGE Cohort Study evaluated 1954 infants born between 22 and 32 weeks of gestation in 9 regions of France and re-examined again at 2 years age. He exposed that the risk of cerebral palsy varies according to the presence and type of cranial ultrasound abnormalities diagnosed in the neonatal period.[29]Eugenio Mercuri in 1998 studied in a cohort of 177 asymptomatic newborns and determined that abnormalities were associated with maternal risk factors or deviant neurological signs.[30]

A study done by Holling EE in 1999 found echolucency as the sonographic indicator of white- matter destruction and suggested the term echolucency to the diagnosis of cystic periventricular leukomalacia.[32]

Pierrat V in 2001 strongly recommended that weekly US examinations to be performed in all at risk infants until discharge to compare the ultrasound (US) evolution and neurodevelopmental outcome of infants with localised (grade II) and extensive (grade III) cystic

periventricular leukomalacia.[34]

De Vries in 2006 identified that periventricular leukomalacia (PVL) is the most vital factor of neurologic morbidity seen in preterm infants. The cystic form well visualised by cranial ultrasonography, is usually associated with the development of cerebral palsy (CP) in infancy, the diffuse form, may relate to cognitive and behavioural problems seen commonly in these pre-terms.[39]

Soghier LM[40]in 2006 did serial Neurosonograms among 289 neonates with gestational age (GA) <34 weeks during a 21-month period and reported, diffuse BGTH was associated with factors similar to those previously reported in various studies.

Cowan FM in 2007 indicated that cranial ultrasound offers bedside imaging access to the neonatal brain and is sensitive modality in the early identification of neonatal encephalopathy in the term infant and the subsequent monitoring of brain injury.[42]

Thus Cranial ultrasonography is commonly used to identify neonates at increased risk for neurodevelopmental impairment. Another study done by Michael O'Shea T in 2008 explained the relationships between neurosonogram abnormalities and delayed milestones at 2 years of age in extreme preterms and also found that the association of white matter damage with developmental impairments applied to extreme preterm newborns and is stronger for motor, as compared with mental

development.[44]

Gupta SN in 2009 studied intracranial hemorrhage in term neonates and found that its incidence although rare is a diagnostic challenge and prompt uncovering of Intracranial hemorrhage is facilitated by selection of appropriate neuroimaging, identification of the treatable etiologies and management directed to limit secondary braininjury.[47]Later in 2009, Karl C. K. Kuban described in his study that cranial ultrasound scans obtained in NICU predicted cerebral palsy types and severity of motor dysfunction when these children were followed up at 2 years old, corrected age.[48]

IDENTIFYING HIGH RISK PREGNANCIES AND HIGH RISK NEWBORNS

Basically Neonates at risk should be recognized promptly to decrease neonatal morbidity and mortality. The term high-risk infant designates an infant who should be under close observation by experienced physicians and nurses. If these high-risk deliveries are recognized in advance, their progress during labour and delivery should be closely monitored and resuscitation can be initiated at birth.[50]For any given duration of gestation, the lower the birth weight, the higher the neonatal mortality; for any given weight, the shorter the gestational duration, the higher the neonatal mortality. The highest risk of neonatal mortality occurs in infants who weigh<1,000 grams at birth and whose gestation was <28 weeks. The lowest risk of neonatal mortality occurs in infants with a birth weight of 3,000-4,000 gms and a gestational age of 38-42weeks.[50-51]

The following factors influences the newborn health condition

A. Age atdelivery

a. If more than forty years. Chromosomal abnormalities, macrosomia, intrauterine growth restriction (IUGR), blood loss (abruption, previa).

b. Under 16 years. IUGR, prematurity, child abuse/neglect (mother herself may beabused).

c. Personal Factors

d. Poverty causes Prematurity, infection,IUGR.

e. Smoking causes IUGR, increased perinatal mortality.

f. Drug/alcohol use: IUGR, fetal alcohol syndrome, maternal heroine abuse, withdrawal syndrome, sudden infant death syndrome and child abuse/neglect.

g. Maternal medical conditions and associated risk for fetus or neonate

h. Diabetes mellitus: Congenital anomalies, stillbirth, respiratory distress syndrome (RDS), hypoglycemia, macrosomia/birthinjury.

i. Thyroid disease: Goitre, hypothyroidism,hyperthyroidism.

j. Renal disease: IUGR, stillbirth,prematurity.

k. Urinary tract infection: Prematurity,sepsis.

l. Heart and lung disease: IUGR, stillbirth,prematurity.

m. Hypertension (chronic or pregnancy-related): IUGR, stillbirth, asphyxia,prematurity.

n. Anemia: IUGR, stillbirth, asphyxia, prematurity,hydrops.

o. Obstetric history and associated risk for fetus or neonate

p. Past history of infant with prematurity, jaundice,RDS.

q. Maternal medications.

r. Bleeding in early pregnancy: Stillbirth and prematurity.

s. Hyperthermia: Fetal demise, fetal anomalies.

t. Premature rupture of membranes:Infection/sepsis.

u. TORCH infections.

v. Trauma: Fetal demise, prematurity.

B. FETALCONDITIONS

1. Multiple gestation: Prematurity, twin-twin transfusion syndrome, IUGR, asphyxia, birth trauma.
2. Macrosomia: Congenital anomalies, birth trauma,hypoglycemia.
3. Abnormality of fetal heart rate or rhythm: Hydrops, asphyxia, congestive heart failure, heartblock.
4. Polyhydramnios
5. Oligohydramnios

C. CONDITIONS OF LABOUR ANDDELIVERY

1. Premature labour: RDS, other issues ofprematurity.

2. Post-term labour: Stillbirth, asphyxia, meconium aspiration

3. Maternal fever, infection/sepsis.

4. Maternal hypotension, stillbirth,asphyxia.

5. Rapid labour. Birth trauma, intracranial hemorrhage (ICH), retained fetal lung fluid/transient tachypnea.

6. Prolonged labour, stillbirth, asphyxia, birth trauma.
7. Abnormal presentation, birth trauma,asphyxia.
8. Uterinetetany
9. Meconium-stained amniotic fluid, stillbirth,asphyxia.
10. Placental anomalies

D. IMMEDIATELY EVIDENT NEONATALCONDITIONS

- Prematurity, RDS, other sequelae of prematurity
- Low 5-minute Apgar score, prolonged transition (especially respiratory).
- Low 15-minute Apgar score, cardiac failure, renal failure, severe neurological damage.
- Pallor or shock, Bloodloss.
- Foul smell of amniotic fluid or membranes, infection.
- Small size for gestational age(GA)
- Post-maturity.

PATHOGENESIS

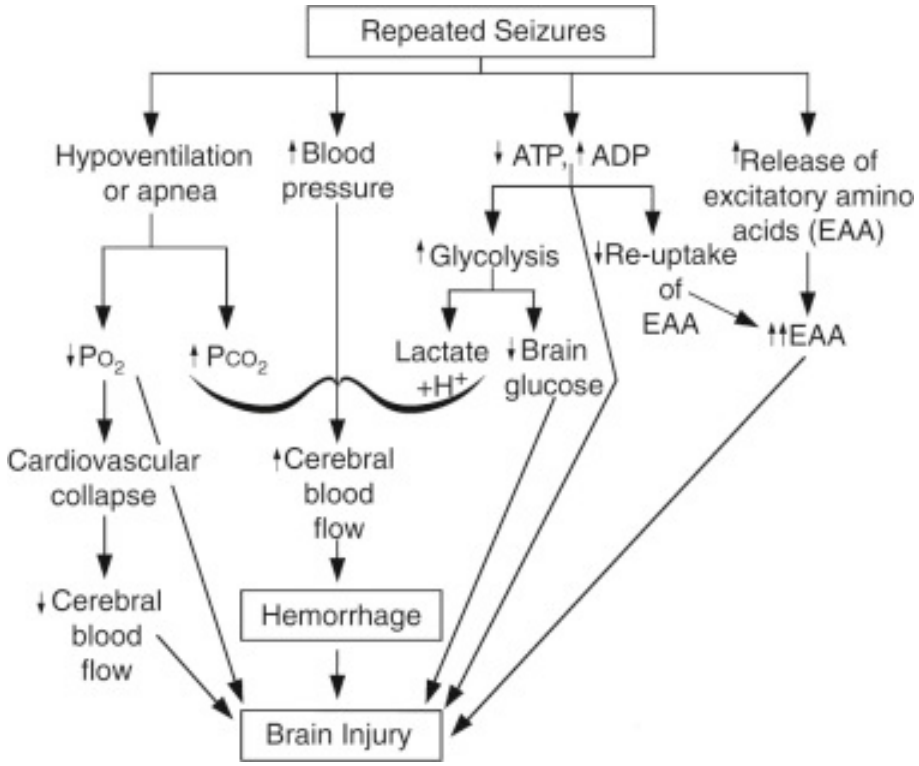
NEONATAL CONVULSIONS

Seizures are the most unique expression of neurologic dysfunction in the newborn infant. Moreover, neonatal seizures often represent possibly devastating forms of brain injury. Recent advances in diagnostic technology have provided significant perceptions into neonatal seizures.[52]

Neonatal seizures differ significantly from seizures witnessed in older children, mainly because the immature brain is not as much of capable of transmitting generalized or organized electrical discharges.[52]

Seizure activity is clinically classified as subtle, clonic, tonic and myoclonic types. Principal etiological factors linked are cerebral hypoxic ischemia, intracranial bleeds, CNS infection, metabolic diseases comprising inborn errors of metabolism and infrequently cerebral dysgenesis, neonatal epilepsy syndromes.[52,53]

Although individual seizure types are not suggestive of specific varieties of brain injury, certain seizure types are related more frequently with some conditions. For example, generalized tonic seizures, which may symbolize brainstem release phenomena or posturing, have been perceived with major germinal matrix/intraventricular hemorrhage (GMH/IVH). Focal clonic seizures may be related with focal cerebral infarction or traumatic cerebral contusion.[52-56]



After seizures are confirmed and treatment has begun, the etiology should be tracked through a sensible and orderly method, with a stepwise understanding of the facts. The assessment should start with a careful history of pregnancy, labour and delivery, and family, followed by a comprehensive clinical examination for signs of dysmorphism, trauma, skin lesions, and unusual odours. The neurologic examination should include a careful and precise clinical description of the seizure features, the infant's detailed central nervous system examination.[52-56]Using such an orderly and rational approach, most neonatal seizure etiologies should be identifiable.[53]

Neurosonogram has an vital role in the finding of significant lesions in infants presenting with hypoxic ischemic encephalopathy (HIE) and seizures; these include focal abnormality in the basal ganglia and thalami (BGT), stroke and other focal lesions and indicators of metabolic and infectious disorders.[52-56] Hence the most important determinant of outcome is the underlying neurologic disease.[52]

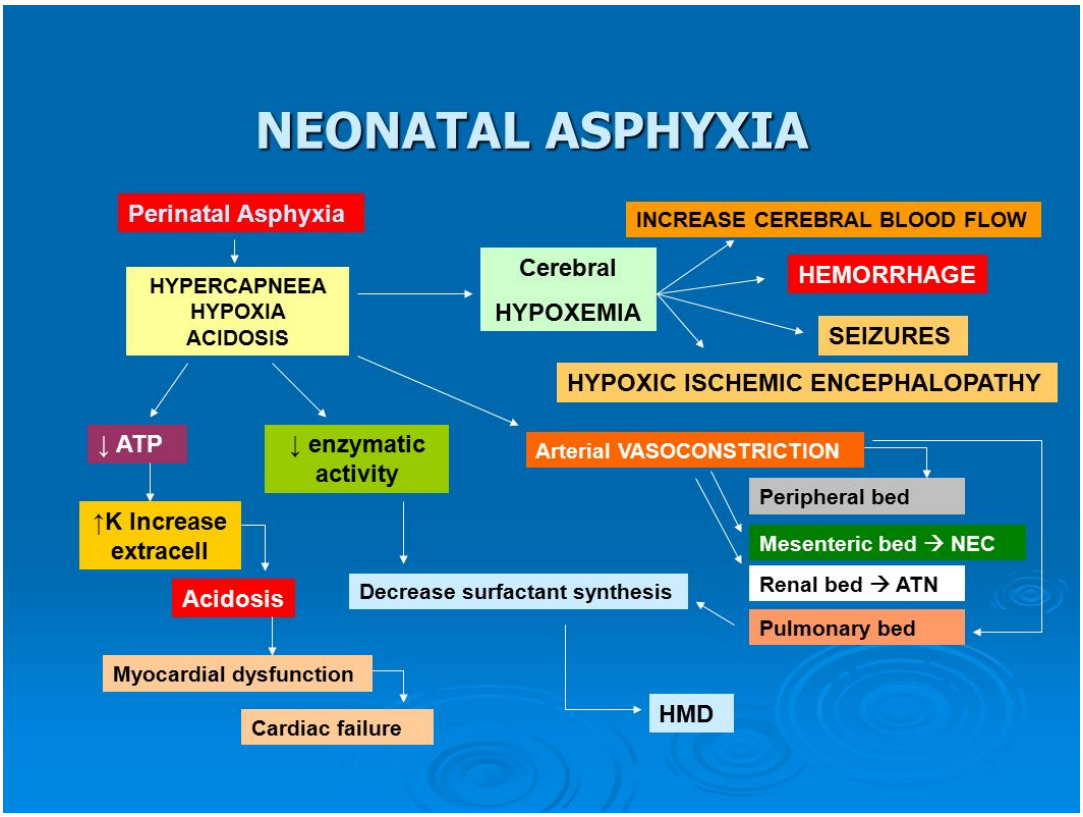
PERINATAL ASPHYXIA

It refers to a illness of impaired gas exchange that leads, to fetal hypoxemia and hypercarbia. It occurs during the first and second stage of labour occurs as result of fetal acidosis which can be measured from umbilical artery blood. The umbilical artery pH of <7.0is most widely accepted as the definite criteria for presence of asphyxia.

In term infants, 90% of asphyxial events occur in the antepartum or intrapartum period as a result of compromised gas exchange across the placenta. The rest of these events occurs in the postpartum period and is usually secondary to pulmonary, cardiovascular, or neurologic abnormalities.[57,58]

Neonatal encephalopathy is an abnormal neurobehavioral state that of decreased level of consciousness associated with abnormal neuromotor tone. It characteristically begins within the first postnatal day and may be

associated with seizure-like activity, hypoventilation or apnea, depressed primitive reflexes and the appearance of brain stem reflexes.[57-59]



Hypoxic-ischemic encephalopathy (HIE) is a clinical condition in which abnormal cerebral blood flow leads to abnormal neurodevelopmental outcome.[57]

NEUROPATHOLOGY OF BRAIN INJURY

The pathogenic mechanisms that decide the major neuropathologic patterns of injury may be described by a combination of regional circulatory and metabolic factors of the affectedbrain.[58] Initial circulatory change to perinatal asphyxia occurs as redistribution of cardiac output

with accelerated perfusion of vital organs, like brain, heart, adrenals and decreased blood supply for other organs. Sustained hypoxia insult cause systemic hypotension which further leads to impairment of cerebrovascular autoregulation. Systemic hypotension, causes moderate reductions in cerebral perfusion which lead to injury involving mainly the watershed zones of arterial supply. In term newborn, the watershed zones between dividons of cerebral arteries that are located in parasagittal regions of cerebral cortex. In the premature neonates, the watershed zone is located in periventricular white matter.[58-61]

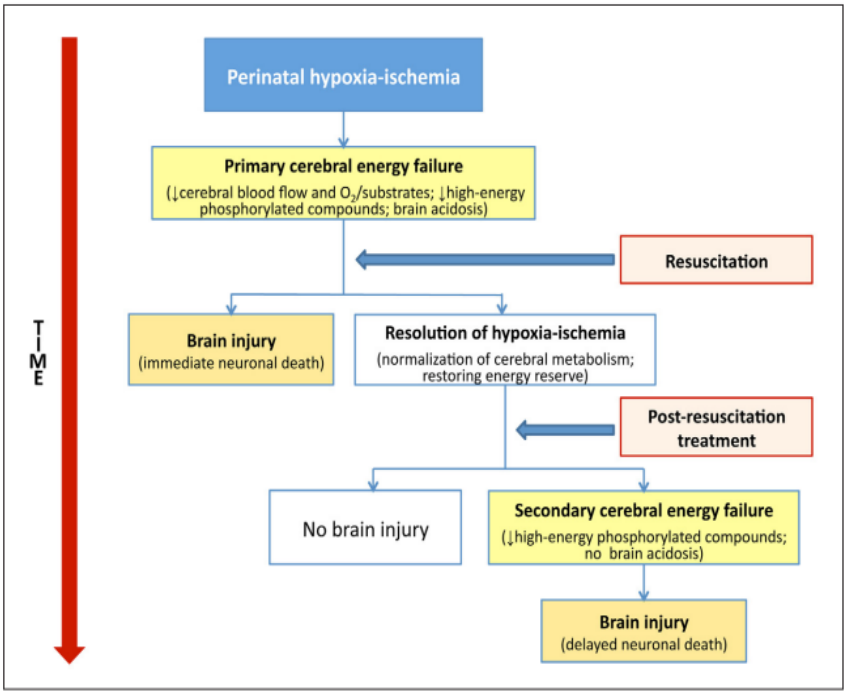


Figure 1. Schematic representation of primary and secondary energy failure in the brain following perinatal asphyxia.

