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Neonatal Hypoxic Ischemic Encephalopathy: Early Diagnosis and Outcome Prediction with Ultrasound

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ABSTRACT

Background: Globally, neonatal hypoxia ischemic encephalopathy (HIE) is a leading cause of both long-term neurodevelopmental impairment and neonatal mortality. It results from perinatal asphyxia leading to impaired cerebral blood flow and oxygen deprivation. Early and accurate diagnosis is crucial for timely management and prognosis prediction. Although MRI is the gold standard for brain imaging, cranial ultrasonography offers a safe, portable, and cost-effective bedside tool that enables early detection of structural and hemodynamic brain abnormalities. Doppler parameters such as resistive and pulsatility indices provide valuable information about cerebral perfusion and correlate with HIE severity and outcomes.

Objective: To describe the ultrasound presentation of the brain and cerebral hemodynamics in neonates with HIE and to assess the outcome prediction.

Patients and Methods: A cross-sectional analytic study was conducted on 50 consecutive neonates admitted with a clinical diagnosis of perinatal asphyxia at the Radiology Department, Faculty of Medicine, Menoufia University and Sheikh Zayed Specialized Hospital during the period of the study from March 2023 till April 2024.

Results: There was statistically significant relation between mortality rate and the degree of HIE severity (p=0.039), severe HIE was more common (n=5, 55.56%) than moderate HIE (n=2, 12.5%). However, neurodevelopmental disability was significantly more common among neonates who had moderate HIE (n=14, 87.5%), followed by severe HIE (n=4, 44.4%) compared to neonates who had mild HIE which hadn't neurodevelopmental disability (p=0.017).

Conclusion: Ultrasound imaging of the brain in neonates with HIE is a crucial tool for assessing cerebral damage and predicting outcomes. The integration of advanced ultrasound techniques, such as Doppler imaging, enables detailed evaluation of cerebral hemodynamics, including blood flow patterns and vascular abnormalities. These imaging modalities provide critical information on the severity and extent of brain injury.

Keywords: Neonatal HIE, Perinatal asphyxia, CUS, Doppler, Cerebral hemodynamics, Resistive index.

INTRODUCTION

A severe kind of neonatal brain injury known as neonatal HIE is brought on by hypoxia and a reduction in cerebral blood flow that is associated to asphyxia. Perinatal asphyxia, or HIE, is still a prevalent postpartum complication worldwide, affecting 1–2/1000 live deliveries in high-income nations. It is the most frequent cause of neurodevelopmental delay and brain damage [1]. Globally, an estimated 4 million of the 130 million babies are thought to experience asphyxia each year; of these, 1 million die and 1 million experience severe, long-term consequences [2].

While most neonates with mild HIE exhibit cognitive issues, survivors with severe HIE typically experience significant long-term neurologic disability, including cerebral palsy, mental retardation, and epilepsy. In order to give proper care and treatment before the ultimate harm develops, it is crucial to determine the neonatal infant's asphyxia severity as soon as possible after birth or during the first few hours following asphyxia [1].

A number of researchers examined the relationship between early long-term results and hypoxic ischemic brain damage identified by ultrasonography. According to research, long-term neuromotor outcomes between the ages of six months and two years were linked to severe aberrant cranial US results at birth [3]. Compared to the more costly and inaccessible computed tomography (CT) and magnetic resonance imaging (MRI) modalities, cranial ultrasound (CUS) is a reasonably accessible tool for evaluating the neonatal brain. While Doppler ultrasonography offers an extra assessment of cerebral perfusion, CUS can identify developmental problems [4].

The most common brain abnormalities in both preterm and full-term neonates may be found by US, which can also be used to track the progression of lesions in both types of neonates. Thus, the present study aims to describe the US presentation of the brain and cerebral hemodynamics in neonates with HIE and to assess the outcome prediction.

PATIENTS AND METHODS

Study design

A cross-sectional analytic study was conducted on 50 consecutive neonates admitted with a clinical diagnosis of perinatal asphyxia at the Radiology Department, Faculty of Medicine, Menoufia University and Sheikh Zayed Specialized Hospital during the period of the study from March 2023 till April 2024.

Patients' criteria

Inclusion criteria: Infants with HIE, underwent CUS within the week following birth, low Apgar score, and fetal acidosis.

