Section: Paediatrics

Original Research Article

# CRANIAL ULTRASONOGRAPHY IN NEONATES ADMITTED IN NICU IN TERITIARY CARE CENTER

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ABSTRACT

**Background:** In parallel to the dawn of modern neonatal intensive care, the survival of the Neonate has greatly improved. Cranial ultrasound is the most available 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. Modern machines, probes, a variety of acoustic windows and sequential scanning at optimal times giving high-quality images has increased with the recognition of more subtle patterns of injury and features suggestive of developmental, metabolic and infectious disorders. **Objectives:** To assess the importance of cranial ultrasound as an investigatory modality for neonates and to find out the morphology of various cerebral lesions and correlate clinically.

**Materials and Methods:** An observational prospective clinical study conducted on 100 neonates at NICU, Department of Paediatrics, Siddhartha Medical College, Vijayawada. During February 2024 to February 2025. After obtaining Informed consent from the parents/guardian details were recorded in a pre-designed pretested, structured proforma. Detailed clinical examination and Cranial ultrasound was performed on 1st,3rd and on 7th post-natal day. If cranial ultrasonography revealed various findings, repeat neurosonogram were done to follow up sequelae if any.

**Results:** On cranial ultrasound, 67% of neonates had abnormal findings. 18% of these had evidence of intracranial bleed, 6% hyperechogenic thalami, 2% definite HIE, 4% had cerebral edema. One preterm neonate on regular follow up developed findings suggestive of cystic periventricular leukomalacia. Two neonates on regular follow up CUS had developed cystic encephalomalacia with hydrocephalus.

**Interpretation and Conclusion:** Cranial ultrasonography (CUS) is the best point of care neuroimaging method available for neonates. It is critical as an investigatory modality in NICU and effectively documents morphology of cerebral damage.

**Keywords:** Cranial ultrasound; NICU; Thalamic hyperechogenecity; Intraventricular hemorrhage; HIE.

## INTRODUCTION

Cranial ultrasonography (CUS) has become an essential diagnostic tool in modern neonatology for depicting normal anatomy and pathological changes in neonatal brain. In the neonate, many sutures and fontanels are still open and these can be used as acoustic windows to "look" into the brain.[1] Any neonate, regardless of birth weight, size, or gestational age, who has a greater than average chance of morbidity or mortality, due to foetal, maternal or placental anomalies or an otherwise compromised pregnancy, especially within the first 28 days of life is categorised as high risk neonate.[2] CUS plays an important role in assessing neurological prognosis of these high-risk infants. It is cheap, easy to perform, non-invasive and can be initiated at a very early stage, even immediately after

birth. It can be repeated as often as necessary, and thereby enables visualisation of ongoing brain maturation and the evolution of brain lesions. In addition, it can be used to assess the timing of brain damage.[1] As a result of ongoing development in ultrasonography, image quality is high nowadays, provided optimal settings and techniques are applied. Using additional acoustic windows can significantly augment the diagnostic power of CUS. Scanning through the posterior and mastoid fontanelle, can help to detect lesions and structural malformations in cerebellum, brainstem and posterior sub cortical white matter. Imaging through the temporal window allows good views of the mesencephalon and brainstem.[3]

Cranial ultrasound (CUS) provides bedside imaging access to the neonatal brain. It is a reliable tool for detecting congenital and acquired abnormalities of the perinatal brain and most frequent patterns of brain injury in preterm and full term neonate. It detects most of the haemorrhagic, ischemic and cystic brain lesions as well as calcifications, cerebral infections and major structural abnormalities in preterm and full term infants.[1] CUS is also very helpful in the early diagnosis of the many aetiologies of neonatal encephalopathy and seizures in the term infant and the subsequent monitoring of progress of hypoxic-ischemic brain injury. In seriously ill neonates and in neonates with serious cerebral abnormalities, either congenital or acquired, it plays a role in decisions on continuation or withdrawal of intensive treatment. In neonates surviving with cerebral injury, it may help to optimize treatment of the infant both during the neonatal period and thereafter.[4] If the quality of CUS is good, timing is carefully chosen, proper transducers are used, and, in the case of preterm birth, serial examinations are continued until term age, most diagnoses will not remain undetected, and the reliability and prognostic value of CUS can be high. Serial CUS examinations enable assessment of the onset of injury and the evolution of lesions.[1]

When using a CUS screening programme, it is important to realise that such a programme is suitable for neonates without neurological symptoms or brain pathology or for neonates with stable brain abnormalities (such as congenital anomalies or acquired but stabilised lesions). In cases of (suspected) cerebral and/or neurological abnormalities, the intensity and frequency of CUS examinations may need to be increased, depending on the clinical picture and the lesion(s).[1] Most newborn intensive care unit centres perform serial cranial ultrasound evaluations early in the course of hospitalization for premature infants and often, a follow-up examination is done at a later age. These evaluations are done to document the presence of intracranial haemorrhage, to guide choice of therapies that may exacerbate risk of further haemorrhage, and to counsel families about neurodevelopment outcomes.[5] While early CUS imaging concentrated on IVH, HPI and cystic PVL, the incidence of these pathologies has diminished considerably. Current studies aim more at detecting subtle white matter disease, assessing brain growth and maturation, and predicting neurodevelopment outcome from CUS. Appropriate timing of CUS and accurate assessment of the site and extent of lesions is crucial for accurate prediction of neurodevelopment outcome. Several studies have suggested that only in 40–50% of preterm infants with cerebral palsy (CP), lesions are detected on CUS. However, if only one or two early or late CUS scans are performed, the detection of cystic PVL, the most predictive CUS marker for CP, is less reliable. It is the site and extent of cerebral lesions that is important in predicting CP.[6]

