

ROLE OF TRANSCRANIAL ULTRASOUND IN HOSPITALISED HIGH-RISK NEWBORNS : A PROSPECTIVE OBSERVATIONAL STUDY.

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Abstract

Introduction:

High-risk neonates are always at increased risk of morbidity and mortality. They contribute to as high as 15% of all morbidities and mortalities in newborns independently. Hence identifying them early and appropriate management of these high-risk newborns is important. Cranial ultrasonography (CUS) is a reliable tool for detecting congenital and acquired abnormalities of the perinatal brain and the most frequent patterns of brain injury in preterm and full-term neonate.

Aim and objective:

To study cranial ultrasound findings in high-risk neonates and to correlate the clinical manifestations with cranial ultrasonogram findings.

Material & Method:

This study was a prospective observational study carried out at the Department of Paediatrics, SVPPGIP & SCBMCH between October 2019 to October 2021.

Observation:

The incidence of CUS abnormalities in high-risk neonates in the present study was 54.5%.

There was a statistically significant correlation between birth asphyxia and abnormal CUS findings ($p < 0.001$), birth trauma and abnormal CUS findings ($p < 0.001$), hypoglycaemia and abnormal CUS findings ($p = 0.004$), and abnormal Central Nervous System examination and abnormal CUS findings ($p = 0.008$).

Conclusion:

Cranial ultrasound is important and critical.

Recommendation :

The concept of 'survival' of the newborn has given way to the importance of 'intact survival' of the high-risk infant, prompting the initiation of strategies to identify neurological sub-normality at the earliest. CUS is an ideal tool for the primary screening of the neonatal brain, it must be done in all high-risk neonates for prognosticate and guide to outcome

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1. Introduction:

Any neonate, regardless of birth weight, size, or gestational age, who has a greater than aver-

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age chance of morbidity or mortality, due to fetal, maternal, or placental anomalies or an otherwise compromised pregnancy, especially within the first 28 days of life is categorized as high-risk neonate[1]. Cranial ultrasonography (CUS), a non-invasive and bedside tool, plays an important role in assessing neurological prognosis of these high-risk infants. The term high-risk infant designates an infant who should be under close observation by experienced pediatricians and nursing staff. Approximately 10-20% of all births require special or neonatal intensive care. Usually needed for only a few days, such care may last from a few hours to several months. These high-risk neonates should be identified as soon as after delivery, even before delivery during routine checks to decrease neonatal morbidity and mortality [2]. Neonatal care in India is advancing at an impressive pace. The government has implemented different programs for safe mothers and childhood. The concept of “survival” of newborns has given way to “intact survival” of high-risk newborns, emphasizing the detection of neurological abnormalities at the earliest [3]. 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 fontanelles are still open and these can be used as acoustic windows to “look” into the brain[4]. CUS is a simple, non-invasive investigation that can be initiated at a very early stage, even immediately after birth. It can be repeated as often as necessary and thereby enables visualization of ongoing brain maturation and the evolution of brain lesions. In addition, it can be used to assess the timing of brain damage, both physical and functional [4]. 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 the most frequent patterns of brain injury in the preterm and full-term neonate. It detects most of the hemorrhagic, ischemic, and cystic brain lesions as well as calcifications, cerebral infections, maturity, and major structural abnormalities in preterm and full-term infants[4]. Many aetiolo-

gies of neonatal encephalopathy and seizures in the term infant and the subsequent monitoring of the progress of hypoxic-ischemic brain injury can be diagnosed by CUS. In seriously ill neonates and neonates with serious cerebral abnormalities, either congenital or acquired, it plays a role in decisions on intensive treatment. In neonates surviving cerebral injury, it may help to optimize the treatment of the infant both during the neonatal period and thereafter[5]. Serial CUS examinations enable assessment of the onset of injury and the evolution of lesions[1]. Most newborn intensive care unit centers perform serial cranial ultrasound evaluations early in the course of hospitalization for premature infants and a follow-up examination is done at a later age. These evaluations are done to document the presence of intracranial hemorrhage, to guide the choice of therapies that may exacerbate the risk of further hemorrhage, and to counsel families about neurodevelopment outcomes [6].

