International Journal of Contemporary Pediatrics
Husain S et al. Int J Contemp Pediatr. 2026 Dec;13(12):2434-2440
https://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20264307

Original Research Article

Ultrasonography findings of the brain of the of neonatal preterm babies with birth asphyxia

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Received: 09 May 2026
Accepted: 09 July 2026

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ABSTRACT

Background: Perinatal asphyxia remains a major contributor to neonatal morbidity and mortality, particularly among preterm neonates. It can lead to hypoxic-ischemic encephalopathy (HIE), causing significant brain injury. Cranial ultrasound (CUS) is a non-invasive, bedside imaging modality widely used to detect brain injuries such as intraventricular hemorrhage (IVH) and periventricular leukomalacia (PVL) in neonates. Therefore, this study aimed to evaluate the ultrasonographic findings of the brain in preterm neonates with birth asphyxia.
Methods: A prospective cohort study was conducted on 57 neonates with perinatal asphyxia admitted to the Department of Radiodiagnosis at Al-Ameen Medical College and Hospital, Bijapur (2023-2025). Gestational age was assessed by LMP and Ballard Score. Staging of HIE was done using the Sarnat and Sarnat criteria. CUS was performed on days 5 to 7 post-birth using standard anterior fontanelle views.
Results: Out of 57 neonates, abnormal cranial USG findings were significantly associated with higher HIE stages. The most common abnormalities included germinal matrix hemorrhage (GMH), IVH, PVL, and cerebral edema. The incidence of abnormal findings increased with HIE severity-most notably in Stage III. Doppler evaluation also demonstrated altered resistive indices in the anterior and middle cerebral arteries correlating with clinical severity.
Conclusions: CUS is an effective initial neuroimaging tool in preterm neonates with birth asphyxia. It provides early detection of brain injury and helps stratify neurodevelopmental risk based on HIE severity. Integration of serial CUS with clinical evaluation improves prognostication and guides timely interventions.

Keywords: Cranial ultrasound, Preterm neonates, Perinatal asphyxia, Hypoxic-ischemic encephalopathy, Intraventricular hemorrhage, Periventricular leukomalacia

INTRODUCTION

Birth asphyxia remains a significant cause of neonatal morbidity and mortality, especially in preterm infants. It results from disrupted oxygen and nutrient exchange during the perinatal period, often leading to HIE and subsequent brain injury affecting the brain, heart, liver, and kidneys, with neurological consequences being the most severe.[1] Neuroimaging plays a vital role in detecting brain injury, guiding management, and predicting outcomes. CUS is widely preferred in preterm neonates due to its bedside accessibility, real-time imaging, and lack of radiation. Although MRI offers more detailed white matter evaluation and outcome prediction.[2] It is less feasible for routine use. The preterm brain, due to its immature structure and predominance of unmyelinated white matter, is highly susceptible to injuries such as PVL and intraventricular hemorrhage (IVH).[3] GMH and incomplete cortico-subcortical development further increase vulnerability to long-term deficits. Compared to term infants, preterms have reduced cortical volume and myelination, making white matter more prone to damage, whereas term neonates often show basal ganglia and thalamic involvement.[4] The progression of hypoxic-ischemic brain injury occurs in three phases: primary energy failure, latent phase, and secondary neuronal injury with oxidative stress and inflammation.[5] CUS

effectively detects IVH, PVL, and cerebral edema, offering real-time prognostic insight.[6] While MRI provides superior detail, CUS remains indispensable in resource-limited NICUs. CT is less favored due to radiation and poor white matter contrast.[7] Serial ultrasound imaging enhances injury detection and outcome prediction, with recommended scans at 24-48 hours (acute hemorrhage), 7-10 days (PVL progression), and term-equivalent age (structural outcomes).[8,9] Early scans guide interventions such as hypothermia therapy in moderate to severe HIE. Doppler ultrasound also assesses cerebral perfusion and monitors therapy effects.[10] Although CUS is valuable, its sensitivity for subtle injuries like non-cystic PVL is limited, and accuracy may vary with operator skill.[11,12] MRI, including DWI and MRS, is advised in moderate to severe cases for comprehensive evaluation.

Therefore, this study aimed to evaluate the ultrasonographic findings of the brain in preterm neonates with birth asphyxia, with the goal of enhancing early diagnosis, monitoring, and prognosis. Cranial ultrasound remains a first-line neuroimaging tool in neonatal care, enabling timely intervention and improved neurodevelopmental outcomes when integrated with structured protocols and adjunct imaging as needed.