In full term infants, CUS has an important role in the diagnosis 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 congenital infectious disorders.[7] CUS very helpful in assessing severity and neurodevelopment outcome in infants with HIE. It is also important to realise that the end stages of hypoxic-ischemic brain damage may not become visible until a variable period of time, often several weeks to months after the event, and that the early stages may seem mild or subtle. In cases of (suspected) ischemic injury, even if apparently mild, it is therefore advisable to intensify CUS examinations until normalisation or stabilisation of abnormalities has occurred.[7] CUS reliable for detecting common markers of metabolic disease in neonates, such as germinolytic cysts, lenticulostriate vasculopathy and more extensive BG calcification, subtle WM abnormalities and cortical and other structural abnormalities.[8] Meningitis and brain infections can have a very rapid, fulminant course and should therefore be intensively monitored by repetitive CUS. The quality of CUS imaging and its diagnostic accuracy depends on the suitability of the ultrasound machine for neonatal work, appropriate settings and probes and also the experience and expertise of the examiner.[10] Modern ultrasound machines and probes and the use of a variety of acoustic windows and adequate scanning protocols give high quality images that are diagnostically accurate.[10]

**Aims and Objectives**

- To assess the importance of cranial ultrasound as an investigatory modality for neonates admitted in neonatal intensive care unit.
- To find out the morphology of various cerebral lesions and correlate clinically.

## MATERIALS AND METHODS

It was a prospective observational clinical study conducted on 100 neonates at NICU, Department of Paediatrics, Siddhartha Medical College, Vijayawada. During February 2024 to February 2025. After obtaining Informed consent from the

parents/guardian details were recorded in a pre-designed pretested, structured proforma. Detailed clinical examination and Cranial ultrasound and doppler evaluation was performed on 1st ,3rd and on 7th post-natal day.

One hundred neonates admitted to neonatal intensive care unit were selected as per the inclusion criteria on non-randomized purposive sampling basis and were subjected to neurosonography on selected days. If cranial ultrasonography revealed various findings, repeat neurosonogram were done to follow up sequelae if any.

**Method of Collection of Data**

Informed consent was obtained from the parents/guardian regarding inclusion of the neonate in the study. All perinatal details were recorded and detailed clinical examination was done including anthropometric measurements. Vital parameters were recorded within 24-48 hrs of admission and complete neurological examination was done during baby's stay in NICU. Gestational age was assessed as per modified Ballard's scoring method for all preterm neonates. Evaluation with baseline routine investigations [septic and metabolic work up] and lumbar puncture in case of neonatal convulsions and neonatal sepsis, chest X-ray in all respiratory distress cases was done. Cranial ultrasound of the neonates admitted in NICU was performed. Follow up cranial ultrasound were done in case of findings revealed and for preterm neonates. Morphology of cranial ultrasound findings was studied and recorded and clinical correlation with various findings on cranial ultrasound was done. Neonates were followed till recovery and discharge from NICU.

**Inclusion Criteria**

All the neonates admitted in NICU

**Exclusion Criteria**

1. Transient tachypnoea of newborn
2. Babies with only hyperbilirubinemia
3. Babies > 28 days

**Statistical Methods:** Prospective statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean±SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance. The following assumption on data is made. Assumptions: 1. Dependent variables should be normally distributed, 2. Samples drawn from the population should be random, Cases of the samples should be independent Chi-square/Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

**Statistical Software:** The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft Word and Excel have been used to generate graphs, tables etc.

## RESULTS

**Study Design:** An observational correlation study of 100 neonates is undertaken to assess the importance of cranial ultrasound as an investigatory modality and find out the morphology of various cerebral lesions.

**Table 1: Incidence of CUS abnormalities in neonates**

| Cranial ultrasound | Number of neonates(n=100) | % |
|---|---|---|
| normal | 33 | 33% |
| abnormal | 67 | 67% |

Incidence of CUS abnormalities in neonates is 67% in the present study.

**Table 2: Gender distribution of neonates studied**

| Gender | Number of neonates | % |
|---|---|---|
| male | 62 | 62 |
| female | 38 | 38 |
| Total | 100 | 100 |

**Table 3: Distribution of neonates as per gestational age and CUS findings**

| Gestational age | Number of neonates | CUS findings | |
|---|---|---|---|
| | | Abnormal | normal |
| 28-32 | 35 | 33(94.28%) | 02(5.71%) |
| 33-36 | 31 | 29(93.54%) | 03(9.67%) |
| 37-40 | 34 | 08(23.52%) | 28(82.35%) |
| Total | 100 | 67 | 33 |

Significance 0.670

There were 66% preterm and 34% term neonates enrolled in the present study. Of these, 35% preterm were less than 32 weeks, 31% preterm were between 33 and 36 weeks and rest 34% were term neonates.

Correlation between gestational age and CUS findings was not statistically significant.