CUS has an important role in the diagnosis of significant lesions in infants presenting with hypoxic ischemic encephalopathy (HIE) and seizures in full-term infants. 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 is very helpful in assessing the severity and neurodevelopment outcomes in infants with HIE. It is also important to realize that the end stages of hypoxic-ischemic brain damage may not become visible until a variable period, often several weeks to months after the event, and that the early stages may seem mild or subtle. It is therefore advisable to repeat CUS examinations until normalization or stabilization of abnormalities in cases of (suspected) ischemic injury, even if mild[7]. It is reliable for detecting common markers of metabolic disease in neonates, germinolytic cysts, lenticulostriate vasculopathy, basal ganglia calcification, subtle white matter abnormalities, and cortical and other structural abnormalities[8]. The quality of CUS imaging and its diagnostic accuracy depends on the suitability of the ultrasound machine for neonates, and the experience and expertise of the examiner(9). Modern ultra-

sound machines and probes and the use of a variety of acoustic windows and adequate scanning protocols give high-quality images that are diagnostically accurate [9].

1.1. AIM:

To assess the importance of cranial ultrasound as an investigation modality for high-risk neonate.

1.2. Objectives:

To study cranial ultrasound findings in high-risk neonates, correlate the clinical manifestations with cranial ultra sonogram findings in high-risk neonates.

2. Materials & Methods:

This was a prospective observational study conducted in neonates (≤ 28 days) admitted in SCBMCH & SVPPGIP, CUTTACK, Odisha, a premier tertiary institute of eastern India, where neonates from the state of Odisha as well as adjacent state of Jharkhand, Chhatishgarh & West Bengal used to be treated. The study was carried out from October 2019 to October 2021 after getting clearance from the institutional ethical committee. After being explained in local understandable language the plan for the study of all the parents of eligible neonates, the consent form was signed. The data were collected in an Excel sheet and analyzed.

2.1. Sample Size:

The sample size was calculated using Fischer's Formula.

FISCHER'S FORMULA: $N = Z^2 \times P(1-P)/d^2$

N = The desired sample size, Z= The value representing a 95% Confidence interval, d=precision of the study or absolute error fixed at 5%, P=Prevalence (based on a study done in a developing country like India previously).

2.2. Inclusion Criteria:

1. Premature rupture of membranes for > 24 hours, 2. PIH, APH, Multiple pregnancies, Gestational Diabetes, Instrumental-delivery, 3. Birth asphyxia, Neonatal seizures, Preterm neonates, Respiratory distress, Small for Gestational Age (SGA) / Large for Gestational Age (LGA), 4. Low birth weight, Metabolic disturbances with abnormal neurological manifestation.

2.3. Exclusion Criteria:

1. Patients who were discharged against medical advice, 2. Patients who get transferred to other departments before the treatment ended.

As the inclusion and exclusion criteria were clear, there was no bias in collecting data.

2.4. Statistical Methods:

Categorical variables are expressed as the Number of patients and percentage of patients and compared across the groups using Pearson's Chi Square test for Independence of Attributes/ Fisher's Exact Test as appropriate. Continuous variables are expressed as Mean, Median, and Standard Deviation and compared across the groups using the Mann-Whitney U test. The statistical software SPSS version 22 has been used for the analysis. An alpha level of 5% has been taken, i.e. if any p value is less than 0.05 it has been considered significant.

2.5. Observation:

All the data obtained from the study were recorded, tabulated, and analyzed. A total of 110 neonates were taken into the study who fulfilled the inclusion and exclusion criteria.