Excitatory aminoacid receptors play crucial role in hypoxi-ischemic injury by causing cell mediated injury. Recent experimental

evidences suggest that excitatory neurotransmitters, like glutamate, play a vital role in the causing hypoxic-ischemic neuronal injury.[57-62]

Selective Neuronal Necrosis[58] involves specific regions of cortex like hippocampus, thalamus, cerebellum, and anterior horn cells of spinal cord. The late sequelae of selective neuronal necrosis which involves the cortex and subcortical white matter may lead to cerebral atrophy following severe injury.

MulticysticEncephalomalacia

In term newborn between parasagittal cortex and white matter there is a watershed zone of arterial supply between major cerebral arteries and its branches, an area which is more prone for ischemic injury.

Periventricular Leukomalacia (PVL)[63-65]

It is a lesion found primarily in the preterm infant, and it is the neuropathologic lesion probably responsible for the cognitive, motor, and sensory impairments.

The characteristic neuropathology of PVL[63-67]was first defined classically by Banker and Larroche in 1962 as bilateral areas of focal necrosis, gliosis, and disruption of axons, with the so-called “retraction clubs and balls.” The structural distribution of the lesions was noted to be in the periventricular white matter dorsolateral to the lateral ventricles,

primarily anterior to the frontal horn (at the level of the foramen of Monro) and lateral to the occipital horns.

They therefore suggested two key features of the pathogenesis of PVL, namely:

Hypoxia-ischemia affecting the watershed regions of the white matter, and vulnerability of the periventricular white matter of the premature brain.

This distinctive lesion of PVL found in the immature white matter of premature newborns likely results from the interaction of multiple pathogenetic factors:

(i) vascular anatomic factors,

(ii) pressure-passive cerebral circulation,

(iii) Intrinsic vulnerability of cerebral white matter of the premature newborn and

(iv) infection/inflammation.

Although there is not one accepted classification system for PVL, **the De Vries classification**[39,63]isused:

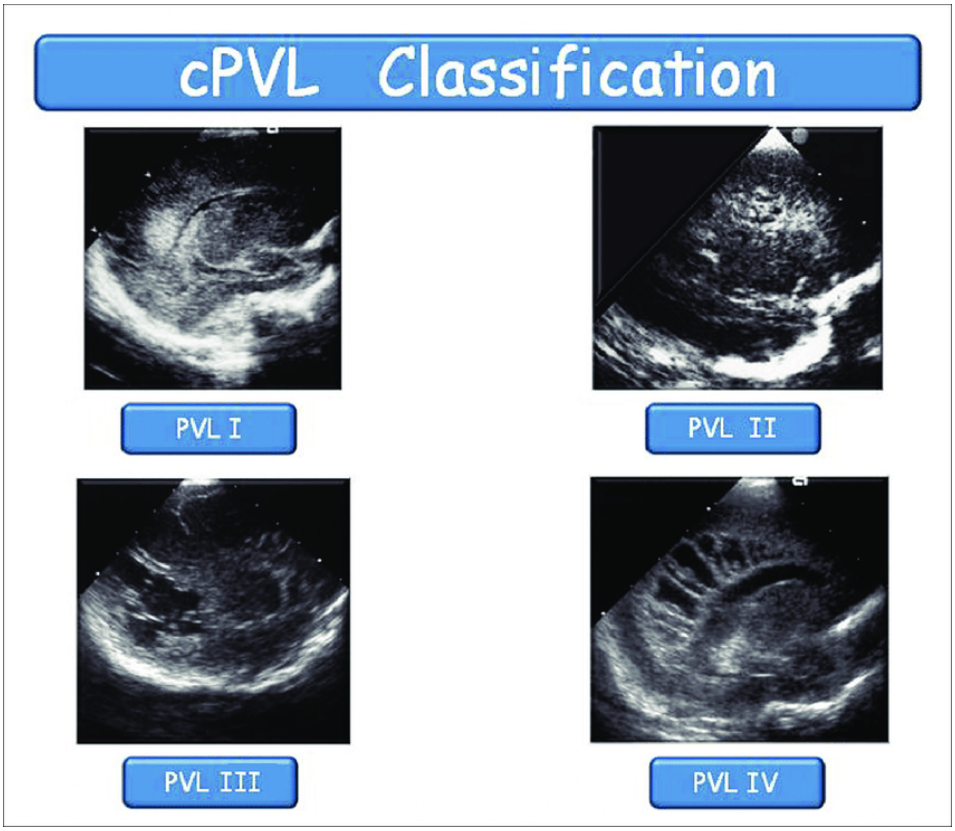
Grade I: Areas of increased echogenicity, usually seen within 24–48h after an insult and persisting beyond day 7 but not evolving into cysts.

Grade II: Localized small cysts, often located in the fronto-parietal periventricular whitematter.

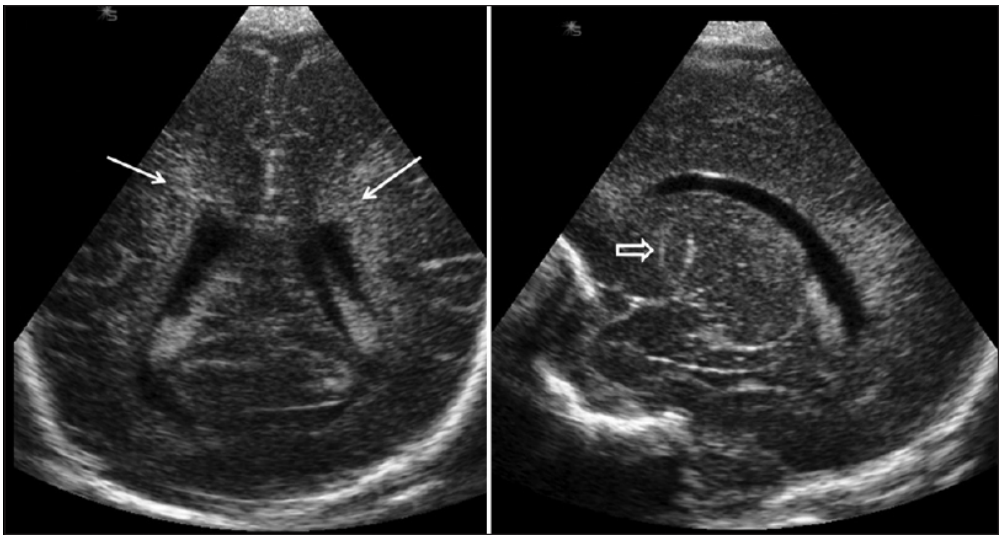
Grade III: Extensive cystic lesions, often particularly prominent in the parieto- occipital periventricular white matter. The cysts usually do not communicate with the lateral ventricle. They collapse after several weeks and are no longer visible on cranial ultrasound once the child is 2–3 months old. At this stage, irregular *ex-vacuo*ventricular dilatation can be noted, due to atrophy of the periventricular white matter.

Grade IV: Extensive cystic lesions extending into the deep (subcortical) whitematter.

FIG 1: DEVRIES CLASSIFICATION



The evolution of echogenicity in the periventricular white matter over the first few weeks after birth, with or without cysts (which are echolucent), is the classical description of PVL by ultrasonographic imaging. Ventriculomegaly due to atrophy of the periventricular white matter (i.e., volume loss) is often present within weeks. Notably, isolated ventriculomegaly is associated with an increased risk ofCP.[63-68]Severe focal lesions may be visualized in vivo by cranial ultrasonography as areas of increased echogenicity in periventricular white matter during the first days of life, which subsequently evolve into cystic lesions after approximately 2 to 3 weeks.



PVL is the principal cause of the cognitive, behavioural, motor and sensory impairments found in children born at <32 weeks' GA. There is an approximately 10% incidence of CP and up to 50% incidence of school difficulties in children born prematurely that is largely due to PVL, with PVHI being the other cerebral lesion that contributes

significantly to neurologicdisabilities.[26,34,68,69]

Term newborns who sustain sufficient intrapartum insult to result in long-term sequelae invariably demonstrate clinical evidence of acute encephalopathy, i.e., altered brain function, during the first days of life. In contrast, infants who sustain hypoxic- ischemic cerebral injury earlier in gestation may be asymptomatic during the neonatal period. It is important to consider that the clinical features of hypoxic-ischemic encephalopathy are non-specific, and similar clinical features may occur in the context of other causes of brain dysfunction.[73-78]

Seizures occur in approximately 50% of asphyxiated infants and are indicative of moderate or severe encephalopathy. In addition to the clinical examination, neuroimaging techniques are of major value.

Ultrasound has better predictive value in premature infants with hypoxic-ischemic brain injury, in whom the principal lesions during in the first days of life are increased periventricular echogenicity and intraventricular hemorrhage. The hyperechoic periventricular abnormalities evolve into cystic lesions (cavitations) during the ensuing 2 to 3 weeks in severe cases. These lesions are transient and if the phase of cyst formation is not documented by serial ultrasound examinations, a later sonogram may show only enlargement of the lateral ventricles with irregular margins.[34,63,66,76]

Table 1: Severity and outcome of hypoxic-ischemic encephalopathy in the full-term neonate

Severity	Level of consciousness	Seizures	Primitivereflexes	Brain stem	Elevated intracranial pressure	Duration	Poor outcome[a]
Mild	Increased irritability. Hyperalterness	-, Jitteriness	Exaggerated	-	-	>24 h	0
Moderate	Lethargy	Variable	Suppressed	-	-	> 24 h (variable)	20-40
Severe	Stupor or coma	+	Absent	+	Variable	>5d	100
[a]Poor outcome is defined as the presence of mental retardation, cerebral palsy, or seizures.							
+, common; -, rare							

With severe HIE, abnormality is mostly characterized on Cranial USG by diffuse brain swelling on day 1–2 followed by appearance of focal basal ganglia-thalamic hyperechogenic lesion around 3 to 4 days and often associated with increased echogenicity in White matter.

INTRAVENTRICULAR HEMORRHAGE

The preterm neonate is distinctive in all most all aspects,. Significant insults to the brain of premature neonates are infact most often clinically silent. Neonatal IVH is a variety of ICH and is

characteristic of preterms.[79-82]

Intracranial hemorrhage (ICH) incidence is 2% ->30% in neonates, based upon the gestational age (GA) and type of hemorrhage . Bleeding withcan occur: (i) into the epiduralspace , subdural space or subarachnoid spaces; and also(ii) into parenchyma; or (iii) into ventricles from subependymal germinal matrix or to the choroid plexus.

SITES OF HAEMORRHAGE

The meninges consist of the dura, arachnoid and pia mater. Hemorrhage outside the dura layer and skull is termed extradural. The inner layers fold down into the brain to form the falxcerebri, tentorium cerebelli and the falxcerebelli. The dura encircles the sinuses,namely the superior and inferior sagittal, the straight and the right and left

transverse sinus. The space inner to the dura layer and arachnoid layer is minimal and is the site of subdural hemorrhage. The subarachnoid space, however, contains CSF, arteries and veins. In the region of the sagittal sinus the arachnoid layer forms granulations that pierce the dura. These return CSF to the blood in the superior sagittal sinus. The pia mater is thin, rich in capillaries and closely adherent to the brain.[83-84]

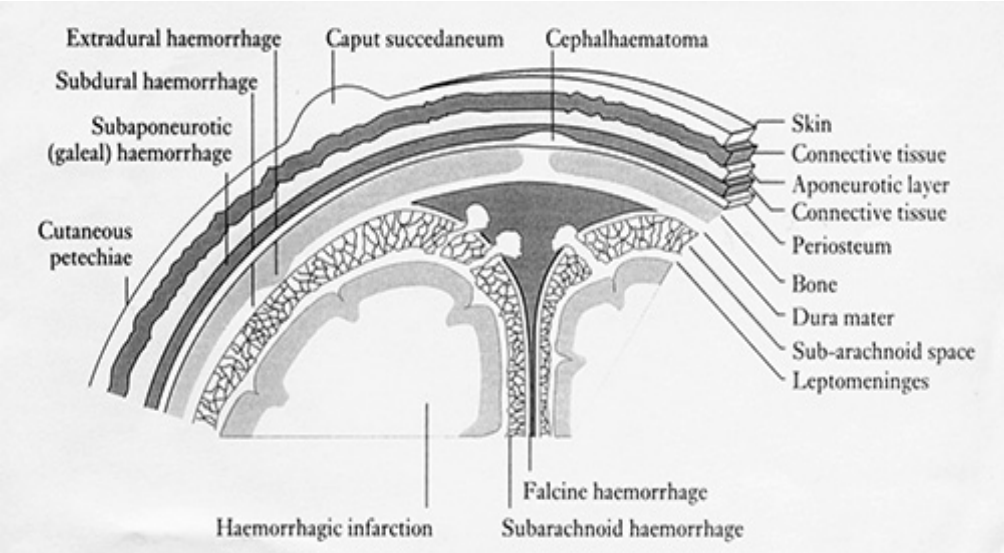


Fig. 2: Sites of intracranial haemorrhage

GERMINO-MATRIX-HAEMORRHAGE (GMH)[79-84]

GMH is found principally in the preterm infant, where the incidence is currently 15% to 20% in infants born at <32 weeks' GA, but is uncommon in the term newborn. The etiology and pathogenesis are different for these two groups of infants.In the preterm infant, GMH/IVH originates from the fragile involuting vessels of the subependymal germinal matrix, located in the caudothalamic groove.

Cranial ultrasonography is considered the neuroimaging modality of choice for the diagnosis of GMH/IVH because of its portability, high resolution, and lack of ionizing radiation. Computed tomography and MRI remain superior for the diagnosis of other types of intracranial

hemorrhage, including primary subarachnoid, convexity, and posterior fossa subdural or epidural hematomas and for differentiating between hemorrhagic and ischemic parenchymal infarction.[90-93]

Grading of GMH/IVH is important for determining management and prognosis. Grading of GMH/IVH should be assigned based on the earliest cranial USG obtained when the IVH itself is of maximal size. Specifically, ventricular dilation that occurs days to weeks following GMH/IVH is not a grade 3 IVH; it represents either PHH or ventriculomegaly secondary to parenchymal volume loss.[83,86]

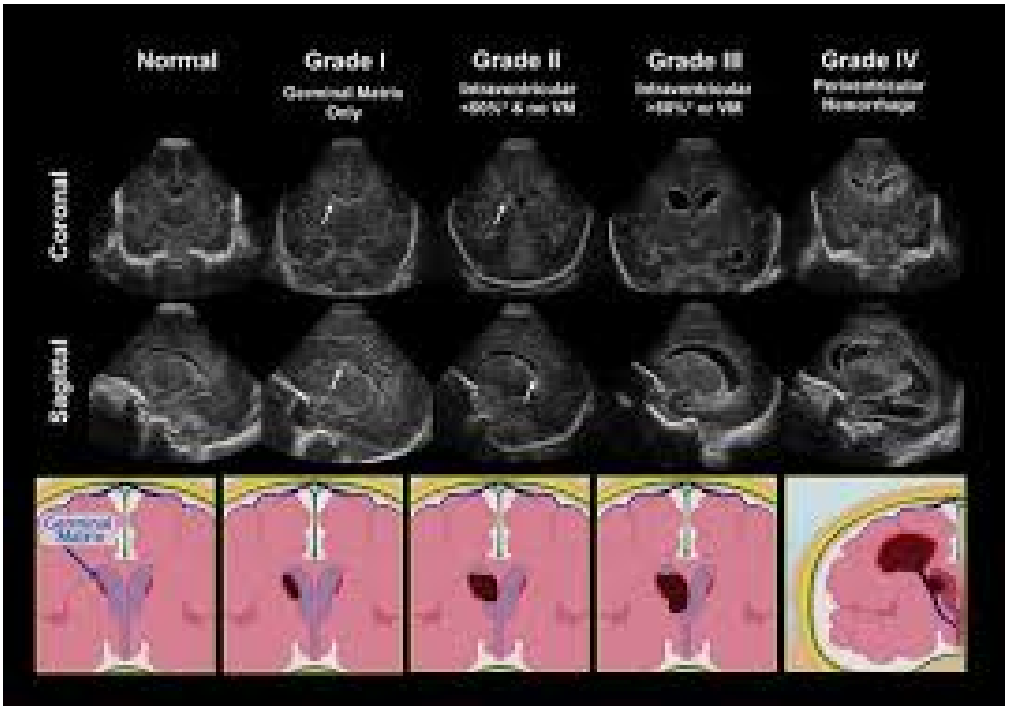


FIG 3: Grading systems reported by Papille and Volpe[83,84,86,94]is used most often.