**Table 4: Correlation of birth weight (kg) with cranial ultrasound findings**

| Birth weight (kg) | Cranial ultrasound | |
|---|---|---|
| | Abnormal (67) | Normal (33) |
| <1.5 | 11(14.9%) | 4(15.4%) |
| 1.5-2.0 | 19(25.7%) | 7(26.9%) |

| | | |
|---|---|---|
| 2.0-2.50 | 15(20.3%) | 5(19.2%) |
| 2.5-3.0 | 16(25.7%) | 9(23.1%) |
| >3.50 | 06(13.5%) | 8(15.4%) |
| Mean ±SD | 2.21±0.74 | 2.15±0.68 |
| Inference | Mean birth weight is less in abnormal CUS (2.15kg) compared to normal CUS with p=0.656 | |

The mean birth weight of neonates with abnormal CUS (2.15±0.68) was less than those normal CUS (2.21±0.74).

Correlation of birth weight of neonates with abnormal cranial ultrasound findings revealed that 50.74% low birth weight neonates and 14.9% very low birth weight neonates had abnormal CUS.

**Table 5: Distribution of neonates as per mode of delivery**

| Mode of delivery | Number of neonates | % |
|---|---|---|
| Vaginal | 51 | 51 |
| LSCS | 49 | 49 |
| Total | 100 | 100 |

In the present study, 51% neonates were born via normal labour and 49% via LSCS for various reasons.

**Table 6: Correlation of perinatal risk factors with cranial ultrasound findings in neonates**

| Perinatal factors | Cranial ultrasound | | Significance |
|---|---|---|---|
| | Normal | Abnormal | |
| PIH | 12(16.2%) | 9(34.6%) | 0.048* |
| APH | 0(0%) | 0(0%) | - |
| PROM | 11(14.9%) | 4(15.4%) | 0.995 |
| MP | 5(6.8%) | 1(3.8%) | 0.591 |
| BIRTH TRAUMA | 2(2.7%) | 2(7.7%) | 0.264 |

***Moderately significant (p value: 0.01)***

In the present study, of the 67% neonates having abnormal CUS findings, 34.6% of neonates were born to mothers with PIH, 15.4% neonates born with PROM, 3.8% born out of multiple pregnancy 7.7% had birth trauma. In the correlation of perinatal risk factors with abnormal cranial ultrasound findings, there was statistically significant correlation with PIH. Correlation of APH, PROM, multiple births and birth trauma was statistically not significant.

**Table 7: Incidence of abnormal clinical examination in neonates with abnormal CUS findings and their correlation**

| Clinical examination | Cranial ultrasound | | significance |
|---|---|---|---|
| | Normal (n=33) | Abnormal (67) | |
| Abnormal cry | 10(13.5%) | 8(30.8%) | 0.049* |
| Poor activity | 12(16.2%) | 10(38.5%) | 0.018* |
| Poor/Abnormal tone | 3(4.1%) | 5(19.2%) | 0.014* |
| Poor reflexes | 4(5.4%) | 2(7.7%) | 0.673 |
| Abnormal posture | 3(4.1%) | 2(7.7%) | 0.464 |
| Presence of pallor | 1(1.4%) | 2(7.7%) | 0.103 |
| Presence of icterus | 19(25.7%) | 4(15.4%) | 0.283 |
| Presence of cyanosis | 18(24.3%) | 12(46.2%) | 0.037* |
| Tachycardia (HR>160) | 3(4.1%) | 1(3.8%) | 0.963 |
| Tachypnea (RR >60) | 74(100%) | 26(100%) | 1.000 |
| Abnormal CFT | 5(6.8%) | 3(11.5%) | 0.439 |
| Abnormal temperature | 4(5.4%) | 0(0%) | 0.226 |
| Presence of CHD (CVS column) | 18(24.3%) | 9(34.6%) | 0.30 |
| Abnormal RS | 35(47.3%) | 11(42.3%) | 0.740 |

***Moderately significant (p value: 0.01<p≤ 0.05)***

Of the 67% neonates having abnormal CUS, 100% had respiratory distress, 46.2% were cyanosed, 42.3% had abnormality in respiratory system, 38.5% had poor activity, 34.6% had CHD, 19.2% had abnormal tone, 15.4% were icteric, 7.7% were pale, 7.7% had abnormal posture and poor neonatal reflexes, 11.5% had poor capillary filling time. There was statistically significant correlation between abnormal cry, abnormal tone, abnormal activity, and presence of cyanosis on clinical examination and presence of abnormalities on cranial ultrasound.

**Table 8: Correlation of gestational age with various cranial ultrasound findings**

| Cranial ultrasound | Number of neonates n=100 | Gestational age (weeks) | | | P value |
|---|---|---|---|---|---|
| | | >32 weeks n=35 | 33-36 weeks n=31 | <37 weeks N=34 | |
| Normal | 67 (67%) | 02(5.7%) | 08 (25.8%) | 28(82.35%) | 0.170 |
| Abnormal | 33 (33%) | 33 (94.28) | 29 (93.5%) | 08(23.52%) | |

| | | | | | |
|---|---|---|---|---|---|
| 1.GMH | 17 (17%) | 04(11.42%) | 07(22.58%) | 06(17.64%) | 0.140 |
| 2.OTHER BLEEDS | 01 (1%) | 0 (0%) | 0 (0%) | 01(2.94%) | 1.000 |
| 3.HIE | 02 (2%) | 01(3%) | 0 (0%) | 01(2.94%) | 0.322 |
| 4.THALAMIC HYPERECHOGENICITY | 06 (6%) | 01(3%) | 0 (0%) | 05(14.7%) | 0.464 |
| 5.CEREBRAL EDEMA | 04 (4%) | 0 (0%) | 0 (0%) | 04(11.76%) | 0.467 |
| OTHER FINDINGS | 03 (3%) | 01(3%) | 02 (6%) | 0 (0%) | 0.0057 |

There was no statistical correlation between different findings on CUS and gestational age of neonate. Of the neonates with gestational age less than 32 weeks having abnormal findings on CUS 11.42% had GMH, 3% had HIE and thalamic hyperechogenecity. Of the neonates with gestational age between 33 and 36 weeks having abnormal findings on CUS, 22.58% had GMH. Of the neonates with gestational age more than 37 weeks having abnormal findings on CUS 14.7% had thalamic hyperechogenecity, 11.76% had cerebral oedema and 20% had intracranial bleeds.

