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Research Article

Early Cranial Ultrasound Findings in Preterm Infants Under 37 Weeks of Gestational Age. Retrospectively Evaluated Experience of a Single Neonatal Intensive Care Unit and Review of the Literature

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Abstract

Background: Cranial ultrasound (CUS) is currently recommended between 7 and 14 days of life for infants born before 30 weeks of gestational age (GA). It provides information about possible perinatal brain injury and helps in predicting long-term outcomes. Brain damage occasionally begins already in utero. This retrospective study was designed to evaluate whether early CUS screening is a useful technique providing important information when performed for a broader population of premature infants.

Objective: To evaluate CUS findings in premature infants born before 37 weeks of GA for whom CUS was performed during the first day of life.

Method: A total of 365 premature infants were enrolled in the study. CUS and clinical data were retrospectively collected from the intensive care electronic database.

Results: Day 1 CUS showed pathological findings for 141 infants (38.6%). Intraventricular Hemorrhage (IVH) was demonstrated in 58 infants (15.9% of the total study population). Signs of intraventricular clot liquefaction suggesting in utero IVH were demonstrated in 31 cases out of a total of 58 with IVH (53%). In 14 cases of day 1 CUS showing IVH (24.1%), subsequent CUS was normal. 49 infants (12.6% of total 365) had white matter hyper-echogenic lesions.

Conclusions: An addition to common CUS practices, we propose considering CUS on the first day of life as a means to rule out brain injury which originates in utero, and suggest the use of CUS for a wider population of premature infants born beyond 30 weeks of GA.

ABBREVIATIONS

CUS: Cranial Ultrasound; GA: Gestational age; IVH: Intra-ventricular Hemorrhage; PVL: Periventricular Leukomalacia; WM: White matter; NICU: Neonatal Intensive Care Unit

INTRODUCTION

Cranial ultrasound (CUS) is the most commonly used technique for serial bedside neonatal brain imaging. It provides information about possible perinatal and postnatal brain injury and helps in predicting long-term outcomes [1-3]. Considering the high risk of brain injury in the most fragile premature babies, routine CUS is currently recommended for infants born before 30 weeks of gestational age (GA) (4). However, moderate and late preterm infants can also suffer from significant or subtle brain lesions that may influence their neurodevelopmental outcomes [5-8].

Brain damage occasionally begins already in utero during crucial phases of brain development, and it is important to consider the timing of brain injury [9-13].

This retrospective study was designed to recognize the prevalence of CUS findings during the first day of life in premature infants and to evaluate the usefulness of early CUS screening in preterm infants born after 30 weeks of gestation.

METHODS

On receiving the Ethical Committee approval, preterm infants admitted to Carmel Medical Center Neonatal Intensive Care Unit (NICU) during the period June 2014 - July 2020 for whom CUS was performed during the first day after birth were identified retrospectively. Infants younger than 24 weeks of GA were excluded from the study. The decision to exclude the most extremely premature infants from the study was based on Israeli

Neonatology Association and Israel Obstetrics and Gynecology Association policy, in accordance to which infants younger than 23 weeks +6 days are under the threshold of viability. In the middle of 2020, this threshold policy was changed for 22 weeks +6 days (14), however, it was after the inclusion of all participants in our study.

CUS reports as well as perinatal and clinical data were retrospectively retrieved from the intensive care electronic database (Metavision, *i*MD*Soft*) and analyzed. In our medical institution, CUS screening was performed using ZONARE Z.One PRO: ZS3 ultrasound system, USA and transducer 7.5-MHz. The procedure was carried out by a senior neonatologist. The anterior fontanel was used as the principal acoustic window, and the scanning procedures included six standard coronal and five standard sagittal planes [1,15-17]. The second opinion was provided by another specially trained senior neonatologist.