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Exclusion criteria: Major congenital malformations, including chromosomal or genetic syndromes, or central nervous system malformations. Severe coagulopathy with active bleeding or major intracranial hemorrhage. Inborn errors of metabolism or suspected metabolic encephalopathy.

Sample size estimation

Based on a review of past literature **Aly and Al-Ghannam** [5] who conducted handicapping of survivors showed a significant correlation with an abnormality of RI of ACA (r=0.399), the minimal sample size calculated was 47 participants at 80% power and 95% CI.

Methodology

All patients were subjected to:

Demographic information was obtained for each participant including name, gestational age (GA), sex, residence, and medication.

US examination: All patients were **subjected to**: US exams were done using an SSA-660A scanner with a 12-MHz linear array transducer. A radiologist with competence in neonatal cranial ultrasound performed the initial US evaluation within 72 hours of delivery. Neonates were kept calm and in a supine position. First, the following elements were measured on sagittal and coronal planes through the anterior fontanel: The morphology of brain tissue, the size and position of US anomalies, and the size and shape of the lateral ventricles. Color Doppler flow image (CDFI), and pulsed wave Doppler (PWD) to assess the brain vascularity. Anterior or Middle cerebral artery (ACA/MCA) blood flow parameters using CDFI, and PWD.

The US examinations of each neonate of the study group were reviewed to determine the severity of HIE. The severity of HIE for each neonate was divided into three groups as follows:

- ***Mild:*** tiny hyperechoic foci of brain parenchymal echogenicity were primarily seen in the periventricular regions.
- ***Moderate:*** more than two lobes of the brain were diffusely hyperechoic, with an unclear border of white-gray matter and a narrowing lateral ventricle.
- ***Severe:*** when there were widespread parenchymal hyperechoic patches, some without echoes, gray and white matter borders vanished, and the lateral ventricle was squeezed by intracranial bleeding.

Neurodevelopmental outcome

We gather clinical data on all neonates retrospectively. A neonatologist was undertaken neurodevelopmental follow-up at the age of two years using the Bayley Scales of Infant and Toddler Development, third edition (BSITD-III). Adverse outcomes at 2 years of age include mortality, cerebral palsy, or a cognitive/motor composite score <85 based on BSITD-III (USA standards) [6].

Outcomes of the study: Mortality rate. Major neurodevelopmental disability (in surviving babies).

Ethical Considerations

The Ethical Committee of the Faculty of Medicine at Menoufia University approved the study. The research's purpose and processes were explained to the participants' parents, and signed informed permission was acquired from all of them following an explanation of the nature and scope of the study. The Helsinki Declaration was followed throughout the course of the study.

Statistical analysis

On a computer that was compatible with IBM, SPSS software, version 26.0, was used to gather, tabulate, and statistically analyze data. Both analytical and descriptive statistical techniques were used. For qualitative variables, descriptive statistics were presented as numbers and percentages, whereas for quantitative variables, they were presented as mean ± SD, and range. Analytical statistics comprised the Mann-Whitney (U) test for quantitative data that was not regularly distributed. The one-way ANOVA test (F) was used to detect significant difference between three groups of normally distributed quantitative variables. The Shapiro-Wilk test was used to determine the normality of quantitative data, which was assumed to have a normal distribution at $p > 0.05$. A significant p-value was defined as equal to or less than 0.05.

RESULTS

Figure (1) depicts a flowchart representing the study population. Of the 50 neonates with perinatal asphyxia were willing to participate in the study, 25 of them had mild HIE, 16 had moderate and 9 had severe HIE **(Figure 1)**.