**Table 9: Correlation of CUS abnormalities in birth asphyxia neonates**

| Cranial ultrasound | Number of neonates N=100 | Apgar score | | | |
|---|---|---|---|---|---|
| | | NORMAL N=68 | MILD N=15 | MODERATE N=10 | SEVERE N=07 |
| Normal | 33(37%) | 18(26.5%) | 7(46.6%) | 5(50%) | 3(42.9%) |
| Abnormal | 67(67%) | 48(70.6%) | 09(60%) | 06(60%) | 04(57.14%) |
| a)GMH | 17 (17%) | 11(16.3%) | 3(20%) | 2(20%) | 1(14.3%) |
| b)other bleeds | 01 (1%) | 1(1.5%) | 0(0%) | 0(0%) | 0(0%) |
| c)HIE | 02 (2%) | 2(2.9%) | 0(0%) | 0(0%) | 0(0%) |
| d)thalamic hyperechogenicity | 06 (6%) | 7(10.3%) | 1(6.7%) | 0(0%) | 1(14.3%) |
| e)cerebral edema | 04 (4%) | 0(0%) | 4(26.7%) | 0(0%) | 0(0%) |
| f)other findings | 03 (3%) | 3(4.4%) | 1(6.7%) | 0(0%) | 0(0%) |

**Patient had multiple CUS findings**

Of all the neonates with perinatal asphyxia, 46.9% had normal and 27.5% had abnormal CUS findings. Correlation between CUS findings of neonates with perinatal asphyxia was not statistically significant. All neonates in the present study who were found to have cerebral oedema on CUS had evidence of mild perinatal asphyxia. Of the 67% of neonates with abnormal findings on CUS 70.6% had no evidence of perinatal asphyxia, 57.14% had severe, 60% had moderate 60% had mild perinatal asphyxia as per Apgar scoring.

**Table 10: Distribution of neonates based timing of cranial ultrasound**

| Day of life of CUS | Cranial ultrasound | | Significance |
|---|---|---|---|
| | NORMAL (N=33) | ABNORMAL (N=67) | |
| <24 hours | 3(9.09%) | 0 | 0.566 |
| 24-72 hours | 19(57.5%) | 67(100 %) | 0.019* |
| >72 hours | 11(33.3%) | 0 | 0.060+ |

+ Suggestive significance (p value: 0.05<p<0.10), * Moderately significant (p value: 0.01<p≤ 0.05)

There was statistically significant correlation between findings on CUS and day of life of neonate when CUS was done. All the abnormal findings on CUS were picked up during 24-72 hours of life. 57.5% normal CUS were picked up during 24-72 hours, 9.09% in less than 24 hours and 33.3% after 72 hours of life.

**Table 11: Distribution of neonates based timing of cranial ultrasound and gestational age**

| Gestational age | Day of life of CUS | | | Significance |
|---|---|---|---|---|
| | <24 hours | 24-72 hours | >72 hours | |
| Preterm | 0 | 47(54.0%) | 0 | <0.001** |
| Term | 3(100.0%) | 40(45.9%) | 10(100.0%) | |
| Total | 3(100.0%) | 87(100.0%) | 10(100.0%) | |

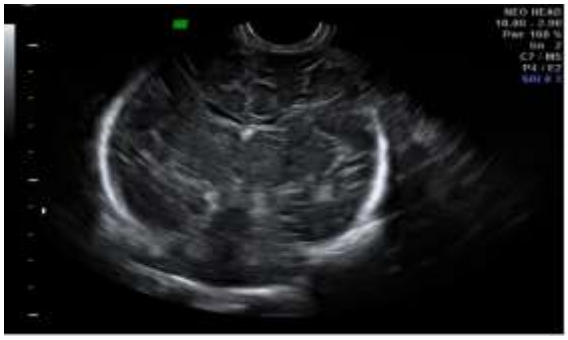
There was statistically significant correlation between gestational age of the neonates included in the study and day of life CUS was done for them. All the preterms enrolled in the study CUS was done between 24 to 72 hours of life while for majority of the term neonates also, CUS was done between 24 to 72 hours of life.