Statistical analysis was performed using IBM SPSS Statistics 24.0 (IBM, New York, NY). The continuous variables are presented by mean, median and standard values. The categorical variables are presented in percentage. Abnormal CUS findings were compared between two groups of GA (24-30 weeks of GA versus 30.1-36.6 weeks of GA) using the Chi square or Fisher exact test, as appropriate. Bonferroni correction was used for findings including more than two groups. Changes in findings between the day 1 CUS and a later CUS were analyzed using the McNemar test. Factors associated with abnormal CUS findings on first day were identified using the logistic regression. OR with 95% CI are presented. Variables with $p<0.1$ at the univariate analysis were introduced into a multivariate model. $P<0.05$ was considered statistically significant.

RESULTS

A total of 365 patients were screened by means of CUS during the first day of life and met the inclusion criteria for the study. There were 213 (58.4%) male and 152 (41.6%) female infants. Abnormal day 1 CUS findings were found in 88 (41.3%) and in 53 (34.9%) infants, respectively (p=0.213).

Data on pregnancy and participant data at birth and during hospitalization are presented in Table 1.

Due to the relatively limited number of participants for statistical analysis, the study population was divided into two GA groups. One group included infants born from 24 to 30 weeks and the other group included infants born from 30.1 weeks to 36.6 weeks of GA (combined data for 30.1-33.6 and 34-36.6 GA groups). The data was compared between these two groups. When possible, the differences between all the three GA groups were taken in account (24-30, 30.1-33.6 and 34-36.6 weeks of GA) (Table 2).

Out of the total study population, 58 infants (15.9%) demonstrated the presence of IVH on day 1 CUS: 23 (33.3%) infants from 24-30 week GA group (N=69) and 35 (11%) infants from 30.1-36.6 week GA group (N=296) (p=0.0001). Regarding 167 infants from 34-36.6 week GA group, IVH was demonstrated in 16 (9.6%) infants.

Out of 23 infants from 24-30 week GA group with IVH demonstrated on day 1, as many as four (5.8%) were diagnosed with IVH grade 3-4, but no infant had severe IVH in 30.1-36.6 week GA group (p=0.0001).

Out of the total of 58 infants in whom IVH was demonstrated on day 1 CUS , in 14 (15.7%) patients no abnormalities were seen on subsequent CUS on days of life 5-7 and/or on days of life 10-14. Additionally, 89 participants (25% of total study population) were diagnosed with IVH later than on day 1 of life.

Among 58 infants with IVH on day 1, 31 participants (53%) of all GA groups demonstrated signs of clot liquefaction in the ventricles suggesting that IVH had formed at least a few days before the birth (Figure 1A,B). Out of 23 infants born at 24-30 weeks of GA with IVH on day 1 CUS, nine (39%) showed signs of resolving IVH. Among 35 participants from 30.1-36.6 week GA group with IVH on day 1 CUS, 22 (63%) already had intraventricular clot liquefaction signs (p=0.120).

A total of 49 participants (12.6%) had white matter hyperechogenic and/or cystic lesions as demonstrated on day 1 CUS. These lesions were shown in 25 (36.2%) infants in 24-30 week GA group (total of 69 infants), as compared to 23 (17.8%) infants in 30.1-33.6 week GA group (total of 129 infants) (p=0.004). One (0.6%) infant in 34-36.6 week GA group had white matter lesions (total of 167 infants). Concerning white matter lesions, statistically significant differences were demonstrated between 24-30 week GA group and 34-36.6 week GA group (p<0.0001) as well as between 30.1-33.6 week GA group and 34-36.6 week GA group (p=0.0001), with more findings in the younger GA group.

22 (44.9%) infants out of 49 infants with white matter hyperechogenic lesions on day 1 CUS had normal CUS on days 5-7 and/or days 10-14 of life, which means the echodensities were transient.

However, 27 (55.1%) participants (16 from 24-30 week GA group, 10 from 30.1-33.6 week GA group, and one from 34-36.6 week GA group) continued to show signs of periventricular leukomalacia (PVL) as white matter hyperechogenic lesions on days 5-7 and/or days 10-14 of life. Two of them (7.4%) had cystic lesions already on day 1 CUS. In addition, five (18.5%) infants with white matter hyperechogenicity diagnosed on day 1 CUS developed cystic lesions later. Seven (25.9%) infants with not resolved white matter hyperechogenicity demonstrated cystic PVL.