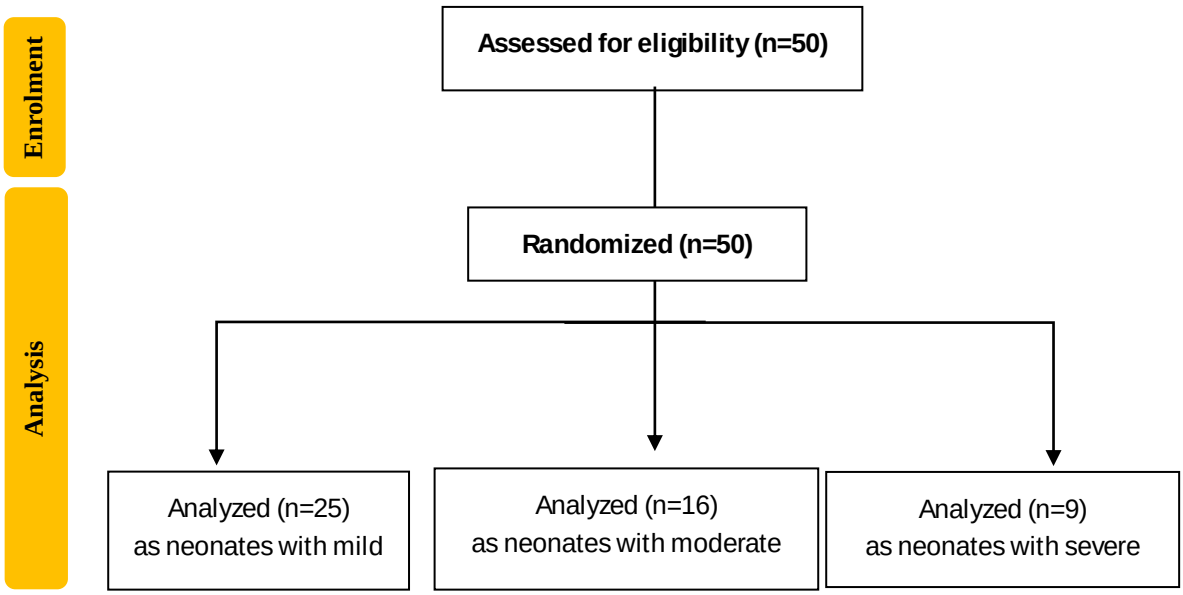

Figure (1): Flowchart of the studied neonates with HIE included in the current study.

The mean age of the studied neonates was 3.58± 1.03 days and 26 of them were males. Mean gestational age (GA) was 31.18± 2.06 weeks. Most of the them were delivered by cesarean section (56%). Moreover, the mean weight and height of studied neonates was 2093.00± 224.75 g and 16.88± 0.98 cm, respectively. Regarding Apgar score at 5 minutes, its mean was 6.22± 1.47 **(Table1).**

Table (1): Socio-demographic and clinical data of the neonates studied.

Variables	Mean ± SD.	Range
Age, days	3.58± 1.03	2.0-6.0
Sex	**N**	**%**
Male	26	52.0
Female	24	48.0
GA/ weeks	31.18± 2.06	28.0-35.0
Mode of delivery		
Normal	22	44.0
CS	28	56.0
Weight/g	2093.00± 224.75	1800.0-2700.0
Height/cm	16.88± 0.98	15.0-19.0
Apgar score at 5 min.	6.22± 1.47	3.0-9.0

The mean systolic velocity (SV), diastolic velocity (DV), resistive index (RI), pulsatility index (PI), and TMAX of studied neonates were 62.29± 33.44, 20.68±13.75, 0.67±0.11, 1.17±0.42 and 39.56±20.61respectively. As well as most of the studied neonates had mild HIE (50%), followed by 16 neonates who had moderated HIE (32%) and 9 neonates who had severe HIE (18%) **(Table 2).**

Table (2): Doppler measurements and degree of HIE severity among the studied neonates.

Variables	Mean ± SD.	Range
SV	62.29± 33.44	15.1-176.0
DV	20.68± 13.75	5.8-63.7
RI	0.67± 0.11	0.48-0.89
PI	1.17± 0.42	0.63-2.30
TMAX	39.56± 20.61	17.2-112.8
Severity	**N**	**%**
Mild	25	50.0
Moderate	16	32.0
Severe	9	18.0

There was no statistically significant relation between demographic and clinical data in relation to the degree of HIE severity. However, the Apgar score at 5 minutes significantly decreased among neonates who had severe HIE than mild and moderated HIE **(Table 3)**.

Table (3): Demographic and clinical data in relation to the degree of HIE severity.