**Table 12: Correlation of clinical outcome with various cranial ultrasound findings**

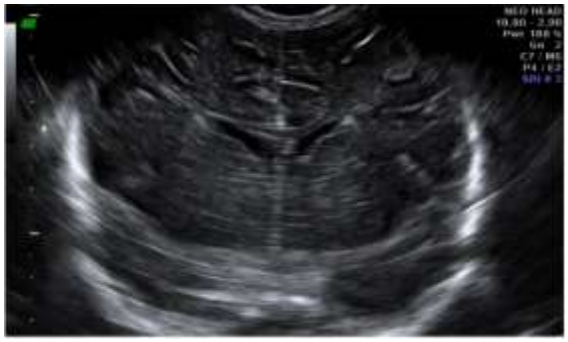
| Cranial ultrasound | Number of neonates (n=100) | Clinical outcome | | | P value |
|---|---|---|---|---|---|
| | | Recovery (n=95) | DAMA/Discharge (n=9) | Death (n=2) | |
| Normal | 33(37%) | 31(32.63%) | 2(22.22%) | 0(0%) | 0.285 |
| Abnormal | 67(67%) | 58(61.05%) | 07(77.77%) | 2(100%) | |
| a)GMH | 17 (17%) | 10(10.52%) | 6(66.66%) | 1(50%) | 0.419 |
| b)other bleeds | 01 (1%) | 0(0%) | 0(0%) | 1(50%) | 0.110 |
| c)HIE | 02 (2%) | 2(2.10%) | 0(0%) | 0(0%) | 1.000 |
| d)thalamic hyperechogenicity | 06 (6%) | 5(5.26%) | 1(11.1%) | 0(0%) | 0.513 |
| e)cerebral edema | 04 (4%) | 4(4.2%) | 0(0%) | 0(0%) | 1.000 |
| f)other findings | 03 (3%) | 3(3.15%) | 0(0%) | 0(0%) | 1.000 |

There was no statistically significant correlation between various findings on CUS
and clinical outcome of the neonate.
Of the neonates having good clinical recovery, 32.63% had normal CUS and 61.05% had abnormal findings of which 10.52% had evidence of GMH, 5.26% had thalamic hyperechogenecity, 4.2% had cerebral edema, 3.15% had other findings and 2.10% had HIE.
Of the neonates who were discharged from NICU before clinical recovery, 22.22% had normal CUS and 77.77% had abnormal findings of which 33.3% had evidence of intracranial bleed, 11.1% had thalamic hyperechogenecity. Both the neonates that died during NICU stay had abnormal CUS which had evidence of intracranial bleed.