In the study group 59.1% were male (n=65) and 40.9% were female (n=45), 26.4% were preterm neonates (n=29) and 73.6% were term neonates (n=81). The majority were term neonates (n=81, 73.6%) followed by preterm neonates of 28-32 weeks (n=17, 15.5%) and 32-37 weeks (n=12, 10.9%). 65.5% (n=72) of the study group were born through vaginal delivery and 34.5% (n=28) were born through LSCS. 48.2% (N=53) of neonates were with birth weight more than 2500 grams, 30.9% (n=34) with birth weight

Table 1: Demographic Distribution of study groups

Parameter		Frequency	Percentage
Sex	Male	65	59.1
	Female	45	40.9
Gestational age	Preterm	29	26.4
	Term	81	73.6
Gestational week	28-32	17	15.5
	32-37	12	10.9
	37-40	45	40.9
	>40	36	32.7
Mode of delivery	Vaginal	72	65.5
	LSCS	38	34.5
Parity	Primi gravid	58	52.9
	Multi gravid	20	18.1
	Other	32	29
Birth weight	<1.5Kg	23	20.9
	1.5-2.5Kg	34	30.9
	>2.5Kg	53	48.2

between 1500-2500grams and 20.9% with birth weight <1500grams.

PROM was the major perinatal risk factor accounting for 8.18% (n=9), followed by PIH (5.45%,n=9) and multiple pregnancies (5%,n=5). The major neonatal risk factors were birth asphyxia (n=70,63.64%), seizures (n=69,62.73%), sepsis (n=43,39.09%), respiratory distress (n=40,36.36%) and prematurity (n=26, 23.64%).

There were 34 (31.8%) with abnormal activity, 13(12.4%) with an abnormal cry, 10 (8.8%) with poor reflexes, 8(7.1%) with abnormal tone, and 3(2.9%) with abnormal pupillary reaction. There were 43(39.09%) with positive CRP , 20(18.18%) with hyponatremia, 15(13.64%) with hypoglycemia and 14(12.7%) with positive blood culture.99(90%) neonates recovered and discharged, 10(9.1%) neonates died and 1(0.9%) baby left against medical advice. Cranial ultrasonography showed abnormalities in 60(54.5%) high-risk neonates.

Of the 60 neonates with abnormalities in CUS, 38(63.33%) were male and 22(36.67%) were female neonates. There was no statistically significant correlation of abnormal CUS findings with

gender (p=0.322). Also, gestational week, mode of delivery, and birth weight were not statistically significant(p=0.333) as per CUS is concerned. Of the 60 neonates with abnormal CUS, Perinatal risk factors were PIH in 6(10%), PROM in 2(3.33%), and multiple pregnancies in 1(1.67%). There was a statistically significant correlation between PIH and abnormal cranial ultrasound findings(p=0.021)and also between PROM and abnormal cranial ultrasound findings(p=0.042). There was a statistically significant co-relation between abnormal CUS findings and birth asphyxia (p<0.001), birth trauma (p=<0.001), hypoglycemia (p=0.004), and abnormal CNS examination (p=0.008). There was no statistically significant correlation between abnormal CUS findings and sepsis (p=0.080). There was a definite significant relation between abnormal CUS and hypoglycemia, positive CRP, and hyponatremia statistically.

There was statistically significant relation between abnormal CUS and laboratory findings of mean, median, standard deviation of hemoglobin, total leucocytes count and total platelet count.

Major CUS abnormalities found in the study group were intra-ventricular hemor-

Table 2: Perinatal & neonatal risk factors

Parameter		Frequency	Percentage
Perinatal risk score	FNOM	9	8.18
	FIII	6	5.45
	Multiple pregnancy	5	4.55
	Birth asphyxia	70	63.64
	Seizures	69	62.73
Neonatal risk factor	Sepsis	43	39.09
	Respiratory distress	40	36.36
	Prematurity	26	23.64
	Hypoglycemia	15	13.64
	Birth trauma	15	13.64
	Other organ anomalies	7	6.36

Table 3: Clinical , Laboratory finding & Outcome of study group

Parameter		Frequency	Percentage
Abnormal CNS Finding	Poor activity	34	31.8
	Abnormal activity	13	12.4
	Poor reflexes	10	8.8
	Poor /abnormal tone	8	7.1
	Abnormal pupils	3	2.9
Lab findings	Hypoglycemia	15	13.64
	Positive CRP	43	39.09
	Hyponatremia	20	18.18
Cranial ultrasound	Blood C&S positive	14	12.7
	Normal	50	45.5
Outcome	Abnormal	60	54.5
	Discharge	99	90.0
	Death	10	9.1
	LAMA	1	0.9

rhage(n=12,20%), followed by cerebral edema, sub galeal hemorrhage, and periventricular hemorrhage each corresponding to a frequency of 9(15%), then cephalohematoma (n=7,11.67%), caudothalamic groove hemorrhage (n=5, 8.3%) and other findings which included arachnoid cyst, dandy-walker malformation and encephalocele which was n=9(10%).