METHODS

Source of data and study design

This prospective cohort study was conducted in the Department of Radiodiagnosis at Al-Ameen Medical College and Hospital, Bijapur (Karnataka), over a period of two years from 2023 to 2025. A total of 57 newborns diagnosed with perinatal asphyxia were enrolled.

Inclusion and exclusion criteria

The study included term and preterm newborns aged 0 to 7 days with a clinical diagnosis of perinatal asphyxia, and whose parents or guardians provided informed consent. Newborns with sepsis, major congenital anomalies, respiratory distress syndrome, intrauterine infections, hyperbilirubinemia, inborn errors of metabolism, or birth trauma were excluded.

Data collection

Clinical and demographic details were recorded using a structured proforma. Informed written consent was obtained from the guardians of all enrolled newborns.

Methodology

The diagnosis and management of perinatal asphyxia were based on FBNC and NNF India protocols. Severity was staged using the Sarnat and Sarnat classification into stages I, II, and III. Gestational age was determined by the last menstrual period (LMP) and confirmed using the modified Ballard score on admission. Newborns were classified as preterm (<37 weeks) or term (37-42 weeks). Standard treatment was administered, and cranial ultrasonography (CUS) was performed between days 5 and 7 using anterior fontanelle views in sagittal and coronal planes.

Statistical analysis

Data were entered in Microsoft excel and analysed using statistical software SPSS version 26 (SPSS Inc., Chicago, IL, USA). The continuous variables were evaluated by mean (standard deviation) or range value when required. The dichotomous variables were presented in number/frequency and were analysed using the Chi-square. A $p<0.05$ or 0.001 was regarded as significant.

RESULTS

A slight male predominance was observed, with 32 males (56.1 %) and 25 females (43.9 %). Regarding birth weight, most neonates (68.4 %) weighed 1500-2500 gm, while 31.6 % weighed <1500 g, classifying them as very low birth weight (VLBW) infants (Figure 1).

Gestational ages ranged from 28 to 36 weeks: 38.6 % were extremely to very preterm and 61.4% were late preterm. All neonates had severely depressed Apgar scores (<3 at 1 minute and <6 at 5 minutes), confirming severe birth asphyxia (Figure 2).

Based on Sarnat and Sarnat staging, 35.1% had stage 1 HIE, 40.4 % had stage 2, and 24.5 % had stage 3, indicating a high proportion with moderate-to-severe encephalopathy (Figure 3). Initial cranial USG revealed abnormalities in 61.4% of neonates. Follow-up scans after 72 hours showed persistent or progressive abnormalities in 64.9% (Table 1).

A statistically significant association was observed between HIE stage and abnormal cranial USG findings at both time points. At≤36 hours, abnormal findings were present in 30.0% of stage 1, 65.2% of stage 2, and 100.0% of stage 3 neonates (p=0.0002). At ≥72 hours, the rates increased slightly to 30.0%, 73.9%, and 100% respectively (p<0.0001) (Table 2).

Birth weight showed stronger association with abnormal USG findings at ≥72 hours than at ≤36 hours. At ≤36 hours, 77.8% of neonates <1500 g and 53.8% of those 1500-2500 g had abnormalities (p=0.0z45). At ≥72 hours, abnormalities increased to 83.3% and 56.4% respectively, reaching statistical significance (p=0.0477) (Table 3).

No significant association was noted between gestational age and specific USG abnormalities at either time point. At ≤36 hours, total abnormalities were seen in 21 neonates of 28-32 weeks and 14 of 33-36 weeks (p=0.4735). At ≥72 hours, totals remained similar at 21 and 16 respectively (p=0.9377) (Table 4).