Variables	Mild (n=25)	Moderate (n=16)	Severe (n=9)	F	*P value*
Age/days					
Mean ± SD.	0.30± 0.85	0.30± 1.39	0.30± 0.60	1.020	0.392
Range	2-5	2-6	3-5		
GA/weeks					
Mean ± SD.	31.57 ± 1.83	31.19 ± 2.32	30.89 ± 2.09	0.419	0.660
Range	28-34	28-35	28-34		
Weight/g					
Mean ± SD.	2154.76±238.70	2084.38±229.29	2022.22 ±204.80	1.308	0.283
Range	1800-2700	1800-2600	1800-2400		
Height/cm					
Mean ± SD.	16.81 ± 0.98	17.19 ± 0.98	16.89 ± 0.93	0.764	0.471
Range	15.00-18.00	16.00-19.00	16.00-18.00		
Apgar score					
Mean ± SD.	7.10 ± 0.30	6.00 ± 0.00	3.56 ± 0.53	U=	**<0.001***
Range	7.00-8.00	6.00-6.00	3.00-4.00	309.45	

***** significant.

There was no statistically significant relation between SV, and TMAX in relation to the degree of HIE severity. However, the DV significantly decreased among neonates who had severe HIE than mild and moderated HIE. While RI and PI significantly increased among neonates who had severe HIE than mild and moderated HIE **(Table 4)**.

Table (4): Doppler measurements in relation to the degree of HIE severity.

Variables	Mild (n=25)	Moderate (n=16)	Severe (n=9)	F	*P value*
SV					
Mean ± SD.	66.19 ± 35.75	62.63 ± 39.53	54.51 ± 22.35	0.268	0.848
Range	30.50-176.00	24.20-176.00	15.10-96.40		
DV					
Mean ± SD.	25.45 ± 14.56	19.19 ± 13.78	10.17 ± 5.51	3.184	**0.032***
Range	9.10-63.70	6.40-63.70	5.80-24.10		
RI					
Mean ± SD.	0.62 ± 0.06	0.69 ± 0.08	0.78 ± 0.13	13.151	**<0.001***
Range	0.48-0.70	0.52-0.78	0.54-0.89		
PI					
Mean ± SD.	0.99 ± 0.17	1.09 ± 0.24	1.85 ± 0.48	22.738	**<0.001***
Range	0.63-1.22	0.75-1.41	0.81-2.30		
TMAX					
Mean ± SD.	42.99 ± 23.09	42.40 ± 22.25	26.91 ± 5.98	1.468	0.236
Range	18.20-112.80	17.20-112.80	20.30-35.70		

***** significant.

There was statistically significant relation between mortality rate and the degree of HIE severity, severe HIE was more common (55.56%) than moderate HIE (12.5%). However, neurodevelopmental disability was significantly more common among neonates who had moderate HIE (87.5%), followed by severe HIE (44.4%) compared to neonates who had mild HIE, which hadn't neurodevelopmental disability **(Table 5)**.

Table (5): Outcomes prediction in relation to the degree of HIE severity.

Variables	Mild (n=25)	Moderate (n=16)	Severe (n=9)	X^2	*P value*
Mortality rate	0	2 (12.5%)	5 (55.56%)	3.85	**0.039***
Neurodevelopmental disability	0	14 (87.5%)	4 (44.4%)	5.67	**0.017***
Normal	25 (100%)	0	0	NA	**---**

***** significant.

DISCUSSION

A significant cause of infant morbidity and mortality worldwide, HIE, which is a subset of neonatal encephalopathy [7]. While severe HIE will leave lifelong neurological sequelae, less severe HIE combined with prompt management may have a better prognosis. Therefore, using a trustworthy imaging equipment to obtain an early diagnosis of HIE is crucial. Although MRI is the most reliable imaging test for diagnosing brain damage, it cannot be done right away on a neonate who is severely asphyxiated. Brain hemodynamics may be thoroughly studied using 2-D transcranial Doppler US [8]. So**,** the aim of the current study was to describe the US presentation of the brain and cerebral hemodynamics in neonates with HIE and to assess the outcome prediction.

Our study showed that a total of 50 neonates with HIE were included in this study, their ages ranged from 2-6 days, with a mean of 3.58± 1.03 days, males were (n=26, 52%), and 48% were females (n=24), also, GA ranged from 28-35 weeks, with mean 31.18± 2.06 weeks. Most of the neonates studied were delivered by cesarean section (n=28, 56%), and 22 were delivered by normal labor (44%).