3. Discussion:

The incidence of CUS abnormalities in high-risk neonates in the present study is 54.5%. Ayala Gover et al[10]and Eugenio Mercuri et

al[11]reported an incidence of CUS abnormalities in 11.2% and 20% respectively in apparently well neonates. Niranjana et al[12], and Ruchi Jha[13], reported an incidence of CUS abnormalities of 16.1% and 25.4% respectively in their study on preterm neonates. In the present study, out of 110 neonates, there were 59.1%(n=65) male and 40.9%(n=45) female neonates. Abnormal CUS was found in 38 (63.3%) male and 22(36.67%) female neonates. There was no statistically significant correlation of abnormal CUS findings with gender (p value=0.322). Niranjana et al[12], in their study, had 62.9%% male and 37.1%% fe-

Table 4: Distribution of different parameters of the study group with abnormal CUS findings

Parameter	Abnormal cranial USG finding				p-value	Significance	
	No	Yes	Frequency	Percentage			
Sex	Male	27	54	38	63.33	0.222	Not significant
	Female	23	46		36.67		
Gestational week	28-32	10	20	7	11.67	0.222	Not significant
	32-37	6	12	6	10		
	37-40	16	32	29	48.3		
Mode of delivery	>40	18	36	18	30	0.770	Not significant
	V D	32	64	40	66.67		
Birth weight	LSCS	18	36	20	33.33	0.387	Not significant
	<1.5Kg	13	26	10	16.67		
	1.5-2.5Kg	16	32	18	30		
Perinatal risk factor	>2.5Kg	21	42	32	53.33	0.021	Significant
	UTI	0	0.00	6	10.00		
Neonatal high risk factors	ROM	7	14	2	3.33	0.042	Significant
	Multiple pregnancy	4	8	1	1.67	0.112	Not significant
	Birth asphyxia	22	44.00	48	80.00	<0.001	Significant
	Seizures	28	56.00	41	68.33	0.183	Not significant
	Sepsis	24	48.00	19	31.67	0.080	Not significant
	Respiratory distress	20	40.00	20	33.33	0.469	Not significant
	Prematurity	13	26.00	13	21.67	0.594	Not significant
	Hypoglycemia	12	24.00	3	5.00	0.004	Significant
	Birth trauma	0	0.00	15	25.00	<0.001	Significant
	Other organ anomalies	4	8.00	3	5.00	0.521	Not significant
Laboratory findings	Abnormal CNS examination	15	30.00	33	55.00	0.008	Significant
	Hypoglycemia	12	24.00	3	5.00	0.004	Significant
	Positive CRP	28	56.00	13	25.00	0.001	Significant
	Blood C&S	8	16.00	6	66.67	0.769	Not significant
Outcome	Hyponatremia	5	10.00	15	25.00	0.042	Significant
	Discharge	48	96	51	85	0.149	Not Significant
	Death	2	4	8	13.33		
	LAMA	0	0	1	1.67		cant

Table 5: Correlation between laboratory findings and abnormal CUS findings

Parameter	Abnormal cranial USG finding						p-value	significance
	No			Yes				
	Mean	Median	SD	Mean	Median	SD		
Hemoglobin	15.94	16.10	3.78	14.72	13.65	2.93	0.025	Significant
TLC	22615	13775	96641.52	15789.43	15680	5369.58	0.012	Significant
TPC	155600	150000	55868.29	223422.33	180500	129916.84	0.005	Significant

Table 6: Various cranial ultrasound findings in the study group

Cranial ultrasound findings	Frequency	Percentage
Intra-ventricular hemorrhage	12	20.00
Hydrocephalus	6	10.00
Cerebral edema	9	15.00
Sub Galeal Hemorrhage	9	15.00
Cephalhematoma	7	11.67
Caudothalamic groove	5	8.3
Periventricular leucomalacia	9	15.00
Others	9	15.00

male neonates. There was no significant correlation between the incidence of abnormal cranial ultrasound findings in males and females.