Table 2: Grading system of GMH/IVH by NEUROSONOGRAM

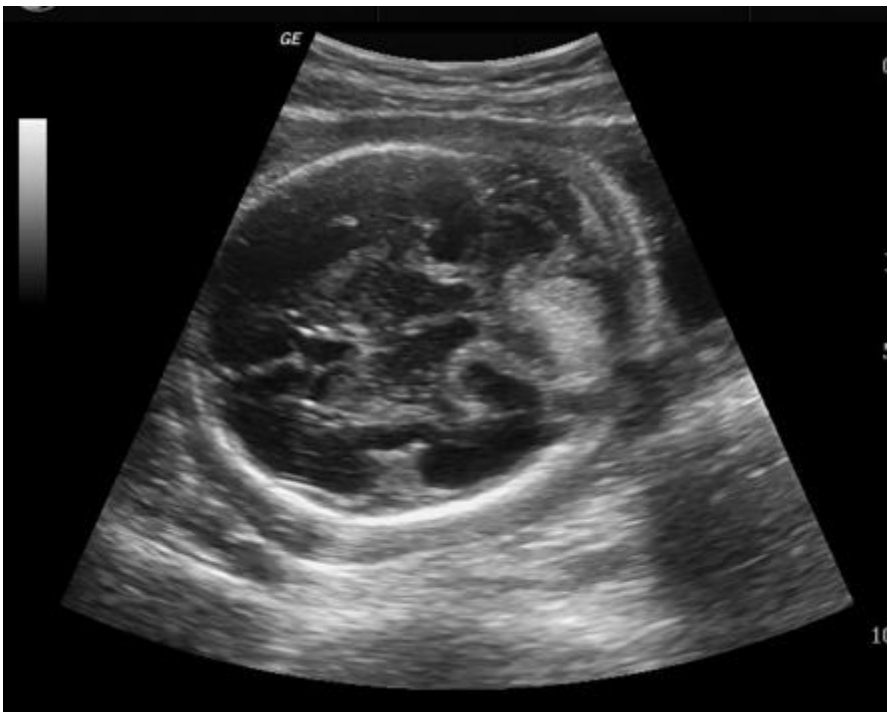
Grading System	**Severityof GMH/IVH**	**Description of findings**
Papille	1	Isolated GMH (no IVH)
	2	IVH without ventricular dilatation
	3	IVH with ventricular dilatation
	4	IVH with parenchymal hemorrhage
Volpe	1	GMH with no or minimal IVH (<10% ventricle)
	2	IVH occupys 10%- of Ventricle area on Parasagittal 50% view
	3	IVH occupying more than 50% of ventricular area on parasagittal view, usually upto lateralventricle **(at time of IVH diagnosis)**
	Separate note	Periventricular echodensity (location and extent)

Clearly, the primary goal is prevention of GMH/IVH. Prevention of premature labour and delivery may be considered to be the optimal management strategy.

SUBDURAL HAEMORRHAGE

Improvements in obstetric practice and a decrease in mechanical

birth trauma have reduced considerably the incidence of severe subdural hemorrhage. Subdural hemorrhage occurs in premature and term infants and results from laceration of the major veins and sinuses, usually associated with a tear of the dura or dural reflections (e.g., falx, tentorium) overlying the cerebral hemispheres or cerebellum.



PRIMARY SUBARACHNOID HEMORRHAGE (SAH)

SAH is located most prominently in the subarachnoid space over the cerebral convexities and in the posterior fossa, refers to hemorrhage within the subarachnoid space that is not secondary to extension of subdural hemorrhage, IVH, or cerebellar hemorrhage. Subarachnoid hemorrhage in the newborn is usually self-limited and of venous origin,

originating from small vessels in the leptomeningeal plexus or in bridging veins within the subarachnoid space. Clinically, it may be silent or manifest as seizures and uncommonly as rapid neurologic deterioration when it is massive.[80,84]

INTRACEREBELLAR HAEMORRHAGE

It is said that primary haemorrhage into the cerebellum is a relatively common lesion, especially in infants born prematurely. The routine use of neurosonogram through the posterolateral fontanelle in premature infants has confirmed that this lesion may have been under diagnosed previously in surviving infants.[80]

The lesion may be suspected on the basis of the history and physical features as described previously. Definitive diagnosis is made by CT or MRI. Traditional cranial ultrasonography through the anterior fontanelle is of limited value for visualizing intracerebellar hemorrhage but can be helpful via acoustic windows.[92,93]

Infants with post-hemorrhagic ventricular dilatation had increased change in flow resistance with anterior fontanelle compression that was correlated with increased intracranial tension and was associated with the consequent need of surgical intervention. Following cerebrospinal fluid (CSF) drainage, Doppler studies are useful in monitoring improving cerebral blood flow.[80,88,89]

NEONATAL BRAIN INFECTION

Neonatal CNS infections, whether acquired in utero, intrapartum or post-natally remain an important cause of acute and long-term neurological morbidity. Pathologic features and associated imaging patterns depend upon the stage of development of the CNS, the affinity of a specific infective agent for a specific CNS cell type, and the ability of the host to respond to that insult

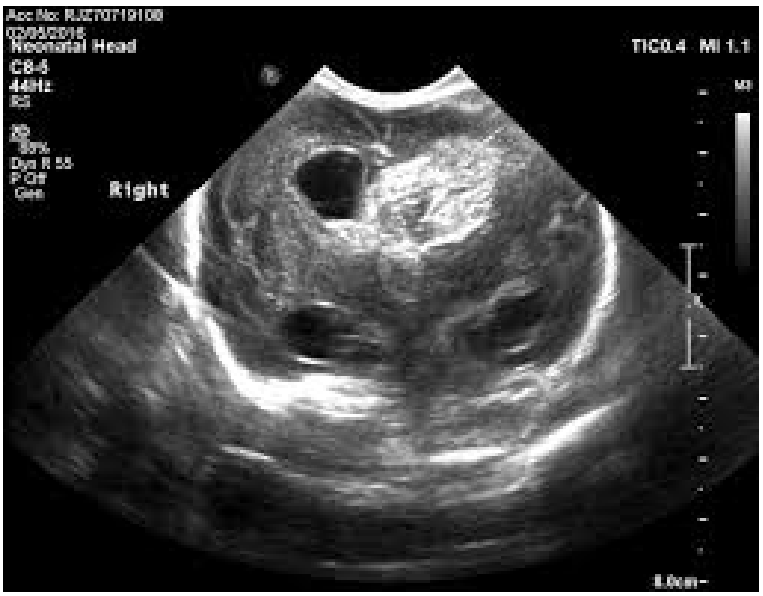
Follow-up with neurosonography and other imaging modalities in specifically indicated conditions is therefore suggested in neonates to exclude the presence of complications requiring surgical intervention or change in therapy. These complications include cerebritis, infarction, brain abscess, subdural effusion or empyema, sinus thrombosis, ventriculitis and hydrocephalus.[9,97]

Ventricular enlargement may result from occlusion of the foramina of Monro, the aqueduct of Sylvius, and the 4^{th} ventricular outlets by fibrinous inflammatory exudate which occurs even in the absence of ventriculitis. Ventriculitis, however, is a common and early component of neonatal meningitis, as the choroidplexus is a common site of bacterial entry into the CNS. Imaging features of ventriculitis should be sought in all patients with meningitis, but may be subtle in early cases. Pus-fluid

levels and ependymal thickening or a periventricular band of increased echogenicity may be seen on cranial ultrasonography is a suggestive feature. This band may also be seen with chemical ventriculitis following intraventricular hemorrhage, in association with interstitial edema related to obstructive hydrocephalus, or with periventricular calcifications in TORCH infections.[99,100]

CONGENITAL INFECTIONS (TORCH)

Considerable progress has been made in averting or handling congenital infection with immunizations and intrauterine diagnosis and therapy. Diagnosis of congenital infections, whether they reach the fetus via a hematogenous, transplacental route or by ascent through the birth canal, remains challenging.[97]



Pathologic and radiologic features differ somewhat amongst the congenital infections, although calcifications and necrosis are hallmarks. Infants with CMV frequently have periventricular injury with subsequent necrosis and calcifications. Toxoplasmosis leads to more diffuse calcifications, although basal ganglia and periventricular calcifications are common. Calcifications are also[44] seen in parvovirus and chronic meningoencephalitis, and HSV with necrotic foci and hemorrhage. Rubella and herpes are the infections most likely to have extensive cerebral cortical calcifications, while rubella and CMV lead to microcephaly. Subependymal cysts occur in CMV and rubella, but also in Zellweger syndrome, following germinal matrix hemorrhage, and D-2-hydroxyglutaric aciduria. Hydrocephalus is common in toxoplasmosis, syphilis and enterovirus. Congenital myxoviruses, including mumps and parainfluenza, also have a predilection for ependymal cells, leading to congenital hydrocephalus.[98,102]

METABOLIC DISORDERS IN THE NEONATE

Infants with inborn errors of metabolism (IEM) are usually normal at birth. In those disorders that present symptomatically in the neonatal period, the signs often develop in hours to days after birth it is important to maintain a high index of suspicion of IEM in sick neonates since most

of these disorders can be lethal unless diagnosed and treated immediately. Even if the disorder is not treatable, establishing the diagnosis in the index case is crucial for prenatal diagnosis in subsequent pregnancies.[112,113]

The diverse patterns of presentation can be neurological abnormalities as encephalopathy, seizures, and tone abnormalities or disorders of acid-base status, hypoglycaemia, lactic acidosis, dysmorphic features, and cardiac dysfunction.[112]

Neuroimaging forms a part of the investigation of neonates with metabolic disorders. MR imaging is generally considered the optimal technique. Cranial ultrasonogram is very good for detecting most structural brain abnormalities, destructive lesions, and often more subtle tissue abnormality.[8,114,115]

Leijser LM, de Vries LS[8]did their study to primarily investigate the role and range of abnormalities seen on cranial ultrasound examination in a wide range of metabolic disorders.It is a reliable tool for detecting both structural brain abnormalities and more acute changes highly suggestive of metabolic disorders presenting in the neonatal period.

CONGENITAL ABNORMALITIES

A congenital anomaly is any variation, present at birth, of normal anatomic structure. It may be major or minor, isolated or part of a larger

constellation of defects, of clear or uncertain cause. Several genetic and environmental etiologies are well delineated, but the fundamental etiology of nearly half of all birth defects isunknown.[116]

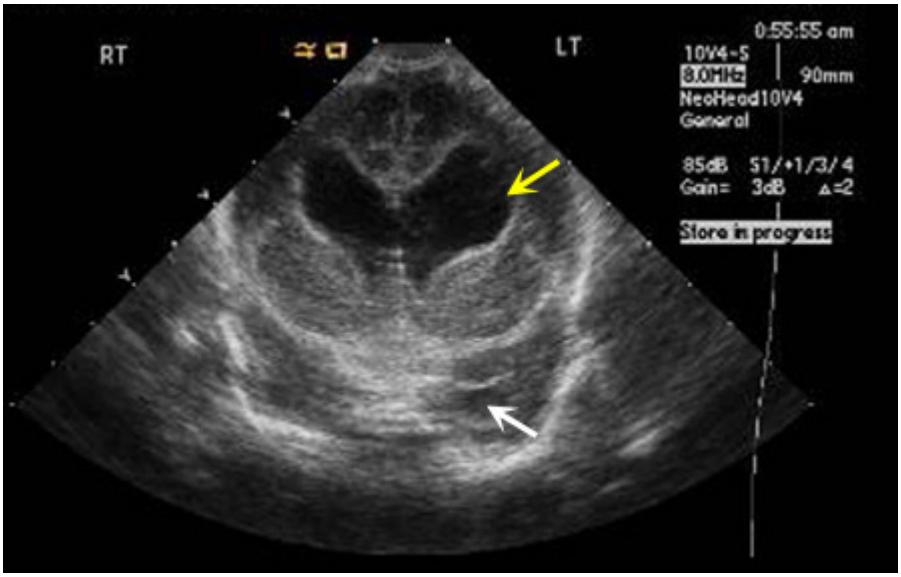
ROLE OF NEUROSONOGRAPHY IN CONGENITAL MALFORMATIONS

Fetal ultrasonographic imaging may detect fetal growth abnormalities or fetal malformations. Serial determination of growth velocity and the head-to-abdominal circumference ratio enhances the ability to detect IUGR. Real-time ultrasonography may identify placental abnormalities and fetal anomalies. Post-natally neurosonogram is done serially to evaluate the progression and course.With the increasing use of routine obstetric US, many congenital brain malformations are recognized prenatally, and the role of neurologic US has changed from allowing a primary diagnosis to confirming a known or suspected abnormality.[116]

HYDROCEPHALUS

At a real level, the most important difference is between progressive and static abnormalities in ventricular volume. This is moderately easy to fix using serial cranial ultrasound examinations. The recent addition of resistive index (RI) measurements adds a physiologic dimension to the anatomic images gathered by ultrasonography.[119,120]Evaluation usually includes neurosurgical

consultation, ultrasound of the head to assess the size of the ventricles, and lumbar puncture to measure CSF pressure. During a head ultrasound, it can be helpful to measure the RI of Anterior cerebral artery.



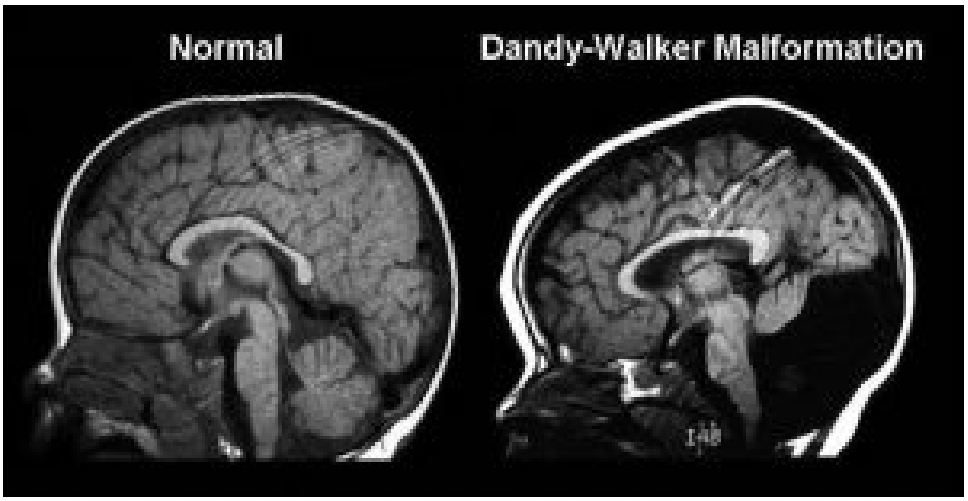
Aqueductal Stenosis

It is a common congenital cause of hydrocephalus, demonstrates severe dilatation of the third and lateral ventricles with no other structural abnormalities present. Mastoid fontanelle imaging affords excellent depiction of brainstem anatomy and can assist in distinguishing between these two anomalies, which have different prognoses and require different treatment.[119]

Dandy-Walker Malformation (DWM)

It classically includes partial or complete vermian agenesis associated with the hypoplastic cerebellar hemispheresand cystic dilatation of fourth ventricle, and the expansion of posterior fossa

associated with high insertion of the tentorium and transverse sinuses. Cerebellar hemispheres are also hypoplastic and abut the petrous ridges. The posterior fossa is occupied by a large cyst corresponding to a dilated fourth ventricle, while the cisterna magna is effaced. Again, mastoid fontanelle imaging of the posterior fossa allows clearer depiction of the cerebellum and visualization of smaller clefts not readily visible through the anterior fontanelle.[119,120]



HEAD TRAUMA IN THE NEONATE

Cephalhematoma is the most frequent cranial injury in a neonate. It occurs in upto 2.5% of live births and is most frequently seen following instrumental delivery, particularly vaccum extraction. Cranial USG can determine the confines of suspected cephalhematoma and can also detect co existent intracranial fluid.[122]

VASCULAR ANOMALIES OF THE NEONATAL BRAIN[125]

It include anatomical, pathophysiological and clinical entities such as cerebral arteriovenousshunts, dural meningeal arteriovenous shunts, and arterial aneurysms.

ANEURYSMAL MALFORMATIONS OF THE VEIN OF GALEN[119,125]

Aneurysmal malformations of vein of Galen can be diagnosed as early as the 28^{th} week of pregnancy by ultrasonography or MRI. They appear as a rounded intracranial mass behind the third ventricle in which flow compatible with an arteriovenous shunt can be detected.

NEED FOR THE STUDY

Neonates born prematurely and sick full-term neonates are at risk of brain injury.Although advances in neonatal intensive care have greatly improved the survival and outcome of these vulnerable patients, brain injury remains a major concern .Early diagnosis is important for prognostication, optimal treatment, and predicting the neurological outcome

MATERIAL AND METHODOLOGY

The study was conducted at Government Mohan Kumaramangalam medical college and hospital, Salem. The duration was from February 2018 to July 2019.

100 critically ill neonates admitted in neonatal intensive care unit were selected as per inclusion criteria and they were subjected imaging on particular day. Follow up imaging was made compulsorily in neonates which showed changes in initial imaging.