Out of all 365 study participants, 316 had normal white matter appearance on day 1 CUS. Eight (2.5%) of them showed signs of white matter abnormalities later on days 5-7 and/or days 10-14 of life.

Day 1 CUS also demonstrated other findings with different clinical significance. Out of total 141 infants with day 1 CUS findings, 20 (14.2%) had mild asymmetry of lateral ventricles and

Table 1: Day 1 CUS and participant data.

	Cranial Ultrasound on Day 1		OR 95% CI	p-value
	Normal N=224	**Abnormal N=141**		
Pregnancy				
Placenta abruption/placenta previa	30 (57.7)	22 (42.3)	1.19 (0.66-2.17)	0.557
Preeclampsic toxemia	43 (63.2)	25 (36.8)	0.91 (0.53-1.6)	0.726
Intrauterine growth retardation	46 (59.7)	31 (40.3)	1.09 (0.65-1.8)	0.741
Premature uterine contractions	128 (62.1)	78 (37.9)	0.93 (0.61-1.4)	0.732
Chorioamnionitis	6 (27.3)	16 (72.7)	0.44 (0.3-0.64)	0.002
Cerclage /Cervical insufficiency	6 (35.3)	11 (64.7)	3.07 (1.1-8.5)	0.031
GDM*/IDDM**/ NIDDM***	21(52.5)	19(47.5)	1.5(0.78-2.9)	0.224
Prenatal Bethamethasone	118(57.6)	87(42.4)	1.45(0.94-2.2)	0.091
Delivery mode				
Vaginal	86 (64.7)	47 (35.3)	ref	
Cesarean section	138 (59.5)	94 (40.5)	1.25 (0.80-1.9)	0.328
Newborn infants characteristics				
Gestational age 24-30 weeks	25 (36.2)	44(63.8)	4.9 (2.7-8.96)	<0.0001
Gestational age 30.1-33.6 weeks	76(58.9)	53(41.1)	1.95 (1.19-3.19)	0.008
Gestational age 34-36.6 weeks	123(73.7)	44 (26.3)	0.23 (0.12-0.46)	<0.0001
			Ref	
Birth Weight (grams)	1900±0.55	1700±0.60	0.44 (0.3-0.64)	<0.0001
Delayed cord clamping	37 (52.9)	33 (47.1)	1.54 (0.91-2.6)	0.105
Intubation	48 (45.7)	57 (54.3)	2.5 (1.6-4.0)	<0.0001
Surfactant supplementation	53 (49.1)	55 (50.9)	2.06 (1.3-3.3)	0.002

**GDM – gestational diabetes mellitus, **IDDM – insulin dependent diabetes mellitus, ***NIDDM – non-insulin dependent diabetes mellitus*

Table 2: Data on Pregnancy and Neonatal Development in Premature Infants with Intraventricular Hemorrhage and White Matter Damage on Day 1 CUS.