In the present study, there were 26.4% (n=29) preterm and 73.6% (n=81) term neonates. Of those neonates with abnormal CUS findings, 11.6% (n=7) were less than 32 weeks, 10% (n=6) between 32-36 weeks and 48.33% (n=29) were between 37-40 weeks and 32.73% (18) were more than 40 weeks. Badrawy N et al[14] showed in their study that the preterm had 37% abnormal CUS findings. Of these, 9% were less than 30 weeks, 33% were between 30-33 weeks and 58% were between 34 and 37 weeks.

In the present study, 65.5% (n=72) of the study group were born through vaginal delivery, and 34.5% (n=28) were born through LSCS. 66.67% (n=40) neonates were born through normal delivery and 33.33% (n=20) born through LSCS had abnormal CUS findings. There was no statistical significance between USG findings and the mode of delivery in the present study. (p value=0.770)

In the present study, the mean birth weight of all the high-risk neonates was 2443.6 grams.

The mean birth weight of neonates with abnormal CUS (2477.5 grams) was more than those with normal CUS (2218 grams) which was not statistically significant (p=0.024). 10 (16.67%) neonates with birth weight <1500grams, 18 (30%) with birth weight 1500-2500grams, and 53 (48.18%) with birth weight 2500-4000grams had abnormal CUS findings. Jeffrey M. Perlman et al[15] in their study found out that up to 50% of neonates weighing less than 1500 g exhibited some abnormality on the initial CUS. Abnormal CUS findings were 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 1500g.

In the present study, Of the 60 neonates with abnormal CUS, perinatal risk factors were PIH in 6 (10%), PROM in 2 (3.33%), and multiple pregnancies in 1 (1.67%). There was a statistically significant correlation between PIH and abnormal cranial ultrasound findings (p=0.021) and also between PROM and abnormal cranial ultrasound findings (p=0.042). Nelson et al[16], Schendel et

al[17], and Michael O'Shea T et al[18] studied prenatal factors like preeclampsia related to cranial ultrasound findings and observed that preeclampsia was associated with abnormal CUS findings, neurodevelopmental outcome, and incidence of cerebral palsy. Badrawy N et al[14] reported that PROM, PIH, gestational diabetes, and maternal fever had a significant correlation with the presence of CUS abnormalities. Vermeulen GM et al[19] reported that PROM is a significant risk factor for cranial ultrasound abnormalities.

In the present study, 39.09% of high-risk neonates had neonatal sepsis of which 31.67% had abnormal CUS findings. There was no significant correlation between sepsis and abnormal CUS findings ($p=0.080$). Niranjana N et al[12], in their study, found that, of 16.1% of neonates who had abnormal CUS findings, 5.2% had sepsis which was statistically significant.

In the present study, 62.73% ($n=69$) of the neonates presented with seizures, of them 59.4% ($n=41$) had abnormal CUS findings. Among these, 24.39% ($n=10$) had IVH, 21.95% ($n=9$) had subgaleal hemorrhage and 21.95% ($n=9$) had cerebral edema. Hannah C et al[20] in their study reported that 3.8% of preterm neonates had clinical seizures. CUS was abnormal in all (100%) of these infants and was accurate for detecting IVH and PVH. Niranjana N et al[12], in their study, found that of all high-risk neonates presenting with seizures 47.6% had normal and 52.4% had abnormal CUS.

In the present study, there were 70 neonates with birth asphyxia, of these 48 (68.5%) had abnormal CUS findings. Eken P et al[21] reported 26.4% full-term neonates with HIE had areas of increased echogenicity in the periventricular and/or subcortical white matter and Boo N et al[22] reported abnormal cranial ultrasound changes in 79% of neonates with birth asphyxia.