The mean weight and height of studied neonates was 2093.00±224.75 g and 16.88± 0.98 cm, ranging from 1800-2700 g, and 15-19 cm, respectively. Regarding Apgar score at 5 minutes, it ranged from 3-9 with a mean of 6.22± 1.47. The mean SV, DV, RI, PI, and TMAX of studied neonates were 62.29± 33.44, 20.68±13.75, 0.67±0.11, 1.17±0.42 and 39.56±20.61 respectively. Most of the studied neonates had mild HIE (50%), followed by 16 neonates who had moderated HIE (32%) and 9 neonates who had severe HIE (18%).

In the same line, **Guan *et al.*** [8] enrolled forty-four (34.2%), sixty (38.0%), and forty-four (27.8%) of the 158 consecutive neonates (82 boys and 76 girls) with HIE. For transcranial US, 120 healthy neonates were chosen at random to act as a control group. Also, **Liu *et al.*** [9] included 40 full-term newborns with HIE who were hospitalized to the neonatal ICU in their study group. Thirty full-term, healthy babies delivered at the same time made up the control group. They demonstrated that the gestational ages (GAs) in HIE groups and healthy controls were 38.6$\pm$0.74 (37.3–39.9) weeks and 38.3$\pm$0.60 (37.4–39.6) weeks, respectively (t=1.77, p=0.96). Birth weights in HIE groups were 3289$\pm$439 g, whereas in healthy controls they were 3413$\pm$436 g (t=1.18, p=0.12).

Our study showed that there was no statistically significant relation between demographic and clinical data in relation to the degree of HIE severity. However, the Apgar score at 5 mins significantly decreased among neonates who had severe HIE than mild and moderated HIE. Consistent with our research, **Guan *et al.*** [8] demonstrated that there was no significant difference in the neonates' body weight, gender, or GA between the 2 groups. Also, **Çeltik *et al.*** [10] showed that there were no variations in GA, birth weight, gender, or delivery method between the patient and control groups. Group 1 had more out-of-birth patients than Group 2 and the control group (P=0.05). Patients in Group 1 had substantially lower Apgar scores at the first and fifth minutes compared to those in Group 2 and the control group (P=0.001; Group 1 versus Group 2 and control group).

Furthermore, **Moghaddam *et al.*** [11] showed that there was no significant relationship between HIE severity stage and birth weight (P=0.706). Likewise, there was no significant difference in GA between the HIE stage groups (P=0.124). They showed the distribution of gender and delivery type among neonates with HIE. The percentage of boys and girls with the HIE stage did not differ significantly, according to the X^2-test (P=0.515). Similarly, there was no significant correlation (p=0.628) between the manner of delivery and the HIE stage. Two emergency cesarean sections and one NVD were performed on the three babies in the third group. Additionally, they demonstrated that Apgar ratings in study groups at 1 and 5 mins after delivery differed substantially in newborns with different HIE stages based on the t-test (P=0.003 and 0.008, respectively). Nonetheless, they demonstrated a substantial correlation between the severity of HIE and either the Apgar score or the necessity for resuscitations. HIE primarily occurred in preterm infants that required neonatal resuscitation due to poor Apgar scores or those delivered by instrument through NVD. Lower GAs were expected to be associated with higher poor neurodevelopmental occurrences, which is consistent with our results.

Our study showed that there was no statistically significant relation between SV, and TMAX in relation to the degree of HIE severity. However, the DV significantly decreased among neonates who had severe HIE than mild and moderated HIE. While RI and PI significantly increased among neonates who had severe HIE than mild and moderated HIE. In the same line, **Guan *et al.*** [8] demonstrated that 43 (97.7%) of the 44 newborns with severe HIE had decreased blood flow velocity and increased RI, whereas only one of the 44 displayed a high perfusion condition with higher blood flow velocity and lower RI. Additionally, they discovered that diminished perfusion of brain tissue in HIE is indicated by higher RI in all but one infant with severe HIE. Blood flow velocity decreases in newborns with clinical HIE when cerebral blood flow velocity is reduced and RI is raised; in particular, the diastolic velocity is dramatically reduced. All of these individuals were clinically diagnosed with severe HIE. The body regulates cerebral blood flow depending on changes in vascular resistance, and a drop in blood pressure following hypoxia is one cause of reduced cerebral blood flow. Another is that elevated cranial pressure can also cause decreased cerebral blood flow. Blood flow changes, local blood supply, and oxygen supply can all be indirectly reflected by RI, which has a positive

correlation with loop impedance and can reflect peripheral circulation impedance.