		24-30 weeks GA N=69	**30.1-36.6 weeks GA N=296**	**p value**
Intrauterine growth retardation	cases	15	62	
	IVH 1-2 (no IVH 3-4)	2	10	>0.99
	WM damage	7	5	0.001
Maternal premature uterine contractions	cases	42	164	
	IVH 1-2	14(33%)	19 (11.6%)	<0.0001
	IVH 3-4	4 (9.5%)	0	<0.0001
	WM damage	14(33.3%)	13(7.9%)	<0.0001
Chorioamnionitis	cases	10	12	
	IVH 1-2	6	3	0.084
	IVH 3-4	1	0	NS
	WM damage	4	5	>0.99
No prenatal Bethametasone	cases	15	145	
	IVH 1-2	2	16	0.645
	IVH 3-4	2	0	0.008
	WM damage	4	7	0.011
Prenatal Bethametasone	cases	54	151	
	IVH 1-2	17	2	0.001
	IVH 3-4	2	0	0.047*
	WM damage	21	17	0.001
No Magnesium neuroprotection	cases	35	272	
	IVH 1-2	6(17.1%)	31 (11.4%)	0.261 0.005 (1-2)+(3-4)
	IVH 3-4	2(5.7%)	0	0.011
	WM damage	10(28.6%)	19(7.0%)	<0.0001
Magnesium neuroprotection	cases	34	24	
	IVH 1-2	13 (38.2%)	4 (16.7%)	0.069
	IVH 3-4	2(5.9%)	0	
	WM damage	15(44.1%)	5(20.8%)	0.066
Maternal antibiotic treatment during delivery	cases	33	108	
	IVH 1-2	12(36.4%)	17 (15.7%)	0.006 0.001 (1-2)+(3-4)
	IVH 3-4	2(6.1%)	0	0.034*
	WM damage	17(51.5%)	14(13.0%)	<0.0001

Vaginal delivery	cases	20	113	
	IVH 1-2	6 (30.0%)	13(11.5%)	0.014 <0.0001(1-2)+(3-4)
	IVH 3-4	4(20%)	0	<0.0001
	WM damage	6(30.0%)	10(8.8%)	0.016
Cesarean Section	cases	49	183	
	IVH 1-2, *no grade 3-4*	13 (26.5%)	22(12%)	0.012
	WM damage	19(38.8%)	14(7.7%)	<0.0001
Delivery room resuscitation	cases	51	119	
	IVH 1-2	14(27.5%)	11(9.2%)	0.001
	IVH 3-4	3(5.9%)	0	0.016
	WM damage	17(33.3%)	17(14.3%)	0.004
No delayed cord clamping	cases	48	247	
	IVH 1-2	16 (33.3%)	26 (10.5%)	<0.0001 <0.0001(1-2)+(3-4)
	IVH 3-4	4(8.3%)	0	<0.0001
	WM damage_	16(33.3%)	16(6.5%)	<0.0001
Intubation	cases	58	47	
	IVH 1-2	17 (29.3%)	6(12.8%)	0.025 <0.010 (1-2)+(3-4)
	IVH 3-4	4(6.9%)	0	0.116
	WM damage_	20(34.5%)	6(12.8%)	0.010
Surfactant supplementation therapy	cases	57	51	
	IVH 1-2	16(28.1%)	6(11.8%)	0.021 0.007 (1-2)+(3-4)
	IVH 3-4	4(7%)	0	0.048*
	WM damage_		4(7.8%)	0.001

**Bonferroni correction*

18 (12.8%) demonstrated lenticulostriate vasculopathy. Benign accumulation of fluid in the subarachnoid spaces was seen in 10 (7.1%) infants. Five (3.5%) infants showed single choroid plexus cyst, and one (0.7%) infant had periventricular pseudocysts. Cerebellar hemorrhage was diagnosed in one (0.7%) infant.

For infants in all GA groups with other findings on day 1 CUS, subsequent CUS was performed on days 5-7 for 17 infants and on days 10-14 for 18 infants. Abnormal findings on CUS were demonstrated only in nine (52.9%) infants and only in eight (52.9%) infants, respectively. In some cases there were more than one finding.

DISCUSSION

Severe IVH and PVL on CUS are reliable predictors of adverse neurodevelopmental outcomes in preterm infants, especially if they are clinically asymptomatic [18]. Early diagnosis of IVH provides a chance for close post-hemorrhagic hydrocephalus monitoring, allows to discuss the risk of neurodevelopmental disabilities with parents and to refer these infants to early intervention services [19].

Even patients born premature and suffering from low-grade IVH (grades I and II) show higher percentage of neurodevelopmental impairment in comparison to controls without IVH, including higher incidence of cerebral palsy in school-aged children [20-22]. The information provided by early US studies may explain later developmental effects even if these findings disappear in later diagnostic imaging studies.