In the present study, there was a significant correlation between laboratory findings like Hb, TPC, TLC, Hyponatremia, positive CRP, and the presence of abnormalities on cranial ultrasound. Badrawy N et al[14] reported that there was no statistically significant difference between the mean I/T ratio, mean HB, mean Ht, and

mean Na among patients with -IVH compared to those without. Levene et al[23], found that anemic neonates had a correlation with the extension of intraventricular hemorrhage. Niranjana et al[12] in their study, found that there was a statistically significant correlation between neonates having positive CRP, and low platelet with abnormal CUS findings. There was no correlation of Hb, PCV, TLC, reticulocyte count, and positive culture and serum electrolytes with abnormal CUS findings

In the present study, there was a statistically significant correlation between abnormal CUS and abnormal CNS examination ($p=0.008$). Ruchi Jha et al[13], in their study found that

poor activity (43.7%, $p=0.03$) and abnormal tone (57.1%, $p=0.03$) had a significant correlation with abnormal CUS findings. Badrawy N et al[14] reported that there was a significant association between poor neonatal reflexes and apnea with abnormal USG findings. Miznahi et al[24], stated that in the presence of IVH, there is often apnea, cyanosis failure to suck well, abnormal cry, and convulsions.

In the present study on CUS, IVH ($n=12$, 20%), followed by cerebral oedema, sub galeal hemorrhage, and periventricular haemorrhage each corresponding to the frequency of 9 (15%), then cephalohematoma ($n=7$, 11.67%), hydrocephalus ($n=6$, 10%), caudothalamic groove haemorrhage ($n=5$, 8.3%) and other findings which included arachnoid cyst, dandy walker malformation and occipital encephalocele which was $n=9$ (10%). Eugenio Mercuri et al[11] reported ischemic lesions, such as periventricular and thalamic densities as the most common finding (8%), followed by intracranial hemorrhagic lesions (6%) on CUS. Badrawy N. et al[14] reported that subependymal intraventricular hemorrhage (SE-IVH) was present in 14%, brain edema in 9%, hypoxic ischemic changes in 4%, post-hemorrhagic hydrocephalus (PHH) in 3.5% as a complication of SE-IVH. Niranjana et al[12], found in a study that 11.2% of had evidence of intracranial bleeding, 1.6% periventricular echogenicity, 1.6% had ventriculomegaly and 1.6% had peri-ventricular leukomalacia. Ruchi Jha[13], in their study on

preterm, reported 8% had IVH, 10.6% had periventricular hyper echogenicity, 4% had PVL and 2.6% had cerebral edema. Badrawy N et al[14], reported intraventricular hemorrhage in 14% of preterm neonates. Niranjana et al[12], reported that of the high-risk neonates with preterm gestation, 83.9% had normal and 17.7% had abnormal CUS. Correlation between CUS findings of neonates with prematurity was statistically significant ($p=0.015$) and 11.2% of had evidence of intracranial bleeding. Batton DG et al[25], Harding D et al[26], and Paneth N et al[27] in their studies concluded that the maximum risk of IVH in infants born before 30 weeks gestation is 20%, 23%, and 24.6% respectively and the incidence of IVH is less than 5% after that time. Chawdhury V et al[28] in their study on 50 preterm neonates, detected intracranial pathology in 12% of preterms and 6% of these had intracranial hemorrhage.

Niranjana et al[12], in their study, reported that of the 10 neonates with abnormal CUS findings, 2(20%) had HIE changes. Joseph J et al[29] in their study detected CUS abnormalities in 56% VLBW neonates, of these 60.7% of 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 changes. Arti Maria et al[30] 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 ventriculomegaly had appeared in few, the rest of the lesions being either flares or cysts of PVL. They concluded CUS remains an important bedside diagnostic tool for PVL.