**B/O DEEPIKA**

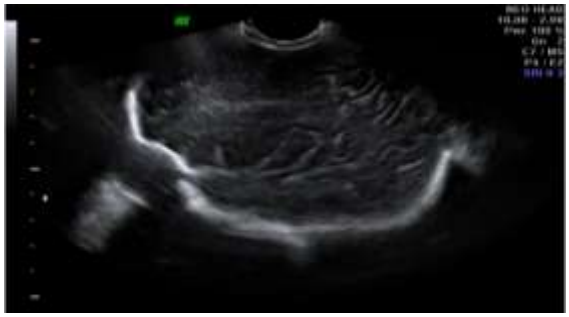


**Coronal Through Sylvian Fissure: Normal**

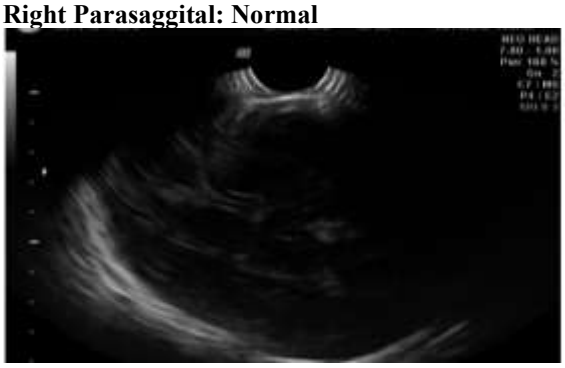


**Coronal through Foramen of Munro: Normal**

**B/o DEEPIKA**

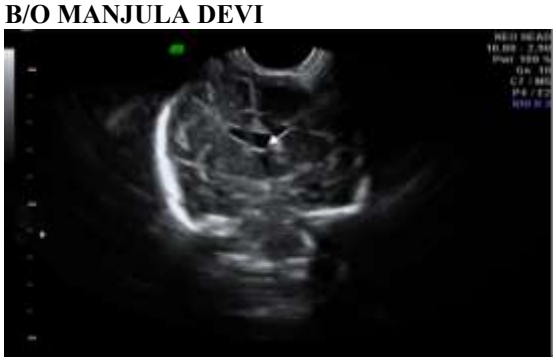


**Right Parasaggital: Normal**



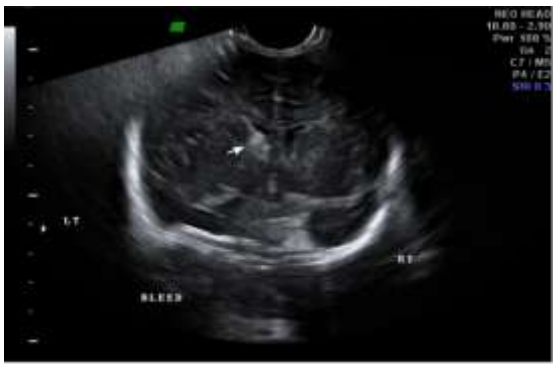
**Normal Transtemporal Axial Scan**

**B/O MANJULA DEVI**



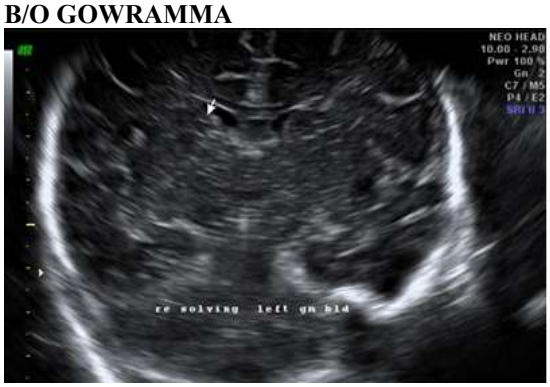
**Left GMH Grade II**

**B/O JYOTHY**



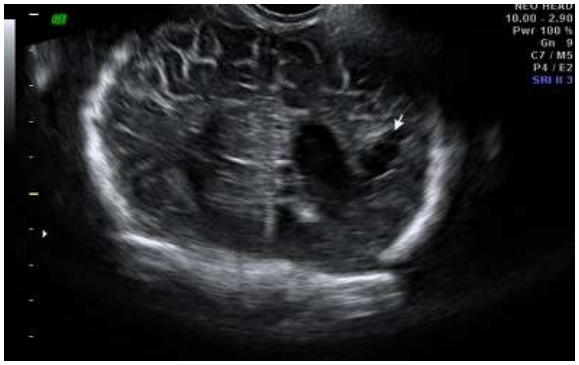

**Right GMH Grade 1**

**B/O GOWRAMMA**




**Resolving Germinal Matrix Bleed on F/ Up**

**B/O BHAVYA**

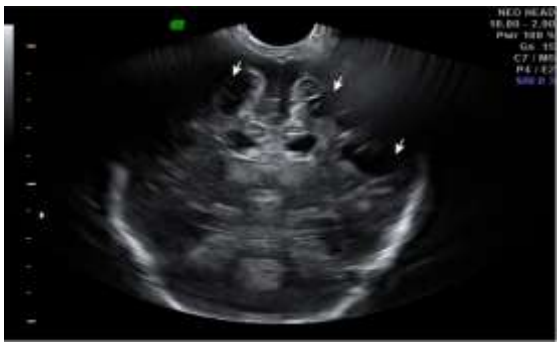


**Old parenchymal bleed communicating with ventricle causing hydrocephalus with intraventricular septations**

**B/O KRISHNAVENI**

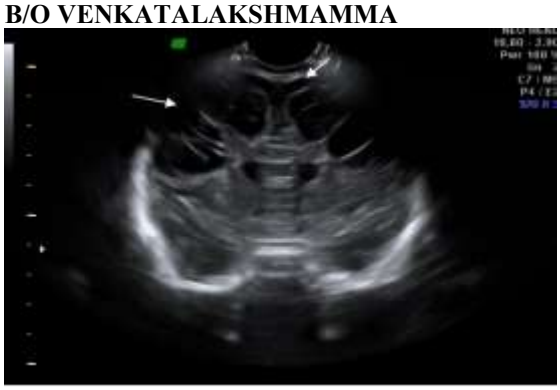

**Hyperechoiec Thalami & Periventricular Hyperechogenecity**

**B/O SANTOSHINI**



**Multicystic Encephalomalacia & Hydrocephalus**

**B/O VENKATALAKSHMAMMA**



**Multicystic Encephalomalacia and Hydrocephalus**

## DISCUSSION

Daneman A, Epelman M et al,[11] proved that CUS remains an extremely useful modality for evaluation of the full-term neonatal brain. De Vries and Cowan et al,[12] have suggested that head ultrasound and MRI are complementary modalities, with ultrasound as an especially useful tool in the early days, when the infant is unstable for transport and ultrasound findings may be sufficient for major clinical decisions. Present study aims at proving the same.

Incidence of CUS abnormalities in Neonates is 67% in the present study. There were 62% male and 38% female neonates enrolled in the study. There were 66% preterm and 34% term neonates enrolled in the present study.

Eugenio Mercuri, Lilly Dubowitz et al,[13] reported an incidence 20% of ultrasound abnormalities in apparently well neonates.

Ayala Gover, David Bader et al,[14] reported an incidence of 11.2% abnormalities on CUS in apparently healthy asymptomatic term neonates.

Linda S. De Vries et al,[15] assessed sequential high-resolution cranial ultrasound (US) in high-risk preterm infants to predict cerebral palsy (CP) and found 79% percent of CP cases had major CUS abnormalities.

Badrawy N, Edrees A, and Mohamed El Ghawas et al16 showed in their study that 37% preterms had abnormal CUS findings. Their study had 64% male and 36% female neonates. Of these, 9% were less than 30 weeks, 33% between 30-33 weeks and 58% were between 34 and 37 weeks. In the present study,