INCLUSION CRITERIA :

Term and preterm neonates normal vaginal delivery inborn and outborn

EXCLUSION CRITERIA:

Caeserian section delivered neonates

DESIGN OF THE STUDY:

PROSPECTIVE OBSERVATIONAL CLINICAL STUDY

METHOD OF COLLECTION OF DATA

After getting informed consent from the parents/guardians based on the inclusion criteria with a detailed antenatal history and clinical assessment critically ill neonates were identified.Perinatal events and detailed clinical assessment was done including anthropometric measurements. Vitals were monitored continuosly and neonate selected was made to undergo complete neurological examination. Neurosonogram was performed on selected days . Follow up were done in case of findings revealed any abnormalities in the selected neonates. Neurosonogram findings recorded were correlated with various clinical findings and were followed till recovery.

INSTRUMENTATION

The imaging was performed using a Voluson 630 pro GE machine with multifrequency high volume -TV/TR probe. The images obtained through anterior fontanella and additional sections of thin part of squamous temporal bone in axial plane were comparable to CT & MRI images. Image quality was improved by fine adjusting the preset for transcranial scans. Gray scale Digital recording of the images on the hard disc of the ultrasound machine for purpose of review was done.

To avoid inter and intra observer variation all imaging was done and reviewed by single radiologist later without clinical information. Most of imaging was done as bedside investigation after taking strict aseptic precautions. Covered probe were used after applying the coupling gel in all planes and In axial plane was done in our study through the thin part of squamous temporal bone from both sides. Dynamic real time recording ofimages through fixed anatomical land marks with additional images through abnormal areas were done . Sagittal section: Plane (a) Midline, (b) & (c) 15, 30 degree parasagittal angulation on bothsides. Coronal plane (a) through frontal horns, (b)throughthesylvianfissure,(c)through3rdventricle

(d)throughposteriorfossa,(e)through occipital horn.

Statistical Methods: Descriptive statistical analysis is carried out in the present study. Mean (Min-Max) is used to present results of continuous measurements and number(%) for categorical measurements . Significance is assessed at 5% level of significance. the population should be random, to find significance in study parameters on categorical scale of two or more groups, Chi-square/Kruskalwallis test is used. Sample Size estimation formula used proportion of Unknown population

$n = [(z^2 * p * q) + ME^2] / (ME^2)$

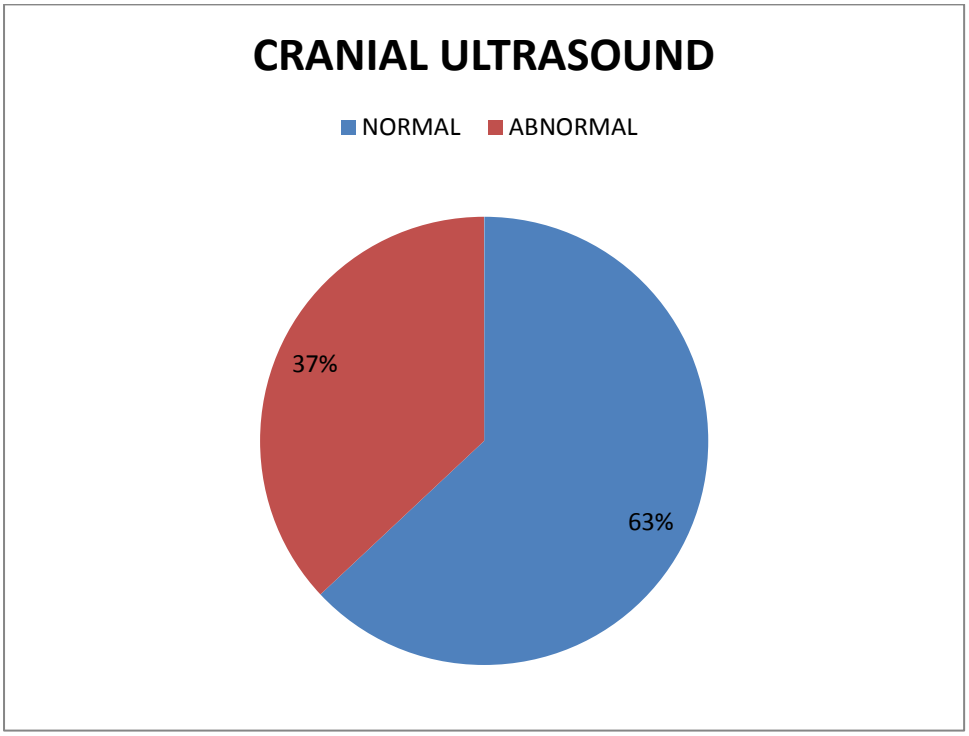
RESULTS

CRANIAL ULTRASOUND

TABLE3: NORMAL AND ABNORMAL OBSERVED

CRANIAL ULTRASOUND	NO OF PATIENTS	PERCENTAGE
NORMAL	63	63%
ABNORMAL	37	37%

FIG 4:NORMAL AND ABNORMAL OBSERVATION

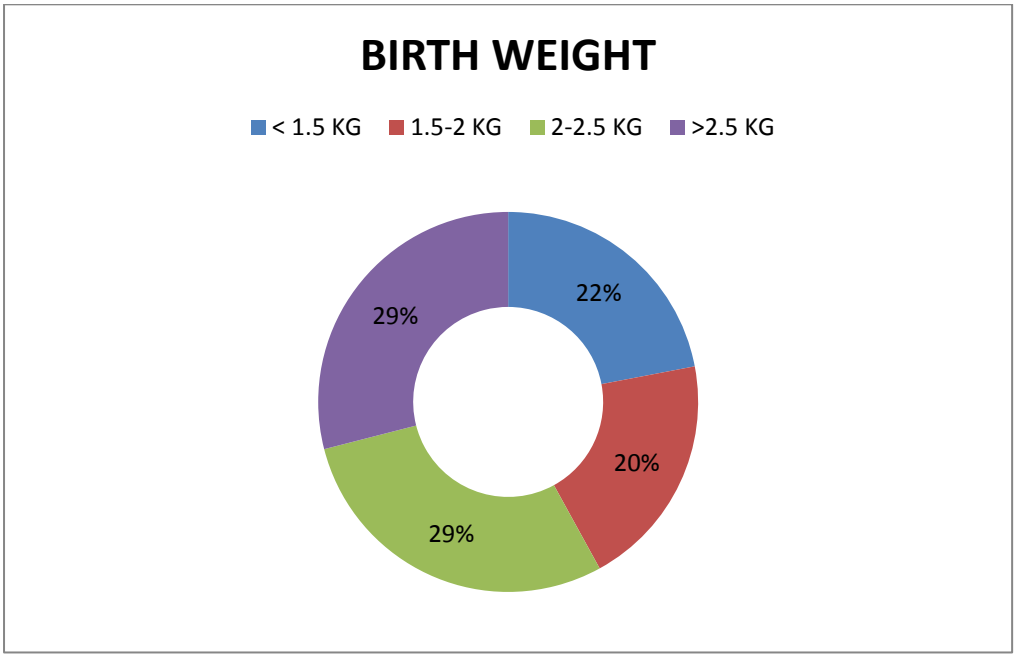


BIRTH WEIGHT

TABLE 4: NEONATES OBSERVED BASED ON BIRTH WEIGHT

BIRTH WEIGHT	NO OF PATIENTS	PERCENTAGE
< 1.5 KG	22	22%
1.5-2 KG	20	20%
2-2.5 KG	29	29%
>2.5 KG	29	29%

FIG 5:**NEONATES OBSERVED BASED ON BIRTH WEIGHT**

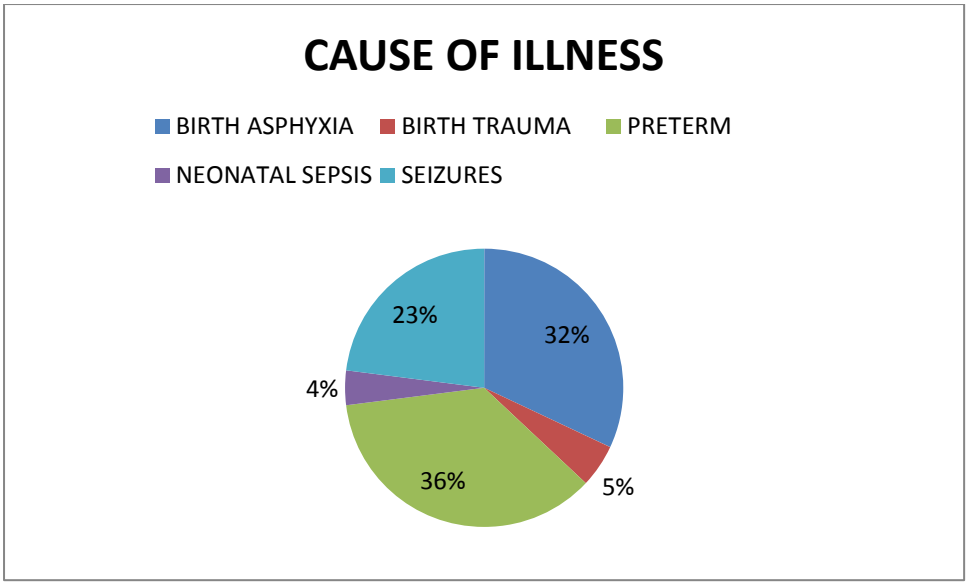


CAUSE OF ILLNESS

TABLE5: NEONATES OBSERVED BASED ON CAUSE OF ILLNESS

CAUSE OF ILLNESS	NO OF PATIENTS	PERCENTAGE
BIRTH ASPHYXIA	32	32%
BIRTH TRAUMA	5	5%
PRETERM	36	36%
NEONATAL SEPSIS	4	4%
SEIZURES	23	23%

FIG:6 CAUSE OF ILLNESS OBSERVED IN THE STUDY

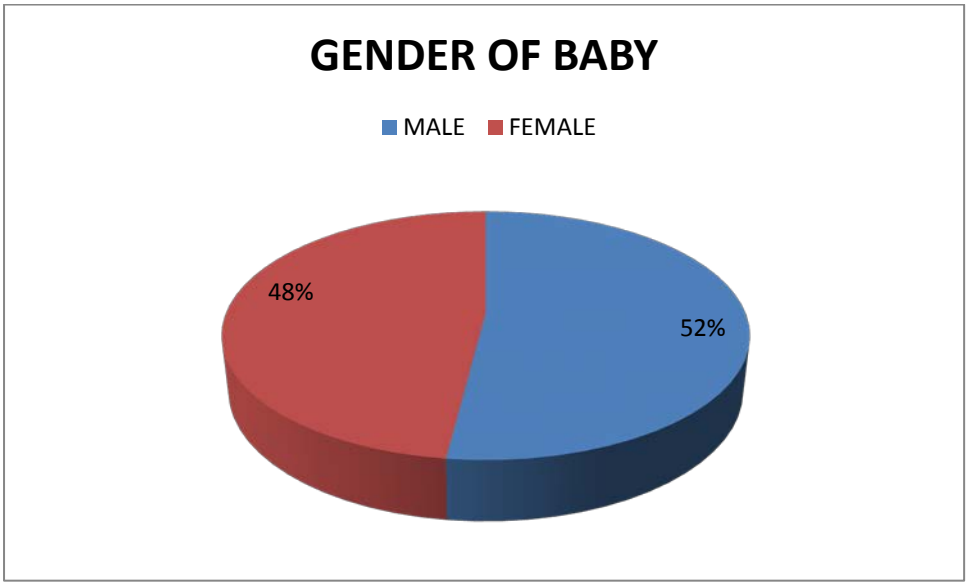


GENDER OF BABY

TABLE 6: NEONATES OBSERVED BASED ON GENDER

GENDER OF BABY	NO OF PATIENTS	PERCENTAGE
MALE	52	63%
FEMALE	48	37%

FIG 7: GENDER DISTRIBUTION

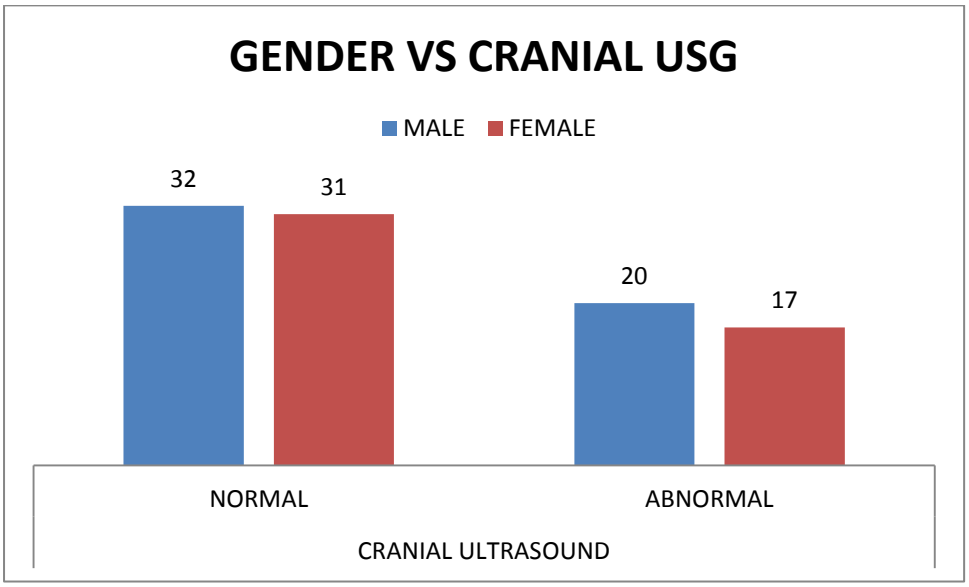


GENDER OF BABY VS CRANIAL USG

TABLE 7: RESULT BASED ON GENDER DISTRIBUTION

GENDER OF BABY	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
MALE	32	20
FEMALE	31	17
CHI SQUARE TEST		
P VALUE - 0.952		
NON SIGNIFICANT		

FIG 8: RESULTS OF GENDER DISTRIBUTION

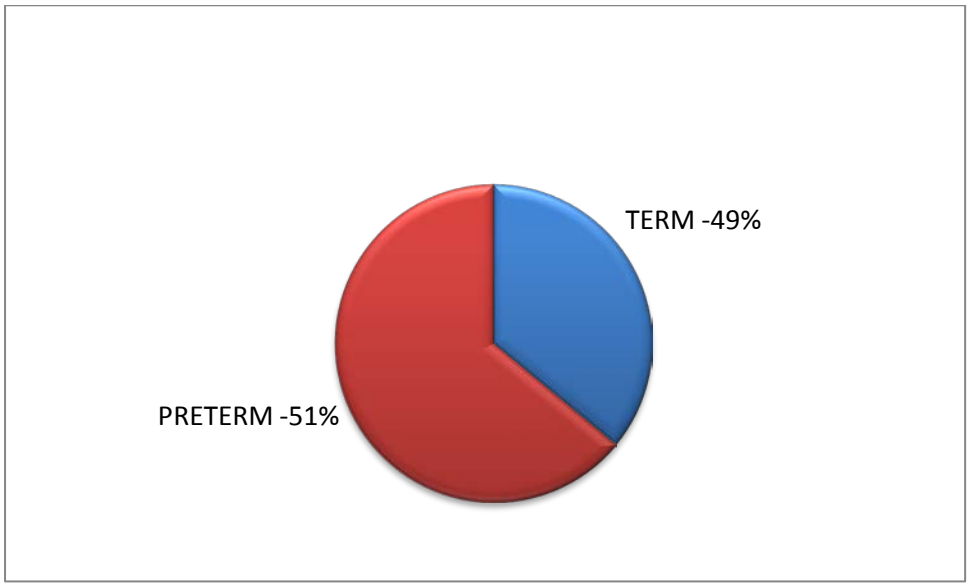


GESTATIONAL AGE

TABLE 8: NEONATES OBSERVED BASED ON GESTATIONAL AGE

GESTATIONAL AGE	NO OF PATIENTS	PERCENTAGE
PRETERM	51	51%
TERM	49	49%

FIG 9: OBSERVATION BASED ON GESTATIONAL AGE

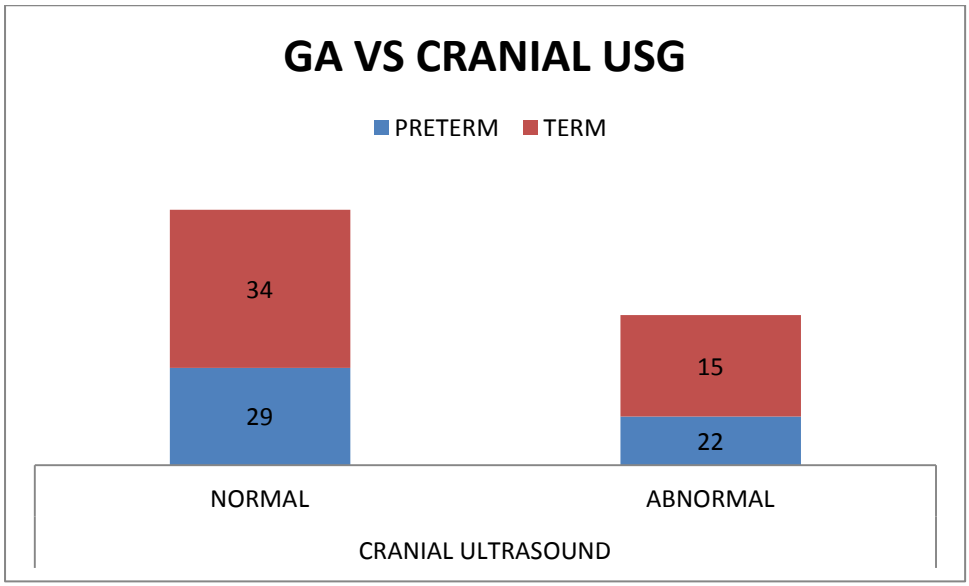


GESTATIONAL AGE VS CRANIAL USG

TABLE:9 RESULTS BASED ON GESTATIONAL AGE

GESTATIONAL AGE	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
PRETERM	29	22
TERM	34	15
CHI SQUARE TEST		
P VALUE - 0.568		
NON SIGNIFICANT		