In the present study, of the 60 neonates with abnormal CUS, 10(23.8%) had thalamic hyper echogenicity in neurosonogram of which 4 (26.67%) were preterm. Soghier LM et al[31] reported diffuse BGTH occurred in 8.3% of preterm neonates studied by CUS and that the incidence of diffuse BGTH was inversely related to GA. Teele RL et al[32] reported basal

ganglia or thalamus hyper echogenicity (BGTH) in 5% of neonates and 12% of preterm infants screened by CUS. Leijser LM et al[33] in their study on preterm neonates concluded that diffuse homogeneous BGTH is a frequent and normal prematurity-related finding BGTH was seen in 9.2% of preterm neonates and 8% of term neonates

In the present study, of the neonates with abnormal CUS (60), 9(15%) had cerebral edema. Both had evidence of moderate birth asphyxia. Ruchi et al[13], in their study, found that of the 19 preterm neonates with abnormal CUS, 2(10.5%) had evidence of cerebral edema.

In the present study, 6 neonates (10%) had hydrocephalus which included post-hemorrhagic ventricular dilation and congenital hydrocephalus. Badrawy N et al[14] reported congenital hydrocephalus to be present in 6% of all neonates screened by them. Soni JP et al[34] suggested from their study that CUS is sensitive and specific for the identification of various types of Intracranial Hemorrhages (SEH, IVH, PVL). 111 high-risk neonates were subjected to CUS, one-quarter of these neonates developed intracranial hemorrhage (ICH) within 120 hours of birth.

In the present study, of the neonates with abnormal CUS, 90% are discharged and 9.1% of neonates died. Badrawy N et al[14] reported that 40% of neonates in their study did clinically well with total mortality of 30%. Niranjana et al[12], found that 61.2% of neonates had cured at the time of NICU discharge, 12.9% died and 17.7% of neonates were relieved at the time of NICU discharge, 8.06% were discharged from NICU for various reasons before clinical recovery (DAMA). Ruchi Jha [13], in their study, found that out of 21.4 % who had expired in their study, 45.4% had abnormal findings on CUS.

4. Conclusion:

Care in India is advancing at an impressive pace both at the community level as well as in tertiary care units. The concept of 'survival' of the newborn has given way to the importance of 'intact survival' of the high-risk infant, prompt-

ing the 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 the NICU's of India is still very little. This study highlights the convenience and diagnostic efficiency of cranial ultrasound in high-risk neonates. There was a statistically significant correlation between CUS abnormalities and perinatal risk factors like PIH and PROM, neonatal risk factors, clinical features, and laboratory parameters. The study concludes CUS is critical as an investigatory modality and effectively documents morphology of brain damage.

5. Limitations:

Expert radiologist needed to focus the point of interest for the detection of abnormality present in the neonatal brain.

6. Recommendations:

Cranial Ultrasound is a non-invasive bedside investigation. High-resolution probes and other modalities detect the degree of brain damage in high-risk neonates accurately. Serial cranial ultrasound is recommended (even multiple times if needed) to measure and guide treatment in all high-risk infants.

7. Acknowledgement:

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8. Author's Contribution:

All authors were involved in research design, data analysis, and manuscript preparation and editing.

9. Disclosure:

The authors report no conflicts of interest in this work.

10. Source of funding:

None

11. List of abbreviation :

BGTH : Basal Ganglia Thalamus Hyperechogenicity
CRP : C-Reactive Protein
CNS : Central Nervous System
CUS: Cranial Ultrasonography
DC: Differential Count
DAMA : Discharge Against Medical Advice
Hb : Haemoglobin
HIE : Hypoxic Ischemic Encephalopathy
IVH : Intra Ventricular Haemorrhage
LSCS : Lower Segment Caesarean Section
NICU : Neonatal Intensive Care Unit
PCV : Packed Cell Volume
PROM: Premature Rupture of Membrane
PIH: Pregnancy Induced Hypertention.
PHH: Post –Hemorrhagic Hydrocephalus
PVL : Periventricular leucomalacia
SEH : Sub ependymal Haemorrhage
TLC : Total Leucocytes Count
VD : Vaginal Delivery
VLBW: Very Low Birth Weight

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