35% neonates were less than 32 weeks, 31% between 33 and 36 weeks and the rest 34% term gestation. Eugenio Mercuri, Lilly Dubowitz et al,[13] reported ischemic lesions, such as periventricular and thalamic densities were the most common finding (8%), followed by intracranial haemorrhagic lesions (6%) on CUS. Badrawy N. et al,[16] reported that subependymal intraventricular haemorrhage (SE-IVH) was present in 14%, brain oedema in 9%, hypoxic ischemic changes in 4%, post haemorrhagic hydrocephalus (PHH) in 3.5% as a complication of SE-IVH. In the present study, on CUS, 67% of neonates had abnormal findings. 18% of these had evidence of intracranial bleed, 6% hyperechogenic thalami, 2% definite HIE, 4% had cerebral oedema. Badrawy Net al,[16] reported congenital hydrocephalus to be present in 6% among all neonates screened by them. In the present study, one neonate had congenital hydrocephalus with aqueductal stenosis. Chowdhury V, Gulati P et al,[17] in their study on 50 preterm neonates of which 58% were males and 42% females, detected intracranial pathology in 12% of preterms and 6% of these had intracranial haemorrhage. In the present study, 66% of neonates were preterms of which 89.3% had intracranial pathology of which 17% had intracranial haemorrhage. Soni JP, Gupta BD et al,[18] in their study suggested that CUS is sensitive and specific for the detection of various types of ICH (SEH, IVH, PVL). One hundred and eleven high risk neonates were subjected to CUS, one quarter of these neonates developed intracranial haemorrhage (1CH) within 120 hours of birth. Trounce et al reported that ultrasonic evidence of haemorrhage was evident within first seven days of life in 78% neonates and second week in 15% neonates. Gupta et al from their study reported that haemorrhage occurred in all neonates within 96 hours of birth. Meidell et al,[19] found that 88% of GM/IVH were diagnosed in the first 24 hours of life. Ment et al,[20] found that 74% of haemorrhages were seen by 30 hours of age, and De Crespigny et al found that 71% of GM/IVH was diagnosed by 6 hours of age. Similar findings have also been reported by Beverley et al and by Murton et al, who detected germinal matrix haemorrhage in some infants immediately after delivery. In the present study 18% neonates have evidence of intracranial bleed (GMH, IVH) all of which were picked up between 24 to 72 hours of life. Elia FM et al, concluded that CUS accurately predicts the presence of GMH, intraventricular and parenchymal haemorrhage. Batton DG, Holtrop et al, Harding D, Evans N et al and Paneth N, Pinto-Martin J et al in their studies concluded that the maximum risk of GM and/or IVH is in infants born before 30 weeks' gestation and the incidence of IVH is less than 5% after that time. In the present study, 93.9% preterms had abnormal findings on cranial ultrasound of which 17% had GMH. The maximum incidence of GMH 22.58% was found in preterms 33-36 weeks. Rehan N, Farooqui R et al concluded that frequency of IVH was found in 47.5% preterm neonates, 47.3%, 23.3%, 15.8% and 10.5% neonates had grade I, grade-II, grade-III and grade-IV intracranial bleeds respectively. De Vries LS, Eken P, Dubowitz LM et al, Batton DG, Holtrop P et al, Pape KE et al and Levene MI reported in their respective studies that CUS was 76% to 100% accurate in detecting grade 1 lesions of > 5 mm and grade 3 and grade 4 haemorrhages. Detection of grade 2 haemorrhages was much less accurate. In the present study, 18% of neonates had intracranial bleeds of which 21% had grade 2 haemorrhages and rest had grade 1 haemorrhages. Jeffrey M. Perlman, Nancy Rollins et al in their study found out that up to 50% of neonates weighing less than 1500 g exhibited some abnormality on the initial CUS. Severe IVH was observed in approximately 11% of the neonates weighing less than 1000 g and in 5% of those between 1000- and 1250-g BW. Cystic PVL was noted in 5% of the neonates weighing less than 1000-g and in approximately 1% of those between 1250 and 1500 g. In the present study, 14.9% of neonates weighing less than 1500g, 25.7% weighing between 1500 and 2000g, 20.3% weighing between 2000 and 2500g exhibited some abnormality on CUS. The overall incidence of abnormalities on CUS in low birth weight neonates in present study was 57.4%. Joseph J. Volpe, Inder TE, Anderson J et al 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 CUS of the VLBW infant demonstrates high reliability in the detection of cystic WM. Arti Maria, Arun Gupta et al reported that 36.2% of enrolled very low birth weight neonates developed various forms of PVL. In their study, about 50% of ultrasound had normalized at discharge and sequelae such as cerebral atrophy and 109 ventriculomegaly had appeared in few, the rest of lesions being either flares or cysts of PVL. They concluded CUS remains an important bedside diagnostic tool for PVL. In the present study, one preterm neonate on regular follow up CUS developed findings suggestive of cystic PVL and two neonates on regular follow up CUS had developed cystic encephalomalacia with hydrocephalus correlating well clinically. Teele RL, Hernanz-Shulman et al reported basal ganglia or thalamus hyperechogenicity (BGTH) in 0.3–2.5% of neonates and 5.1–32% of preterm infants screened by CUS. Leijser LM, Klein RH Meijler G et al in their study on preterm neonates concluded that diffuse homogeneous BGTH is a frequent and normal prematurity-related finding BGTH was seen in 92% preterm neonates and 8% of low-risk neonates. Soghier LM, Vegaet M et al reported diffuse BGTH occurred in 8.3% of preterm neonates studied by CUS and that incidence of diffuse BGTH was inversely related to GA. In the present study, 6% neonates had thalamus hyperechogenicity with 2% neonates having HIE findings by a neurosonogram. Of the neonates having thalamic hyperechogenecity 3% were preterms and 14.7% were term neonates.