FIG 10: RESULTS BASED ON GESTATIONAL AGE



GRAVIDA

TABLE 10: PRIMI VS MULTI

GRAVIDA	NO OF PATIENTS	PERCENTAGE
PRIMI	64	64%
MULTI	36	36%

FIG 11: PRIMI VS MULTI

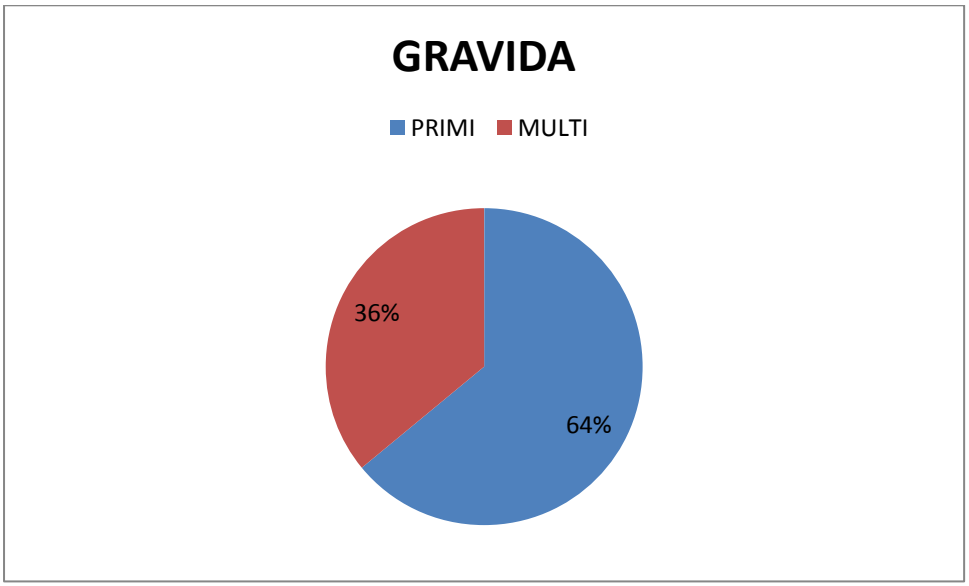
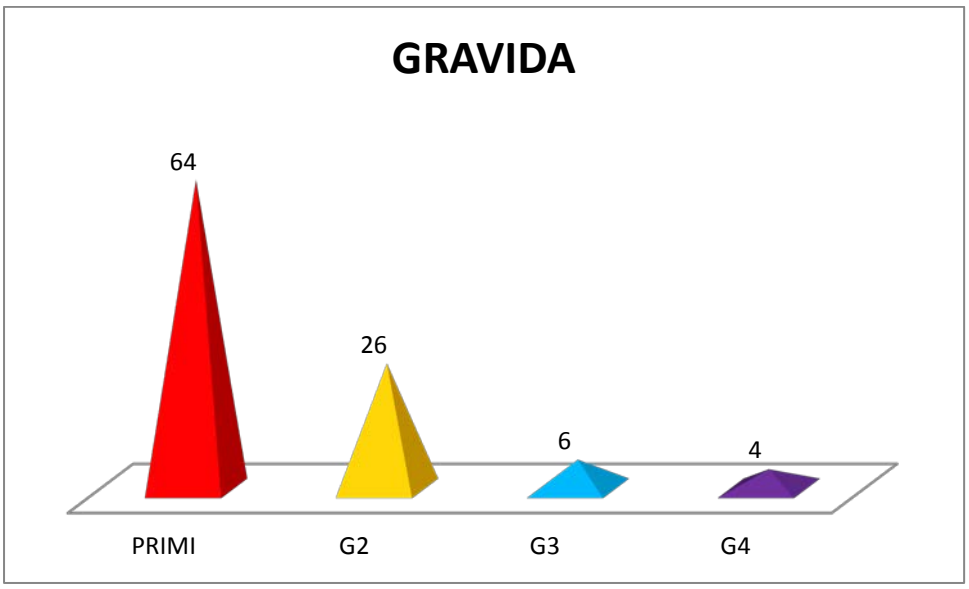


TABLE 11: PERCENTAGE OBERVATIONS OF GRAVIDA

GRAVIDA	NO OF PATIENTS	PERCENTAGE
PRIMI	64	64%
G2	26	26%
G3	6	6%
G4	4	4%

FIG:12 GRAVIDA PERCENTAGES

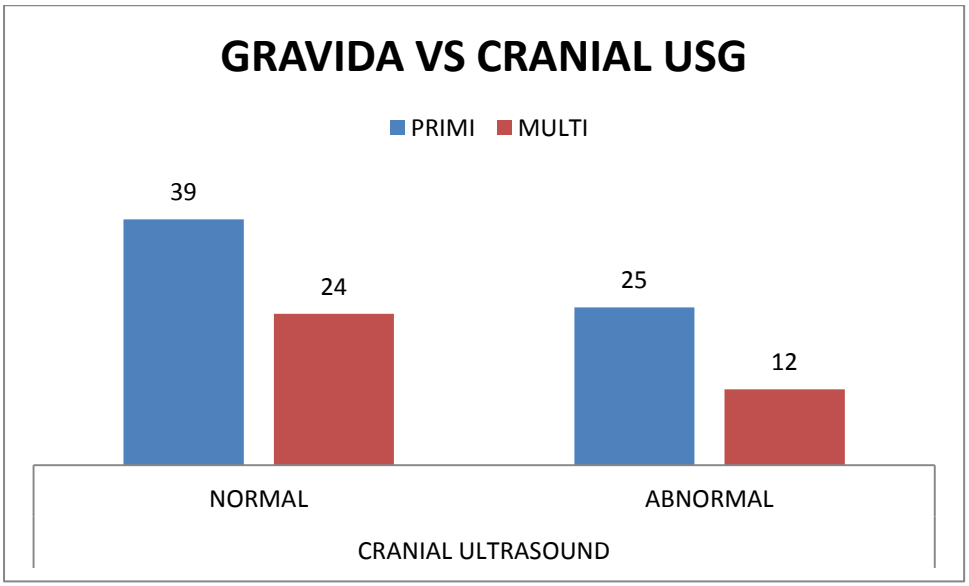


GRAVIDA VS CRANIAL USG

TABLE 12: RESULTS OF PRIMI VS MULTI

GRAVIDA	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
PRIMI	39	25
MULTI	24	12
CHI SQUARE TEST		
P VALUE - 0.952		
NON SIGNIFICANT		

FIG 13: RESULT OF PRIMI VS MULTI

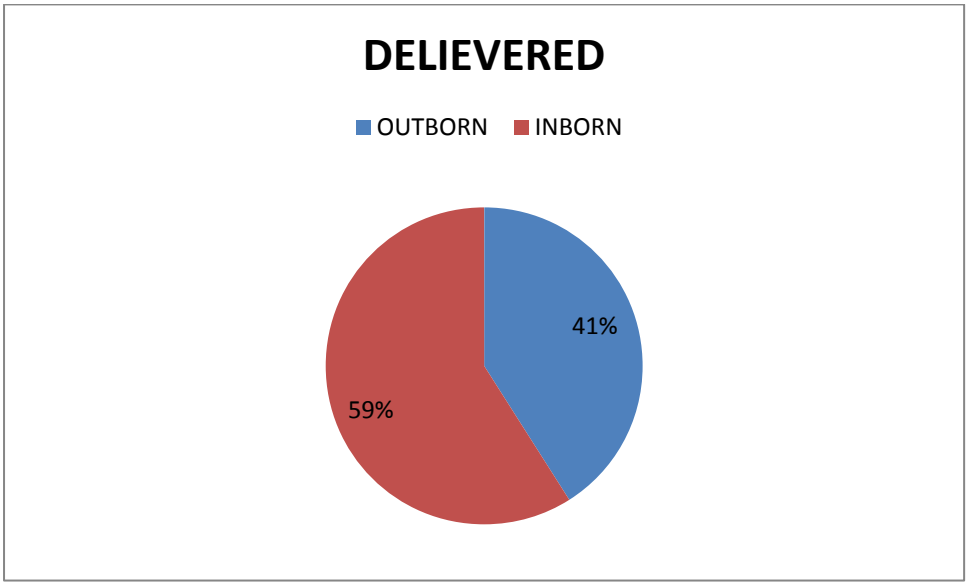


DELIVERED PLACE

TABLE: 13 INBORN VS OUTBORN

DELIVERED	NO OF PATIENTS	PERCENTAGE
OUTBORN	41	41%
INBORN	59	59%

FIG 14: DELIVERED PLACE

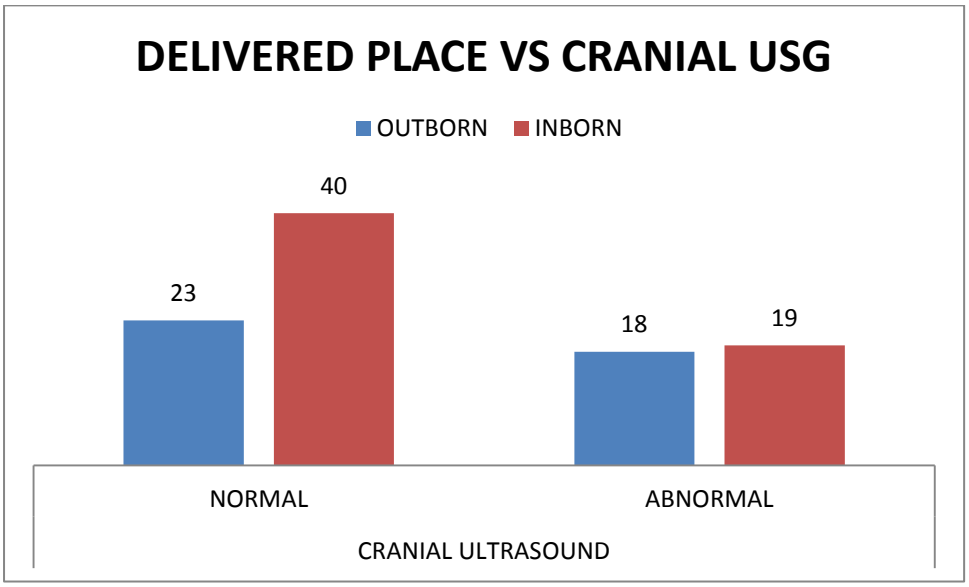


DELIVERED PLACE VS CRANIAL USG

TABLE 14: RESULTS OF INBORN VS OUTBORN

DELIVERED	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
OUTBORN	23	18
INBORN	40	19
CHI SQUARE TEST		
P VALUE - 0.233		
NON SIGNIFICANT		

FIG 15; RESULTS OF DELIVERED PLACE

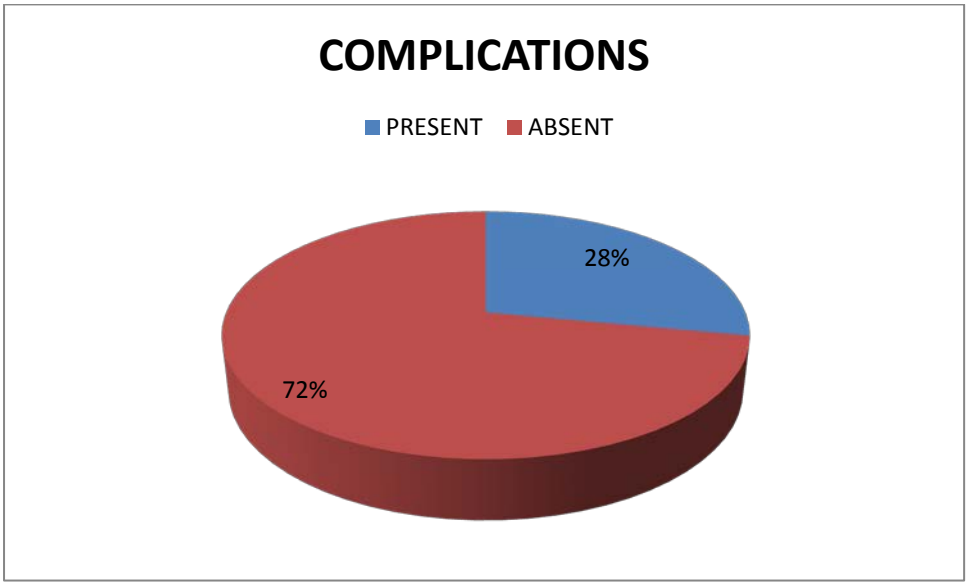


COMPLICATIONS

TABLE 15: NEONATES & COMPLICATIONS

COMPLICATION	NO OF PATIENTS	PERCENTAGE
PRESENT	28	28%
ABSENT	72	72%

FIG 16: COMPLICATIONS



COMPLICATIONS VS CRANIAL USG

TABLE 16: RESULTS SHOWN BASED ON COMPLICATIONS

COMPLICATION	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
PRESENT	6	22
ABSENT	57	15
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		