Additionally, **Aly and Al-Ghannam, [5]** showed that Doppler investigation found that eleven, twelve and thirteen individuals had aberrant RI of ACA, MCA, and ICA, respectively. Three patients (11.5%) had grade-four HIE, eight patients (30.8%) had grade-three, and four patients (15.4%) had grade-two HIE, according to disrupted RI of intracranial arteries. Doppler scanning also confirmed the diagnosis of HIE in fifteen patients (57.7%) and ruled out the existence of HIE in eleven patients (42.3%). Further, **Ahmad *et al.* [12]** showed that newborns were categorized as having normal resistive index (RI=0.56-0.8) N=54 and aberrant resistive index (RI=0.80) N=56 based on color Doppler findings. The percentage of patients with normal RI in stage-two and stage-three of HIE was 75.9%, while the percentage of patients with aberrant RI in stage-two and stage-three was 69.6% and 30.4%, respectively.

Our study showed that there was statistically significant relation between mortality rate and the degree of HIE severity. However, neurodevelopmental disability was significantly more common among neonates who had moderate HIE (n=14, 87.5%), followed by severe HIE (n=4, 44.4%) compared to neonates who had mild HIE, which hadn't neurodevelopmental disability. In the same line, **Çeltik *et al.* [10]** demonstrated that although there was no morbidity and mortality in Group 2 or the control group, eight patients passed away—two in the stage-two HIE group and six in the stage-three HIE group. Additionally, **Guan *et al.* [8]** found that of 54 neonates with moderate HIE, 23 (42.6%) had a slightly narrower lateral ventricle, 2 (3.7%) had slightly increasing parenchymal echo limits, and 29 (53.7%) had no US abnormalities of the brain parenchyma. In babies with mild HIE, diffuse or localized parenchymal echo was hyperechoic in 56/60 (93.3%) with less distinct borders of cerebral sulcus, with 14/56 (25.0%) having periventricular-intraventricular hemorrhage and their lateral ventricles being somewhat dilated. All 44/44 (100%) infants with severe HIE exhibited significant increased parenchymal echo with heterogeneous brain anatomy, with 13/44 (29.5%) showing an unclear or slit lateral ventricle and 17/44 (38.6%) having cerebral hemorrhage.

While **Ahmad *et al.* [12]** showed that during the research period, 110 newborns were enrolled; twenty-three of them died while in the hospital, and 10 of them were not followed up with. At six and twelve months, the presence of an anomalous resistive index was linked to both an aberrant neurodevelopmental outcome and a significantly increased chance of mortality. Our research revealed that newborns with normal RI had a 13% hospital stay mortality rate, but those with aberrant RI had a 28.6% hospital stay mortality rate ($p<0.01$).

STRENGTHS AND LIMITATIONS

This study highlights the value of cranial ultrasonography as a non-invasive, bedside, and cost-effective tool for early diagnosis and outcome prediction in neonates with HIE. The use of Doppler assessment provided additional insight into cerebral hemodynamics, enhancing prognostic accuracy. However, the study's single-center methodology and somewhat small sample size were its limitations. Moreover, ultrasound findings were operator-dependent and less sensitive than MRI in detecting subtle or deep brain injuries.

CONCLUSION

In conclusion, this study highlights ultrasound imaging of the brain in neonates with HIE as a crucial tool for assessing cerebral damage and predicting outcomes. The integration of advanced ultrasound techniques, such as Doppler imaging, enables detailed evaluation of cerebral hemodynamics, including blood flow patterns and vascular abnormalities. These imaging modalities provide critical information on the severity and extent of brain injury. Ultrasound can effectively identify structural abnormalities, such as intraventricular hemorrhage and periventricular leukomalacia, which are common in HIE. Early detection of these abnormalities is essential for timely intervention.

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