The present guidelines for routine CUS were published in 2002 by The American Academy of Neurology and the Practice Committee of the Child Neurology Society. These guidelines recommend to perform a routine CUS in all infants with GA younger than 30 weeks between 7 and 14 days of age and repeat the CUS between 36 and 40 weeks of postmenstrual age [4]. Later The Canadian Pediatric Society suggested the need for CUS in premature infants born before 32 weeks of GA [3,23].

Although CUS screening is recommended routinely for high risk neonates [4], the prematurity itself puts the infant in a high-risk group. Although most of brain lesions in preterm infants are clinically silent, these infants are at risk of abnormal neurodevelopmental outcome that can be improved by means of early diagnosis, follow-up and early multidisciplinary interventions.

There is an increasing evidence of the correlation between late-preterm birth and long-term medical and behavioral morbidities, such as cerebral palsy, attention deficit and antisocial behavior, as well as impaired cognitive and academic performance in school age [8,24]. In a prospective population-based study of outcomes following late to moderate premature birth, these infants were at double risk for neurodevelopmental disability at 2 years of age compared with term-born peers, with most impairments observed in the cognitive domain [8]. There are no standard recommendations for neurologic assessment including brain imaging after birth and developmental follow-up schedule in moderate-late premature infants. As a result, data showing association between neuro-developmental outcomes and brain sonography in these infants is missing. Zhang et al recommend performing neurodevelopmental monitoring for

late preterm infants because of the high risk for early intellectual developmental delay [24].

The Canadian Pediatric Society suggests routine CUS for preterm neonates born between 32 to 36 weeks +6 days of GA only in the presence of risk factors for IVH or ischemia [3]. Bhat et al., recommend CUS screening for infants born between 30 and 34 weeks of GA [7], Ballardini performs CUS for all premature infants born at 33-36 weeks of GA [15]. According to Leijser et al., and Meijler et al., all neonates admitted to NICU after their birth are to be included in CUS screening [16,25].

CUS for infants born before 32 weeks of GA was performed by Diwakar on days 1, 3 and 7, with follow up scan 7-14 days later, at one month, three months and six months of age [18]. Ballardini et al., performed screening CUS preferably by day 3 but not later than on day 7 in all newborns [15], whereas Bhat performed CUS on 5-7 days of life [26].

In 2014, American Institute of Ultrasound in Medicine (AIUM) published practice guidelines for neonatal CUS performance with the reservation that deviations from these guidelines might be needed in some cases. They encourage broadening guidelines to provide additional service and information depending on the patient [27]. Weise et al., examined asymptomatic preterm infants after 30 weeks of GA, and at least in some of them CUS demonstrated clinically significant abnormalities [28]. Due to a higher probability of adverse neurodevelopmental outcomes in moderate-late preterm infants in comparison to term-born infants and taking into consideration that these infants belong to risk population, Leijser et al., suggested performing CUS screening in all late preterm infants [15].

In this study that specifically referrers to premature infants for whom CUS was performed during day 1 of life, IVH was detected in all GA groups, including moderate and late preterm infants. The presence of IVH on day 1 CUS was higher in the youngest GA group compared to two older GA groups, and the difference between the groups was statistically significant ($p<0.0001$). As expected, the risk of severe IVH was inversely related to GA [4].

In previous studies, the overwhelming majority of IVH cases in preterm infants occurs within the first three days of life. Of those, approximately 50% of hemorrhage cases occur within the first 5 hours, and approximately 70% occur within the first 24 hours of life. 95% of IVH cases occur by day 7 [4,29]. In our study, twenty (22.4%) infants with IVH had normal CUS on day 1 CUS.

It is important to mention that in 15.7% of the patients IVH was already diagnosed on day 1. This finding was seen only on day 1 with no abnormalities detected on subsequent CUS on days 5-7 and/or days 10-14 of life. If these participants had not been examined on day 1 of life, the diagnosis of IVH could have been missed. It is particularly true for IVH developed prenatally.