FIG 17: RESULTS OBSERVED BASED ON COMPLICATIONS

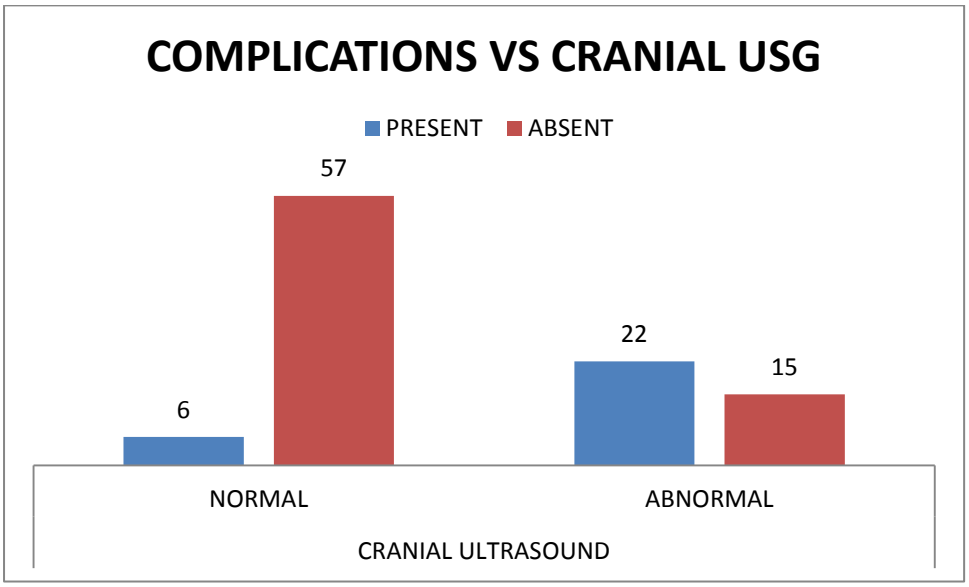


TABLE 17: PATTERN OF COMPLICATIONS OBSERVED

COMPLICATIONS	NO OF PATIENTS	PERCENTAGE
HIE	24	24%
SEPSIS	4	4%
ABSENT	72	72%

FIG 18: PATTERN OF COMPLICATIONS

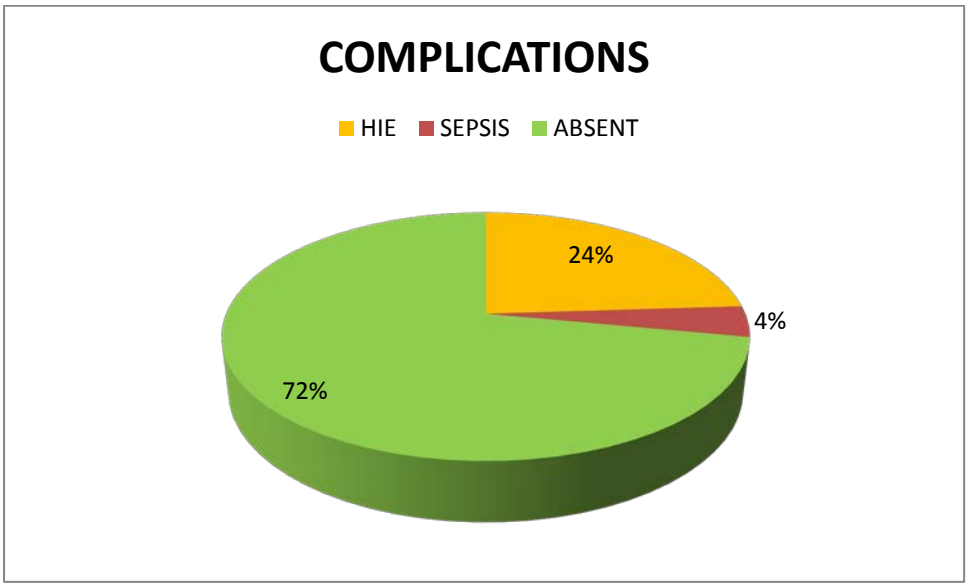
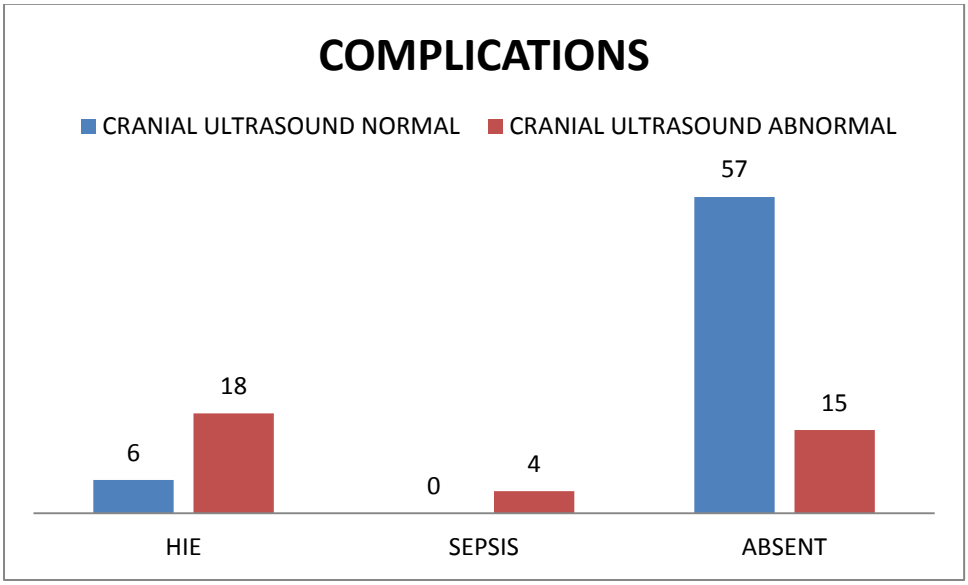


TABLE 18: RESULTS BASED ON COMPLICATIONS

COMPLICATIONS	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
HIE	6	18
SEPSIS	0	4
ABSENT	57	15
KRUSKAL WALLIS TEST		
P VALUE - 0.001		
SIGNIFICANT		

FIG 19 : RESULTS BASED ON COMPLICATIONS

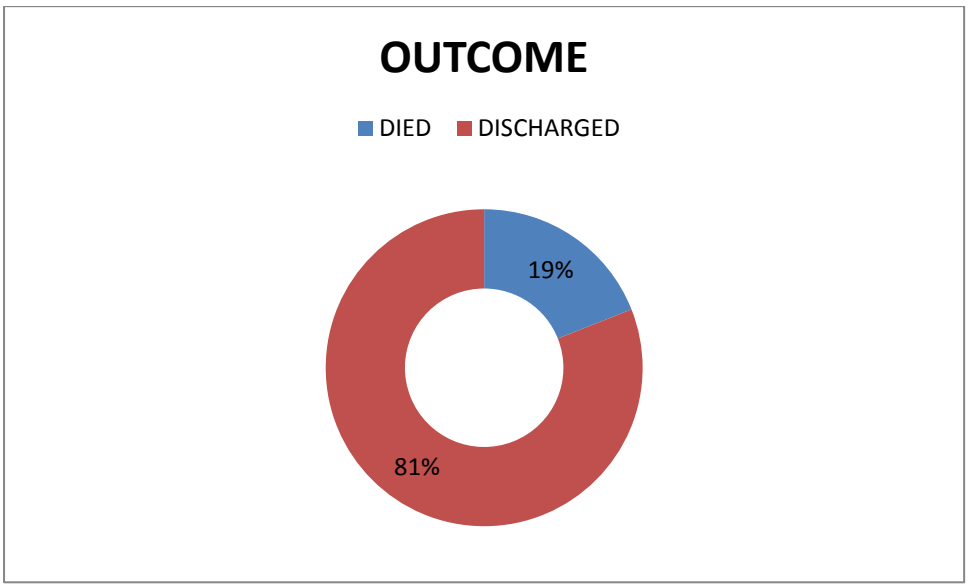


OUTCOME

TABLE19: NEONATES OBSERVED BASED ON OUTCOME

OUTCOME	NO OF PATIENTS	PERCENTAGE
DIED	19	19%
DISCHARGED	81	81%

FIG 20: NEONATES OBSERVED BASED ON OUTCOME

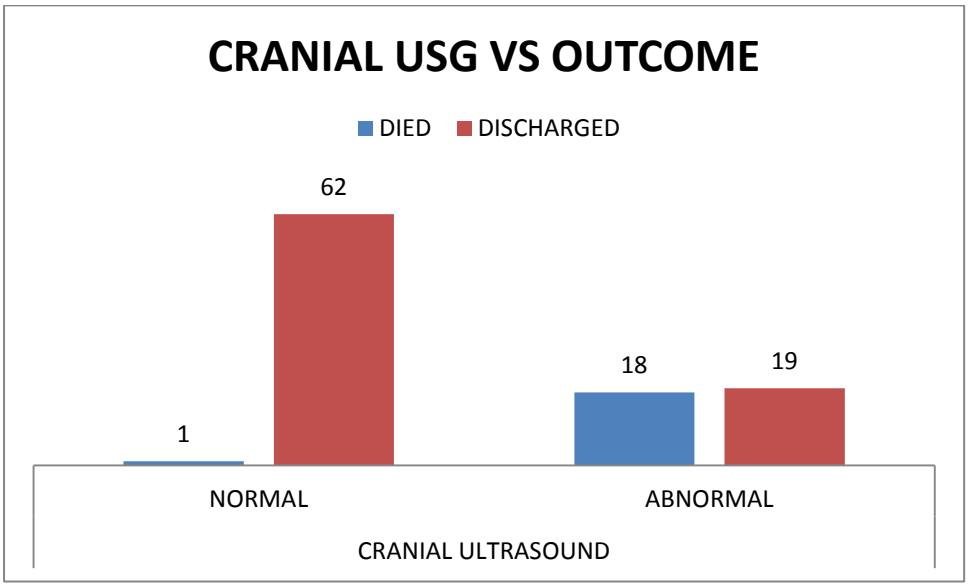


CRANIAL USG VS OUTCOME

TABLE20:RESULTS BASED ON OUTCOME

OUTCOME	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
DIED	1	18
DISCHARGED	62	19
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		

FIG 21: RESULT BASED ON NEONATAL OUTCOME

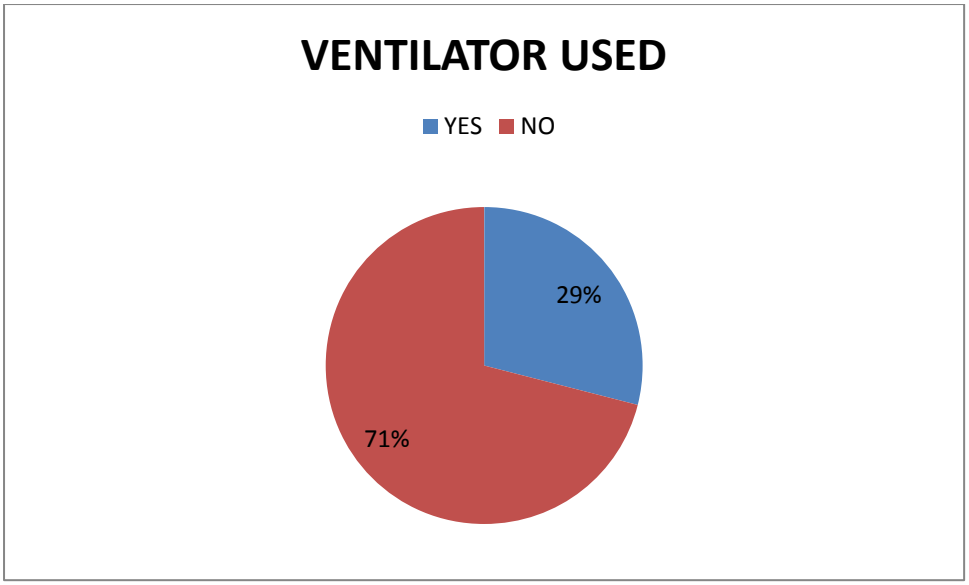


VENTILATOR USED

TABLE 21: NEONATES REQUIRING MECHANICAL VENTILATOR

VENTILATOR USED	NO OF PATIENTS	PERCENTAGE
YES	29	29%
NO	71	71%

FIG 22 : NEONATES REOUIRING MECHANICAL VENTILATION

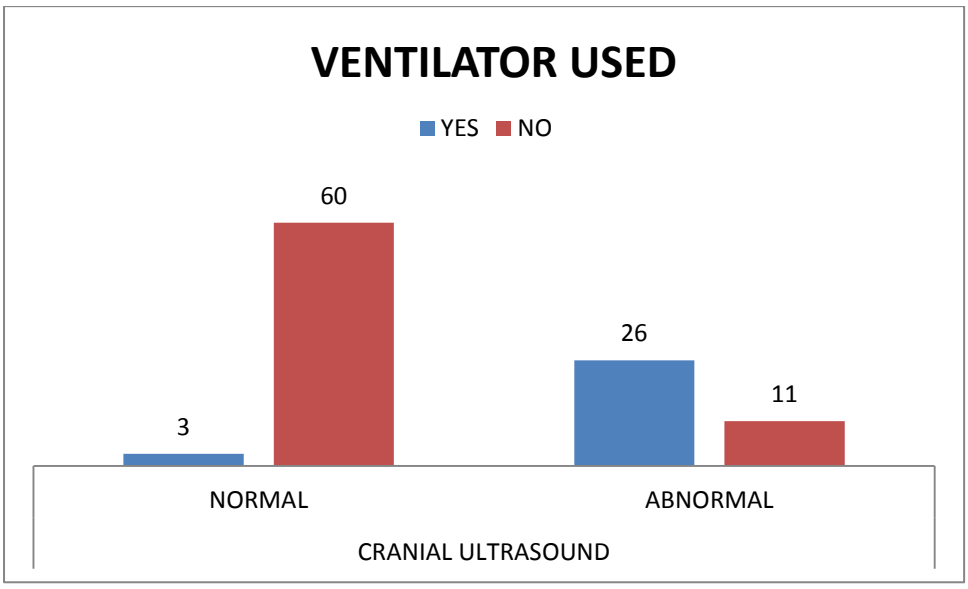


CRANIAL USG VS USAGE OF VENTILATOR

TABLE 22: RESULTS OBTAINED BASED ON MECHANICAL VENTILATOR

VENTILATOR USED	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
YES	3	26
NO	60	11
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		

FIG 23: RESULTS BASED ON MECHANICAL VENTILATOR REQUIREMENT

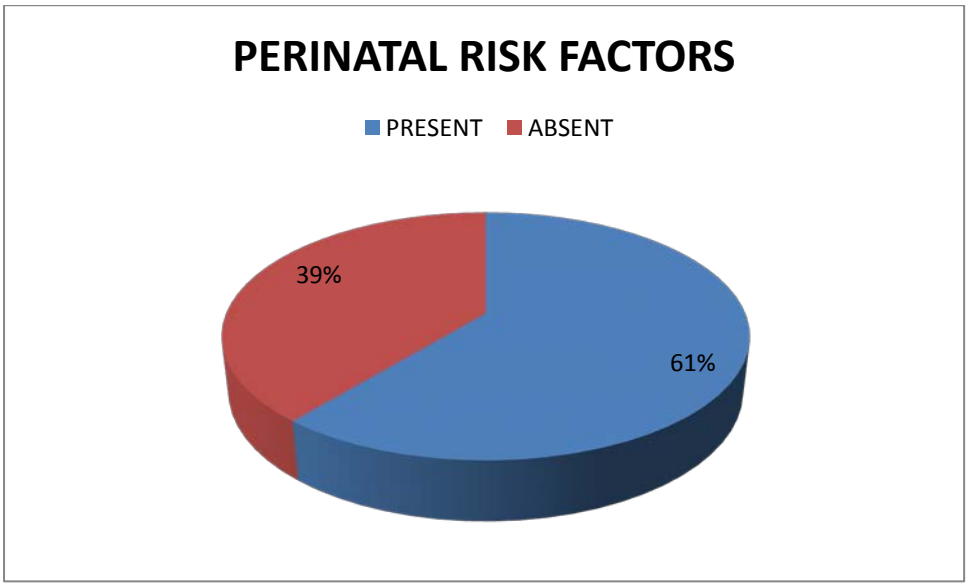


PERINATAL RISK FACTORS

TABLE 23: PRESENCE OF PERINATAL RISK FACTORS

PERINATAL RISK FACTOR	NO OF PATIENTS	PERCENTAGE
PRESENT	61	61%
ABSENT	39	39%

FIG 24: PERINATAL RISK FACTORS OBSERVED

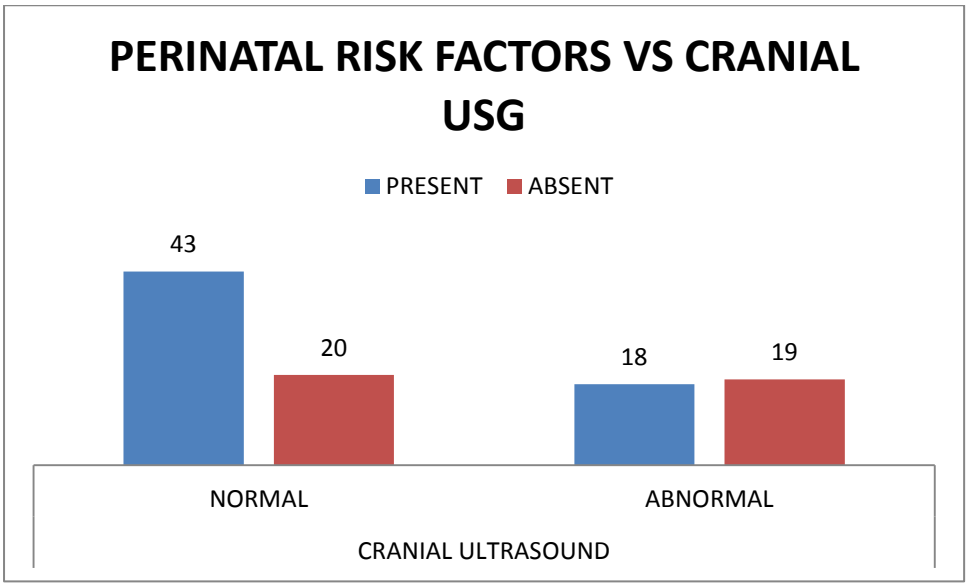


CRANIAL USG VS RISK FACTORS

TABLE 24: RESULTS BASED ON PERINATAL RISK FACTORS

PERINATAL RISK FACTOR	CRANIAL ULTRASOUND	
	NORMAL	ABNORMAL
PRESENT	43	18
ABSENT	20	19
CHI SQUARE TEST		
P VALUE - 0.005		
SIGNIFICANT		