Among the neonates having HIE on CUS, one each was of term and preterm gestation. Leijser LM, de Vries LS, Rutherford MA et al proved that neonatal CUS 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 CUS. In the present study, none of the neonates studied had CUS findings suggestive of metabolic disorder. Boo N, Chandran V, Zulfiqar M et al reported diffuse increase in echogenicity of the cerebral parenchyma was significantly more common in term infants with encephalopathy than in controls (39% versus 1%). Eken P, Toet MC, de Vries LS et al reported 26.4% full term neonates with HIE had areas of increased echogenicity in the periventricular and/or subcortical white matter. In the present study, of all neonates 6% had hyperechogenic thalami, 2% definite HIE, 4% had cerebral oedema. Of the 67% of neonates with abnormal findings on CUS 70.6% had no evidence of perinatal asphyxia, 57.14% had severe, 60% had moderate and 60% had mild perinatal asphyxia as per Apgar scoring. Hannah C. Glass, Sonia L. Bonifacio et al in their study reported that 3.8% preterm neonates had clinical seizures. CUS was abnormal in all these infants and was accurate for detecting IVH and PVHI. In this study, of all neonates presenting with seizures 73.9% had normal and 26.1% had abnormal CUS of which thalamic hyperechogenecity was most commonly detected (13%) by CUS. Nelson and Grether et al, Schendel et al, Michael O'Shea T et al studied prenatal factors like preeclampsia related to cranial ultrasound findings, neurodevelopmental outcome and incidence of cerebral palsy. Badrawy N et al reported that PROM and preeclampsia influenced the presence of CUS abnormalities and risk of developing periventricular intraventricular haemorrhage PIVH. Vermeulen GM, Bruinse HW, De Vries et al reported cranial ultrasound abnormalities in 13% of preterm neonates some of which had perinatal risk of PROM and proved early onset neonatal infectious disease is an independent risk factor for cranial ultrasound abnormalities. In the present study, those neonates with evidence of neonatal sepsis 83% had normal and 17% abnormal CUS findings of which 8.5% had thalamic hyperechogenecity. Also, 15.4% of neonates which had abnormal findings on CUS had PROM. Also, there was significant correlation with PIH with abnormal cranial ultrasound findings with 34.6% of neonates born to mothers with PIH having CUS findings. 15.4% neonates with PROM, 3.8% with multiple pregnancy and 7.7% with birth trauma had abnormal CUS findings. Van Houten JP, Rothman A et al151 reported that 59% infants with CHD had a higher incidence of cranial ultrasound abnormalities. In the present study, 34.6% of neonates with abnormal findings on CUS had CHD. Badrawy N et al reported that the neurological manifestations which had significant association with SE-IVH in were poor neonatal reflexes, seizures and apnoea. Miznahi et al, stated that in the presence of IVH there are often pallor, periods of apnoea, cyanosis failure to suck well, high pitched cry, muscle twitching and convulsions. In the present study, there was statistically significant correlation between abnormal cry, abnormal tone, abnormal activity, presence of cyanosis on clinical examination and of abnormalities on CUS. Nicaise C, Gire C, Fagianelli P et al reported CUS abnormalities in 11.7% of preterm born after preterm premature rupture of membranes (PPROM) and evaluated their outcome. The incidence of neonatal sepsis defined by the association of 2 positive bacterial cultures with a positive CRP was 15% in their study. In the present study, 47% of neonates had neonatal sepsis of which 30.8% had abnormal CUS findings. 28% of the enrolled neonates had positive CRP. Of the 67%, neonates which had abnormal CUS, 7.7% had positive CRP which was statistically significant correlation. 4% of neonates had positive blood cultures in the present study. Of the 67% neonates which had abnormal CUS, 7.7% had positive blood culture. Badrawy N et al reported that there was no statistically significant difference between the mean I/T ratio, mean HB, mean Ht, and mean Na among patients with SE-IVH compared to those without. Faix et al, proved that the occurrence of neutropenia before 14 days of age was not significantly different between the groups (50% with IVH, 56% without IVH). Comparison of immature neutrophil count and immature/total neutrophil ratio also revealed no differences. Levene et al in 1984, found that infants with extension of intraventricular haemorrhage were constantly more acidotic and more anaemic than the control group. In the present study, there was statistically significant correlation with neonates having positive CRP with abnormal CUS findings. There was no correlation of Hb, PCV, TLC, platelet count, retic and positive culture, serum electrolytes, serum bilirubin and CSF analysis with abnormal CUS findings. Badrawy N et al128 reported that 40% of neonates in their study did clinically well with total mortality of 30%. Amess PN et al proved that neonatal cranial ultrasound revealed a subcategory of infants that were at high risk of developing epilepsy. Karl Kuban et al prospective cohort study of extremely low gestational age newborns evaluated that half the children with ventriculomegaly or echolucency on CUS eventually developed cerebral palsy. Linda S. De Vries et al assessed sequential high-resolution cranial ultrasound (US) in high-risk preterm infants to predict cerebral palsy (CP) and in their study Seventy-nine percent of CP cases had major US abnormalities. Canadian Paediatric Society Statement in (2001) concluded that it is important to consider the value of determining the need for routine screening CUS examinations in NICUs. In the present study, of the 67% neonates having abnormal CUS, 61% recovered completely during their stay in NICU and 77.7% were discharged/DAMA before clinical recovery and 2% died. Follow up of these

neonates for neurodevelopmental outcome was not a part of this study.

## CONCLUSION

Neonatal care in India is advancing at an impressive phase at the level of the community as well as in tertiary care units. The concept of 'survival' of the newborn has predictably given way to the importance of 'intact survival' of the infant, prompting initiation of strategies to identify neurological sub-normality at the earliest. CUS is an ideal tool for the primary screening of the neonatal brain. Despite the wide availability of ultrasound machines in the hospitals, the penetration of CUS in Indian NICU's is still very little. This study highlights the convenience and diagnostic efficiency of cranial ultrasound in neonates in NICU. It also emphasizes its use as a screening modality for preterm neonates influencing their neurodevelopmental outcome. High efficacy of CUS in detecting presence of brain damage and its evolution on regular follow up guides clinical decisions and prognosis. This is particularly important in the anticipation of potential preventive, protective, and rehabilitative strategies for the management of critically ill newborn infants. Study concludes CUS is critical as an investigatory modality in NICU and effectively documents morphology of brain damage.

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**Declarations**

**Conflict of interest:** No

**Ethical approval:** Yes

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