In this study, four (5.8%) patients born on week 24 of GA in the youngest GA group (N=69) demonstrated IVH grade 3-4 already on day 1 CUS ($p=0.0001$). Two (2.9%) of them died during the first two days. This group included the smallest and the most fragile infants at high risk for mortality and complicated development, so CUS data might be of help in consulting the parents [18].

Abnormal findings that presumably appear in utero and are demonstrated on day 1 CUS in premature infants coincide with sparse data of IVH diagnosed via prenatal ultrasound in the third trimester of pregnancy [10-13]. It presents an additional issue for consideration. According to Adiego et al., we can presume that in cases with germinal matrix liquefaction cysts on the first day CUS, IVH began forming in utero [12]. Ghi et al., concluded that fetal IVH entails the risk of adverse neurologic outcome [30].

In this study, in 58 infants with IVH already apparent on day 1 CUS, 31 participants (53%) demonstrated signs of clot liquefaction in the ventricles, indicating that IVH had started at least some days before the birth. This finding may be important for better understanding the neurodevelopmental risks for the patients [9,13].

PVL is the most common form of brain injury in preterm infants. It may be present on CUS as white matter hyperechogenicity, cystic lesions and ventriculomegaly subsequently to white matter loss [31]. It appears in the critical period of brain development due to pro-inflammatory cytokines action during inflammatory process or disturbance of periventricular area blood supply (i.e. watershed mechanism) [18]. Campbell et al., found that among children born extremely preterm white matter damage on CUS was predictive of neurodevelopmental impairment at 10 years of age [32].

Out of all 365 study participants, 316 had normal white matter appearance on day 1 CUS. Eight (2.5%) of them had signs of white matter abnormalities on the day 5-7 and/or days 10-14 of life.

49 participants from all GA groups demonstrated white matter hyper-echogenic and/or cystic lesions on day 1 CUS. Presumably, the deleterious influence on brain tissue in these infants began in utero. Rosier-van Dunné et al., showed that in high-risk fetuses, white matter echo-densities were frequently seen on CUS [9]. It takes at least two to four weeks for white mater hyperechogenic lesion to become cystic [33].

As anticipated, due to the critical period of brain development [18], the incidence of white matter hyper-echogenic and/or cystic lesions on day 1 CUS was higher in the youngest GA group compared to the older GA group, and the difference was statistically significant ($p=0.004$).

In this study, 22 (44.9%) infants had normal CUS on subsequent examinations on days 5-7 and/or days 10-14, which can be considered as transient periventricular densities. These data coincide with data received by De Vries et al [34]. However, 27 (55.1%) infants had persistent white matter lesions on consequent CUS. Seven (25.9%) infants developed cystic lesions, two of them (7.4%) had cystic lesions already apparent on day

1 CUS. PVL should be considered as a risk factor for adverse neurodevelopmental outcome [32].

LIMITATIONS

The retrospective nature of this study and relatively small number of participants had their impact on the statistical analysis. Subsequent CUS data were found only for part of the participants, those evaluated between day 5 and day 7 of life and/or between day 10 and day 14 of life. Some infants, mostly late preterm, had only one CUS on day 1 of life, and the data of subsequent follow up is missing. The data of neurodevelopmental outcome is not included in this study.

CONCLUSIONS

Based on the obtained data, we propose to consider CUS on day 1 of life in addition to common CUS practices to rule out brain abnormalities which occurred in utero. We believe that if an early CUS examination on day 1 of life is not performed, it is possible to miss at least part of the pathologic findings. Not examined infants who later may appear to have pathology will not receive appropriate neurodevelopmental follow up and the benefits of early multidisciplinary interventions will be lost. We suggest expanding the indications for CUS for a broader population of premature infants and making it a mandatory part of the neurological evaluation in every premature infant. Undoubtedly, this can be done on condition a trained neonatologist or radiologist is available to perform early CUS. Further studies are needed to verify our assumptions.

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