FIG 25: RESULTS BASED ON PERINATAL RISK FACTORS

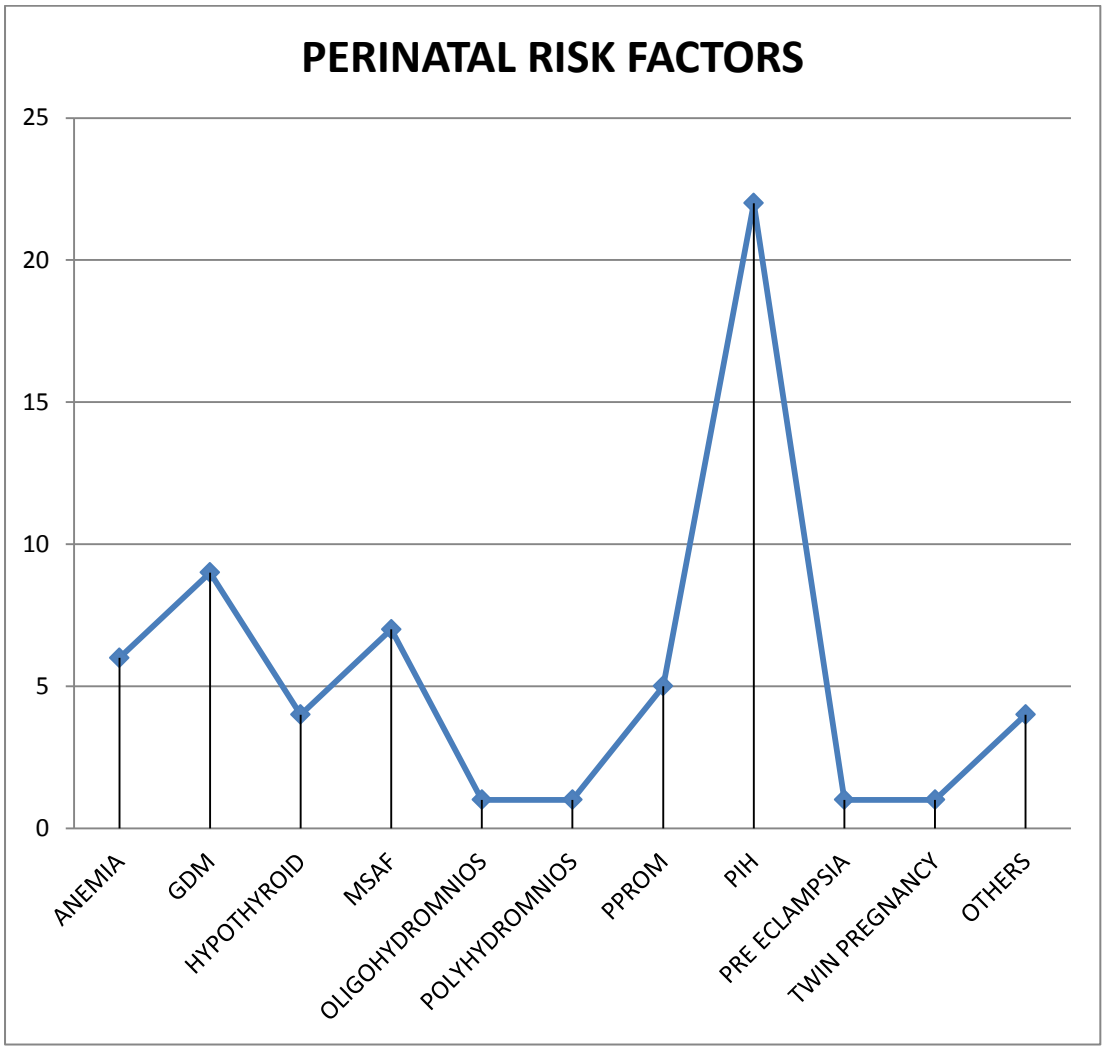


PERINATAL RISK FACTORS

TABLE 25: PATTERN OF PERINATAL RISK FACTORS OBSERVED

PERINATAL RISK FACTORS	NO OF PATIENTS	PERCENTAGE
ANEMIA	6	6%
GDM	9	9%
HYPOTHYROID	4	4%
MSAF	7	7%
OLIGOHYDROMNIOS	1	1%
POLYHYDROMNIOS	1	1%
PPROM	5	5%
PIH	22	22%
PRE ECLAMPSIA	1	1%
TWIN PREGNANCY	1	1%
OTHERS	4	4%

FIG 26: PATTERN OF PERINATAL RISK FACTORS OBSERVED

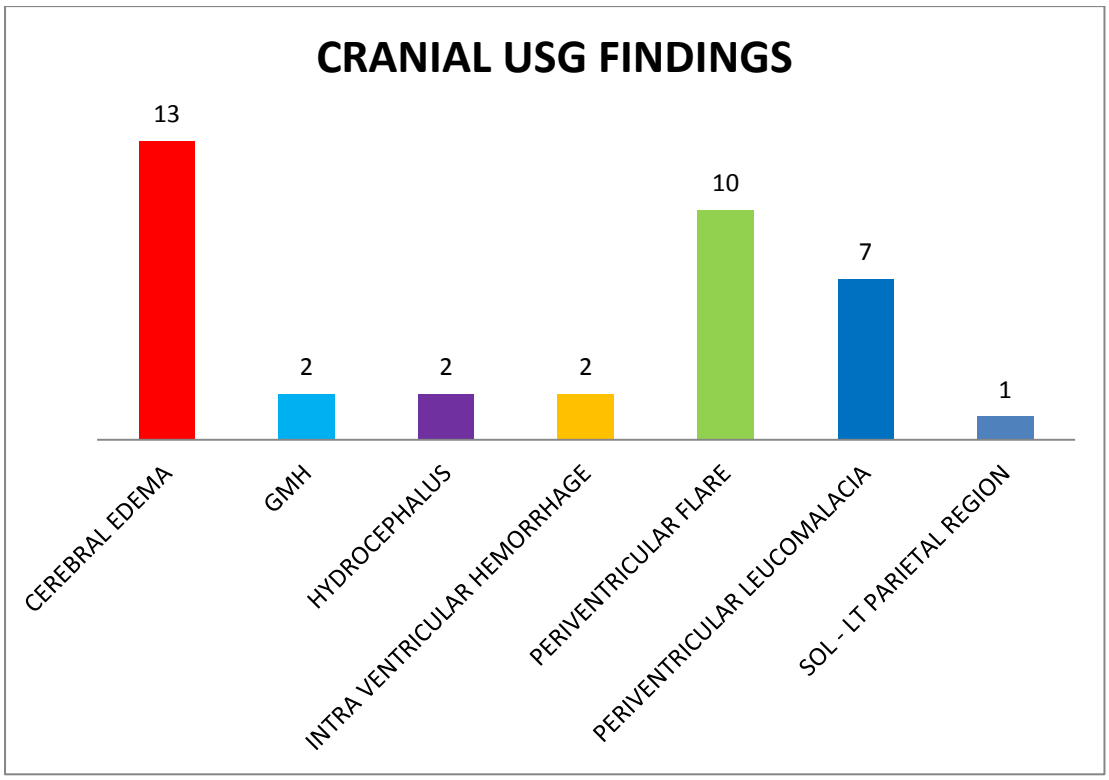


CRANIAL USG FINDINGS

TABLE 26: CRANIAL USG FINDINGS OBSERVED IN THE STUDY

CRANIAL USG FINDINGS	**NO OF PATIENTS**	**PERCENTAGE**
CEREBRAL EDEMA	13	35%
GMH	2	5%
HYDROCEPHALUS	2	5%
INTRA VENTRICULAR HEMORRHAGE	2	5%
PERIVENTRICULAR FLARE	10	27%
PERIVENTRICULAR LEUCOMALACIA	7	20%
SOL - LT PARIETAL REGION	1	3%

FIG 27 : NEUROSONGRAM FINDINGS OBSERVED IN THE STUDY



DISCUSSION

In modern day neonatology cranial sonogram is used as newer diagnostic tool to know anatomical and pathological changes in neonatal brain. Generally all newborns have many sutures and fontanelles which will be open, and can be used as acoustic windows for imaging brain.[1] Inspite of birth weight, gestational age or any other, newborns associated with fetal, maternal or placental anomalies who had an otherwise complicated pregnancy, especially within the first 28 days of birth is identified as critically ill neonates.2] Neurosonogram plays an important role in assessing neurological outcome of these ill neonates.

This study was conducted at Government Mohan Kumramangalam medical college and hospital, Salem. The duration of study was from February 2018*July 2019.One hundred critically ill neonates admitted in neonatal intensive care unit were selected as per inclusion criteria and were subjected for imaging on selected days.Neurosonogram were done to follow up sequelae if any abnormalities were recorded in the primary .

Daneman A, EpelmanM et al[126]proved that neurosonogram is an extremely useful technique for imaging of full-term neonatal brain. Cowan et al[42]have described cranial ultrasound in particular is an useful device in early period, when the newborn is critically ill and immobile requiring continuous supportive measure Our study aims to prove the same.

Incidence of abnormalities in critically ill neonates is 37% in our study. Of all neonates who underwent imaging 52% male and 48% female. There were 51% preterm and 49% term neonates in the present study. Lilly Dubowitzet al[30]study showed an incidence of 20%abnormalities in normal neonates. Ayala Gover et al[127] study showed an incidence of 11.2% abnormalities on healthy term neonates.. In our study 64 mother were primi and rest were multi,there was no signifcicant relation between gravida and abnormal neurosonogram findings, 25 neonate of primi mother had abnormal cranial usg whereas among 36 multigravida who delivered 12 had abnormal cranial USG.

In our study around 41 neonates where delivered outside and refered here for further management, even the time of transition also has effect on the outcome of disease.

Badrawy N et al[128] showed 37% preterms had abnormal neurosonogram findings in their study of which 64% male and 36% female neonates. Among these, 9% were < 30 weeks, 33% between 30-33weeks and 58% were between 34 to 37weeks.

Eugenio Mercuri, Lilly Dubowitz et al[30]reported ischemic lesions , like periventricular and thalamic densities,cerebral edema on cranial ultrasound. BadrawyN. et al[128]reported that sub ependymal intraventricular hemorrhage (SE-IVH) was seen in 14%, brain edema in

9% and 4% hypoxic ischemic changes, 3.5% post hemorrhagic hydrocephalus(PHH) as complications. In present study, neurosonogram showed , 37% of neonates found to have abnormal findings. 2 of them of these had evidence of intracranial bleed, 10 cases had periventricular flare, 13 cases had cerebral edema and 7 cases had periventricular leucomalacia.

Badrawy Net al[128]reported congenital hydrocephalus to be present in 6% among all neonates screened by them. In the present study, two neonate had congenital hydrocephalus with aqueductal stenosis.

Soni JP et al[28]study suggested that the neurosonogram is sensitive and specific for detection of all types of ICH (SEH, IVH, PVL). 111 high risk neonates were subjected cranial ultrasound, one fourth of these neonates developed intracranial hemorrhage(ICH) within 120 hours of birth. Similarly in one another study Trounce et al[130]reported that hemorrhage was seen within first 7 days of life using cranial ultrasound in 78% neonates and 2nd week in 15% neonates. Similar findings have also been reported by Beverley et al[135]and by Murton et al[136], who detected germinal matrix hemorrhage in some infants immediately after delivery using cranial ultrasound.

In the present study 43% of neonates with perinatal risk factor have evidence of abnormal cranial ultrasound findings all of which were picked up between 24 to 72 hours of life. Elia FM et al[137], study

suggested that Neurosonogram predicts the presence of GMH, and other haemorrhages accurately.

Jeffrey M. Perlmanl[85]study showedthat 50% of neonates weighing <1500 g had some abnormality in the initial Neurosonogram. Severe IVH was observed in approximately 11% of the neonates weighing <1000 g and in 5% of those between 1000-g to 1250-g BW showed abnormalities. 5% of the neonates weighing <1000-g and in 1% of those between 1250 - 1500 g showed cystic changes. In the present study, 12 of 22 neonates weighing less than 1500g, 9 of 20 neonates weighing between 1500 and 2000g, 9 of 29 weighing between 2000 and 2500g and 7 of 29 weighing more than 2500gms exhibited some abnormality on Neurosonogram. Joseph J. Volpe et all[141]in their study detected 56% neonates had abnormal cranial US findings of which 60.7% infants had transient echo densities and 33.9% had prolonged echo densities suggesting white matter injury. They concluded that neonatal neurosonogram of the VLBW infant demonstrates high reliability in the detection of cystic WM.

Arti Maria et al[142] in their study reported that 36.2% of enrolled very low birth weight neonates developed various forms of PVL. In their study, about 50% of ultrasound was normal at discharge and sequelae such as cerebral atrophy and ventriculomegaly had appeared in few, the rest of lesions being either flares or cysts of PVL. They established

Neurosonogram remains an important bedside diagnostic tool forPVL.

Leijser LM, de Vries LS, Rutherford MA et al[8]proved that neonatal neurosonogram is a reliable tool for the early bedside detection of abnormality highly suggestive of a metabolic disorder. In their study, all the cerebral lesions and major structural brain abnormalities characteristic of different metabolic disorders were identified on Neurosonogram. In the present study, none of the neonates studied had neurosonogram findings suggestive of metabolic disorder.

Boo N, Chandran V, ZulfiqarM et al[145]reported diffuse increase in echogenicity of the cerebral parenchyma was significantly more common in term infants with encephalopathy than in controls (39% versus 1%).

In the present study of the 28 % of neonates who developed complications abnormal findings on neurosonogram was seen in 22 patients among which 18 had HIE and 4 had sepsis.

Hannah C. Glass, Sonia L. Bonifacioet al[146] study reported that in about 3.8% preterm neonates clinical seizures were present. Neurosonogram identified abnormalities like IVH and PVHI in their study. In this study, seizures was seen in 23% of babies whereas birth asphyxia was seen in 32 neonates in our study. Michael O'Shea T et al[149]studied prenatal factors like preeclampsia related to cranial ultrasound findings, neurodevelopmental outcome and incidence of cerebral palsy.

In the present study, those high risk neonates with evidence of neonatal sepsis 83% had normal and 17% abnormal cranial ultrasound findings .Three studies evaluated Correlation of US findings PVL with neuropathologic among critically ill neonates. All these studies showed 100% correlation between cranial ultrasound findings and their data. Ultrasound is also particularly useful in detecting congenital malformations such as cystic lesions, corpus callosal agenesis and aneurysm of vein of Galen (color Doppler).

In the present study, most of high risk neonates who had neonatal sepsis had abnormal neurosonogram findings.This findings are similar to study done by Nagaraj et al.

Canadian Paediatric Society in their Statement in (2001)[158]concluded that cranial ultrasound examinations are of important value in determining the need for routine screening in NICU.

In the present study, of the 37% neonates having abnormal Neurosonogram, 19 neonates recovered completely during their stay in NICU and while rest died . This show a positive correltion between outcome and neurosonogram findings which was also statistically significant.

Similarly ventilator was also required around in 29 neonates in our study among whom 26 had abnormal ultrasound findings which shows the poor health state of neonates with abnormalities.

CONCLUSION:

In India neonatal care is progressing at an rapid phase to provide care both at the community and in tertiary care units. The survival of the newborn, a newer concept has provided a way to the significance of 'intact survival' of critically ill neonates, leading to initiation of new strategies to identify neuro abnormalities.

Cranial sonogram is the choice of device for screening the neonatal brain. Though ultrasound machines are available in the hospitals, the imaging quality of cranial ultrsound in Indian NICU's is still of low standards.This study highlights role and diagnostic efficiency of neurosonogram in critically ill neonates in neonatal intensive care unit.

Nowadays its use in screening for preterm neonates plays crucial role in assessing their neurodevelopmental outcome. Neurosonogram in identifying brain damage and its evolution is highly efficacious on regular follow up which provides information that guides clinical decisions and prognosis. Thus neurosonogram plays potential role in preventive, protective, and rehabilitative strategies in the management of critically ill neonates.

This study concludes neurosonogram as choice of investigation modality in NICU which effectively documents neuroabnormalities.

SUMMARY

- Neurosonogram abnormalities in critically ill neonates is 37% in the present study.
- Of all neonates included 52% are male and 48% are female .statistical analysis suggested no significant correlation in the incidence of abnormal cranial ultrasound findings regarding gender distribution in critically ill neonates in the present study.
- Of all neonates included 51% were preterm and 49% were term in the present study. There was no statistical significance between gestational age and neurosongram findings in the present study.
- In this study, 12/20 neonates weighing < 1500g, 9/20 weighing between 1.5kg- 2.0kg, 9/29 weighing between 2.0kg – 2.5kg and 7/29 weighing > 2.5kgms showed some abnormalities on Neurosonogram
- 51% neonates were born via normal labour and 49% via assisted normal labour for various reasons.
- Based on the cause of illness this study showed 32% of perinatal asphyxia, 4% of birth trauma, 4% of neonatal sepsis, 23% of neonatal seizures , and of this 36% were preterm
- In this study, 37% of neonates had abnormal findings. Overall 13% had cerebral edema, 10%showed periventricular flare, 7%had periventricular leucomalacia, 2% had germinal matrix hemorrhage, 2% had intraventricular hemorrhage, 2% showed hydrocephalus and 1%

showed SOL in left parietal region.

- Neurosonogram findings of neonates with sepsis, birth trauma, seizures and prematurity was not statistically significant.
- Of the all neonates with term gestation having abnormal findings on Neurosonogram, 60% had cerebral edema, 35% showed periventricular flare and 15% had periventricular leucomalacia
- In general statistical analysissuggested significant correlation between findings on Neurosonogram and clinical outcome . Almost all neonates died had cranial ultrasound abnormality

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ANNEXURE

PROFOMA

BABY DETAILS :

- Name (B/O)
- Age/Sex
- IP. Number
- Ward/Unit
- Date of study

Particulars:

- Ante natal h/o
- Mode of delivery
- Gravida
- Gestational age
- Apgar score 1 and 5 min
- Vitals
- Usg cranium findings:

MASTER CHART

S.NO	NAME	AGE	SEX	AN &N H/O	GRAVIDA	GA	APGAR	CRANIAL USG FINDINGS	POD	COMP	OUTCOME	MV	BW
1	B/OPOUNAMMAL	2/365	FCH	ANEMIA	PRIMI	TERM	7/10, 8/10	NS	OB	NIL	DIS	NO	2.6
2	B/OESWARI	3/365	FCH	MSAF	PRIMI	TERM	7/10,8/10	NS	OB	NIL	DIS	NO	2.7
3	B/O CHANDRA	3/365	FCH	PIH	PRIMI	TERM	6/10,7/10	NS	IB	NIL	DIS	NO	2.6
4	B/O SARSWATHI	3/365	MCH	PIH	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.8
5	B/O PDARSHINI	4/365	FCH	GDM	PRIMI	TERM	7/10,8/10	NS	OB	NIL	DIS	NO	2.9
6	B/O REVATHI	2/365	FCH	PIH	PRIMI	TERM	5/10,8/10	NS	IB	HIE	DIS	NO	3.2
7	B/O SHANOOR	3/365	MCH	GDM	PRIMI	TERM	7/10,8/10	NS	IB	NIL	DIS	NO	3.1
8	B/O SOUMIYA	3/365	FCH	NORMAL	PRIMI	TERM	6/10, 7/10	NS	IB	NIL	DIS	NO	2.9
9	B/O GOPIKA	4/365	MCH	MSAF	PRIMI	TERM	5/10,7/10	NS	OB	HIE	DEAD	YES	3.6
10	B/OSANGEETHA	4/365	FCH	NORMAL	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.8
11	B/O AMSAVALLI	3/365	FCH	NORMAL	PRIMI	TERM	7/10,8/10	NS	OB	NIL	DIS	NO	2.7
12	B/O LATHA	3/365	MCH	OBESITY	PRIMI	TERM	6/10,7/10	NS	IB	NIL	DIS	NO	2.8
13	B/O KANAGA	4/365	MCH	NORMAL	G2	TERM	6/10,7/10	NS	IB	NIL	DIS	NO	3.2
14	B/O KANAGA	6/365	FCH	PIH	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	3
15	B/O MEENA	3/365	FCH	MSAF	G2	TERM	6/10,8/10	NS	OB	NIL	DIS	NO	2.9
16	B/O DIVYA	2/365	FCH	GDM	G2	TERM	7/10,8/10	NS	IB	NIL	DIS	NO	3.1
17	B/O SATHIYA	3/365	MCH	HYPOTHYROID	PRIMI	TERM	6/10,7/10	NS	OB	HIE	DIS	NO	2.7
18	B/O BANUPRIYA	4/365	FCH	MSAF	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.7
19	B/O SHYAMALA	4/365	FCH	GDM	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.8
20	B/O ABINAYA	3/365	FCH	NORMAL	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.8
21	B/O GOKILA	2/365	MCH	PIH	PRIMI	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.9
22	B/O LAVANYA	3/365	FCH	MSAF	G3	TERM	5/10,8/10	NS	OB	HIE	DIS	YES	2.8
23	B/O KAVITHA	4/365	MCH	PIH	G2	TERM	7/10,8/10	NS	IB	NIL	DIS	NO	2.7

24	B/O KRISHNAVENI	3/365	MCH	MSAF	PRIMI	TERM	6/10,7/10	NS	IB	NIL	DIS	NO	3.2
25	B/O THANGAYEE	3/365	MCH	NORMAL	G2	TERM	7/10, 8/10	NS	IB	NIL	DIS	NO	2.6
26	B/O UMA	4/365	FCH	NORMAL	G3	TERM	7/10,8/10	NS	IB	NIL	DIS	NO	2.6
27	B/O MYTHILI	2/365	FCH	NORMAL	PRIMI	TERM	6/10,7/10	NS	IB	HIE	DIS	NO	2.7
28	B/O VAITHEGI	2/365	MCH	NORMAL	G2	TERM	7/10,8/10	NS	OB	NIL	DIS	NO	2.8
29	B/O LAKSHMI	4/365	FCH	PIH	PRIMI	TERM	6/10,7/10	NS	IB	NIL	DIS	NO	2.2
30	B/O SUNDHARI	12/365	MCH	NORMAL	G2	TERM	7/10, 8/10	NS	OB	NIL	DIS	NO	2.3
31	B/O GEETHA	4/365	FCH	NORMAL	G2	TERM	7/10,8/10	NS	OB	NIL	DIS	NO	2.4
32	B/O REETAMARY	5/365	FCH	ANEMIA	G2	TERM	6/10,7/10	NS	OB	NIL	DIS	NO	2.3
33	B/O PARAMESWARI	3/365	MCH	NORMAL	G4	TERM	7/10,8/10	NS	IB	NIL	DIS	NO	2.1
34	B/O RAJAMATHI	3/365	MCH	ELDERLY	PRIMI	TERM	7/10,8/10	NS	IB	NIL	DIS	NO	2.3
35	B/O CHANDRA KUMAR	4/365	MCH	NORMAL	G2	TERM	6/10,8/10	CEREBRAL EDEMA	OB	NIL	DIS	NO	2.2
36	B/O KAVITHA HARI	4/365	FCH	PIH	PRIMI	TERM	5/10, 7/10	CEREBRAL EDEMA	IB	HIE	DIS	YES	2.3
37	B/O SARASWATHI RAJAN	8/365	MCH	NORMAL	G2	TERM	8/10,9/10	CEREBRAL EDEMA	OB	SEPSIS	DEAD	YES	2.1
38	B/O PRIYA	4/365	FCH	HYPOTHYROID	G2	TERM	6/10, 8/10	CEREBRAL EDEMA	IB	NIL	DIS	NO	2.3
39	B/O REVATHI GOVINDAN	3/365	FCH	ANEMIA	PRIMI	TERM	6/10, 8/10	PVF	OB	NIL	DIS	NO	2.1
40	B/O GAYATHRI	3/365	MCH	NORMAL	PRIMI	TERM	6/10,8/10	PVF	OB	NIL	DIS	NO	2.3
41	B/O RAMYA	4/365	FCH	MSAF	PRIMI	TERM	6/10,7/10	PVL	OB	HIE	DIS	NO	2.2
42	B/O SHEENA	4/365	MCH	NORMAL	G2	TERM	5/10,7/10	CEREBRAL EDEMA	OB	HIE	DEAD	YES	2.1
43	B/O SUMATHI	4/365	MCH	AN SCAN HUN	PRIMI	TERM	5/10,7/10	CEREBRAL EDEMA	IB	HIE	DEAD	YES	2.3
44	B/O KARTHIKA	3/365	FCH	NORMAL	PRIMI	TERM	6/10, 8/10	CEREBRAL EDEMA	OB	NIL	DIS	YES	2.2
45	B/O SUDHARANI	3/365	MCH	NORMAL	G2	TERM	6/10,8/10	PVF	IB	NIL	DIS	NO	2.2
46	B/O PARVEEN	2/365	FCH	NORMAL	PRIMI	TERM	6/10,7/10	CEREBRAL EDEMA	OB	NIL	DIS	NO	2.1

47	B/O MUTHALAGI	4/365	MCH	NORMAL	PRIMI	TERM	5/10,7/10	PVF	OB	HIE	DIS	YES	2.2
48	B/O BANUPRIYA ELUMALAI	3/365	MCH	PIH	PRIMI	TERM	5/10,6/10	PVF	OB	NIL	DEAD	YES	2.1
49	B/O PRAJEETHA	3/365	FCH	NORMAL	PRIMI	TERM	5/10, 7/10	CEREBRAL EDEMA	OB	NIL	DIS	YES	2.2
50	B/O SWATHI	3/365	FCH	NORMAL	PRIMI	PRETERM(36)	6/10,7/10	NS	IB	HIE	DIS	YES	2.1
51	B/O PRIYANKA	3/365	MCH	NORMAL	PRIMI	PRETERM(36)	7/10,8/10	NS	IB	NIL	DIS	NO	2.1
52	B/O MATHINA	3/365	FCH	PPROM	PRIMI	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	2
53	B/O LALITHA	3/365	MCH	PPROM	G2	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	2.1
54	B/O SHANTHINI	4/365	MCH	PIH	PRIMI	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	2.1
55	B/O SARALA	4/365	MCH	PIH	PRIMI	PRETERM(34)	7/10, 8/10	NS	IB	NIL	DIS	NO	2.2
56	B/O USHA	4/365	MCH	PRE-ECLAMPSIA	PRIMI	PRETERM(34)	8/10,9/10	NS	IB	NIL	DIS	NO	2.1
57	B/O THILAGAVATHI	3/365	MCH	NORMAL	PRIMI	PRETERM(36)	7/10,8/10	NS	IB	NIL	DIS	NO	2.1
58	B/O PERUMAYEE	3/365	FCH	POLYHYDRAMNIOS	G2	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	2.2
59	B/O PAVITHRA TWINA	3/365	MCH	TWIN	G4	PRETERM(36)	7/10,8/10	NS	IB	NIL	DIS	NO	1.9
60	B/O RESHMI	4/365	FCH	NORMAL	G3	PRETERM(34)	8/10,9/10	NS	IB	NIL	DIS	NO	1.8
61	B/O MANIMALA	2/365	FCH	PIH	G2	PRETERM(36)	7/10,8/10	NS	OB	NIL	DIS	NO	1.9
62	B/O RAJALAKSHMI	3/365	MCH	PIH	G2	PRETERM(36)	8/10,9/10	NS	OB	NIL	DIS	NO	1.7
63	B/O SHANTHI	4/365	FCH	PIH	PRIMI	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	1.6
64	B/O VASUNDRA	4/365	MCH	NORMAL	G2	PRETERM(33)	7/10,8/10	NS	IB	NIL	DIS	NO	1.6
65	B/O YOGALAKSHMI	3/365	FCH	PIH	G2	PRETERM(34)	6/10,7/10	NS	IB	NIL	DIS	NO	1.8
66	B/O SUGANYA	3/365	MCH	PIH	PRIMI	PRETERM(36)	7/10,8/10	NS	IB	NIL	DIS	NO	1.6
67	B/O SUGUNA	4/365	MCH	UTI	G3	PRETERM(36)	8/10,9/10	NS	IB	NIL	DIS	NO	1.7
68	B/O LATHA SIVAKUMAR	3/365	MCH	ANEMIA	G2	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	1.8
69	B/O SARANYA VELU	3/365	MCH	PIH	PRIMI	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	1.9
70	B/O BHUVANESWARI	3/365	MCH	PPROM	PRIMI	PRETERM(35)	7/10,8/10	NS	IB	NIL	DIS	NO	1.6
71	B/O MADHAMMAL	3/365	FCH	NORMAL	G3	PRETERM(36)	7/10,8/10	NS	IB	NIL	DIS	NO	1.7

72	B/O RADHIKA	4/365	FCH	PIH	PRIMI	PRETERM(34)	7/10,8/10	NS	IB	NIL	DIS	NO	1.8
73	B/O THANGAM	3/365	MCH	NORMAL	G2	PRETERM(35)	7/10,8/10	NS	OB	NIL	DIS	NO	1.6
74	B/O JOTHIMANI	3/365	MCH	GDM	PRIMI	PRETERM(34)	8/10,9/10	NS	IB	NIL	DIS	NO	1.7
75	B/O NIGALYA	4/365	MCH	NORMAL	PRIMI	PRETERM(36)	6/10,7/10	NS	IB	NIL	DIS	NO	1.8
76	B/O GAYATHRI AJITH	4/365	FCH	HYPOTHYROID	PRIMI	PRETERM(34)	6/10,8/10	NS	IB	NIL	DIS	NO	1.8
77	B/O KASTHURI	2/365	MCH	PIH	PRIMI	PRETERM(34)	6/10,7/10	NS	IB	NIL	DIS	NO	1.8
78	B/O SHANTHI SELVARAJ	3/365	FCH	HYPOTHYROID	PRIMI	PRETERM(36)	6/10,7/10	NS	OB	NIL	DIS	NO	1.6
79	B/O BANUPRIYA ARIVU	4/365	FCH	NORMAL	PRIMI	PRETERM(34)	6/10,7/10	HYDROCEPHALUS	IB	HIE	DEAD	YES	1.4
80	B/O ABINAYA SARAVANAN	3/365	MCH	PIH	PRIMI	PRETERM(31)	5/10,7/10	PVL	OB	HIE	DEAD	YES	1.1
81	B/O KAMATCHI	4/365	FCH	NORMAL	G4	PRETERM(34)	6/10,8/10	PVL	OB	HIE	DIS	YES	1.2
82	B/O NITHYA	3/365	FCH	NORMAL	G2	PRETERM(36)	6/10,7/10	PVL	IB	HIE	DIS	YES	1.1
83	B/O VASANTHI	4/365	MCH	NORMAL	G4	PRETERM(36)	6/10,7/10	CEREBRAL EDEMA	IB	NIL	DIS	NO	1.3
84	B/O CHANDRAKALA	3/365	MCH	PPROM	G2	PRETERM(31)	7/10,8/10	PVF	IB	NIL	DIS	NO	1.4
85	B/O VEERAMMAL	4/365	MCH	PIH	PRIMI	PRETERM(31)	7/10,8/10	PVF	IB	NIL	DIS	NO	1.2
86	B/O BANU	3/365	FCH	GDM	PRIMI	PRETERM(36)	6/10,8/10	CEREBRAL EDEMA	IB	NIL	DIS	NO	1.1
87	B/O SUREKHA	3/365	MCH	PPROM	G2	PRETERM(34)	6/10,8/10	PVF	OB	HIE	DIS	YES	1.2
88	B/O ROOBI	4/365	MCH	OLIGO	PRIMI	PRETERM(34)	6/10,7/10	CEREBRAL EDEMA	IB	NIL	DIS	YES	1.2
89	B/O SASIKALA	7/365	FCH	NORMAL	PRIMI	PRETERM(33)	7/10,8/10	CEREBRAL EDEMA	OB	SEPSIS	DEAD	YES	1.1
90	B/O KALAIVANI	4/365	FCH	NORMAL	PRIMI	PRETERM(28)	6/10,7/10	PVL	IB	HIE	DEAD	YES	0.9
91	B/O KEERTHIKA	4/365	MCH	GDM	G2	PRETERM(35)	5/10,7/10	IVH	OB	HIE	DEAD	YES	1.3
92	B/O THANGAPONNU	2/365	MCH	AN SCAN	PRIMI	PRETERM(31)	5/10,7/10	SOL	IB	HIE	DEAD	YES	1.3
93	B/O SHYAMALA VENKATESH	4/365	MCH	NORMAL	PRIMI	PRETERM(33)	6/10,7/10	HYDROCEPHALUS	IB	SEPSIS	DEAD	YES	1.1
94	B/O SUDHA	3/365	MCH	ANEMIA	PRIMI	PRETERM(26)	5/10,7/10	IVH	OB	HIE	DEAD	YES	0.9
95	B/O BHARATHI	3/365	FCH	ANEMIA	PRIMI	PRETERM(34)	6/10,7/10	PVL	OB	HIE	DEAD	YES	1.2
96	B/O KARPAGAM	3/365	FCH	NORMAL	PRIMI	PRETERM(30)	5/10,7/10	PVL	IB	HIE	DEAD	YES	1

97	B/O SUMITHRA	4/365	FCH	GDM	PRIMI	PRETERM(30)	6/10,7/10	PVF	IB	NIL	DEAD	YES	1.1
98	B/O JOTHI PRABHU	7/365	FCH	GDM	G3	PRETERM(33)	6/10,7/10	PVF	IB	SEPSIS	DEAD	YES	1.2
99	B/O LATHA THIYAGU	3/365	MCH	NORMAL	PRIMI	PRETERM(28)	5/10,7/10	GMH	IB	HIE	DEAD	YES	0.9
100	B/O DEVI	4/365	MCH	PIH	PRIMI	PRETERM(30)	6/10,7/10	GMH	IB	HIE	DEAD	YES	1

KEY TO MASTER CHART

1.	AN & N H/O	-	ANTENATAL AND NATAL HISTORY
2.	B.W	-	BIRTH WEIGHT
3.	COMP	-	COMPLICATIONS
4.	CUS	-	CRANIALULTRASOUND
5.	DIS	-	DISCHARGE
6.	FCH	-	FEMALE CHILD
7.	GA	-	GESTATIONAL AGE
8.	GDM	-	GESTATIONAL DIABETES MELLITUS
9.	GMS	-	GERMINAL MATRIX HEMORRHAGE
10.	HIE	-	HYPOXIC ISCHEMIC ENCEPHALOPATHY
11.	IB	-	INBORN
12.	IVH	-	INTRAVENTRICULAR HEMORRHAGE
13.	MCH	-	MALE CHILD
14.	MSAF	-	MECONIUM STAINED AMNIOTIC FLUID
15.	MV	-	MECHANICAL VENTILATION
16.	NS	-	NORMAL STUDY
17.	OB	-	OUTBORN
18.	PIH	-	PREGNANCY INDUCED HYPERTENSION
19.	POD	-	PLACE OF DELIVERY
20.	PPROM	-	PRETERM PREMATURE RUPTURE OF MEMBRANE
21.	PVF	-	PERIVENTRICULAR FLARE
22.	PVL	-	PERIVENTRICULAR LEUKOMALACIA
23.	SOL	-	SPACE OCCUPYING LESION