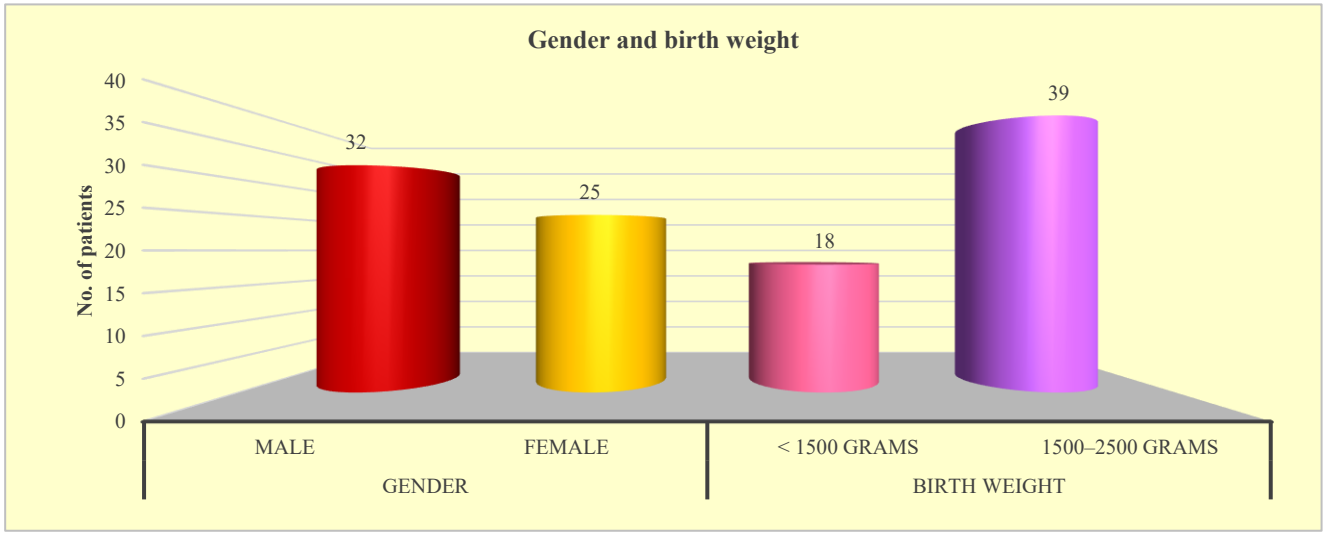


Figure 1: Gender and birth weight distribution of preterm neonates.

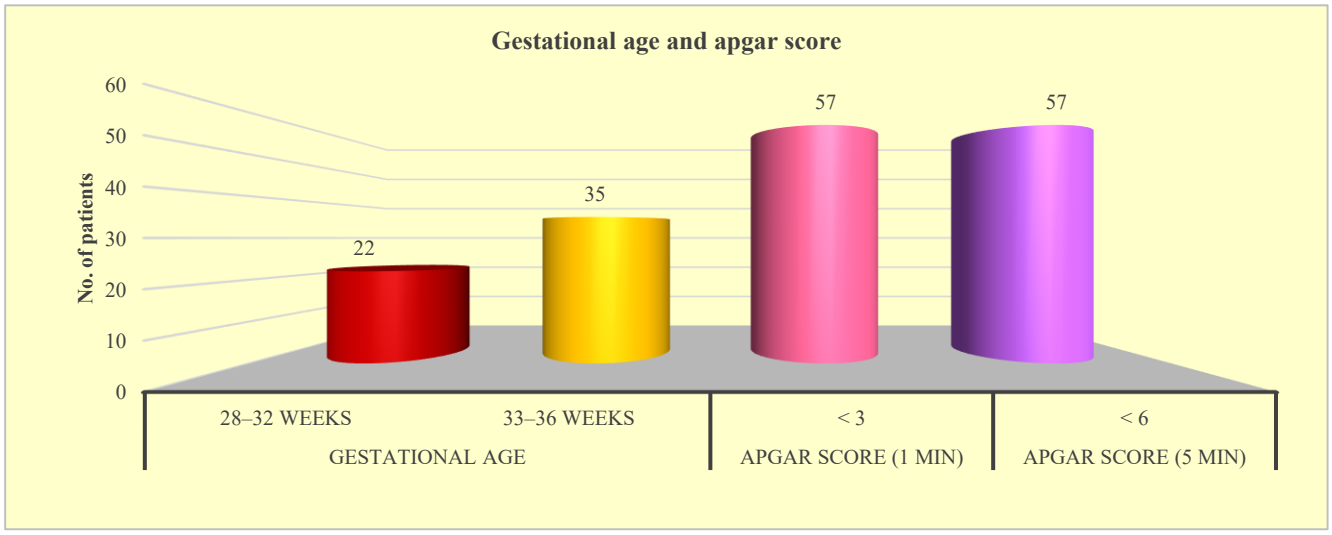


Figure 2: Gestational age and apgar scores of preterm neonates.

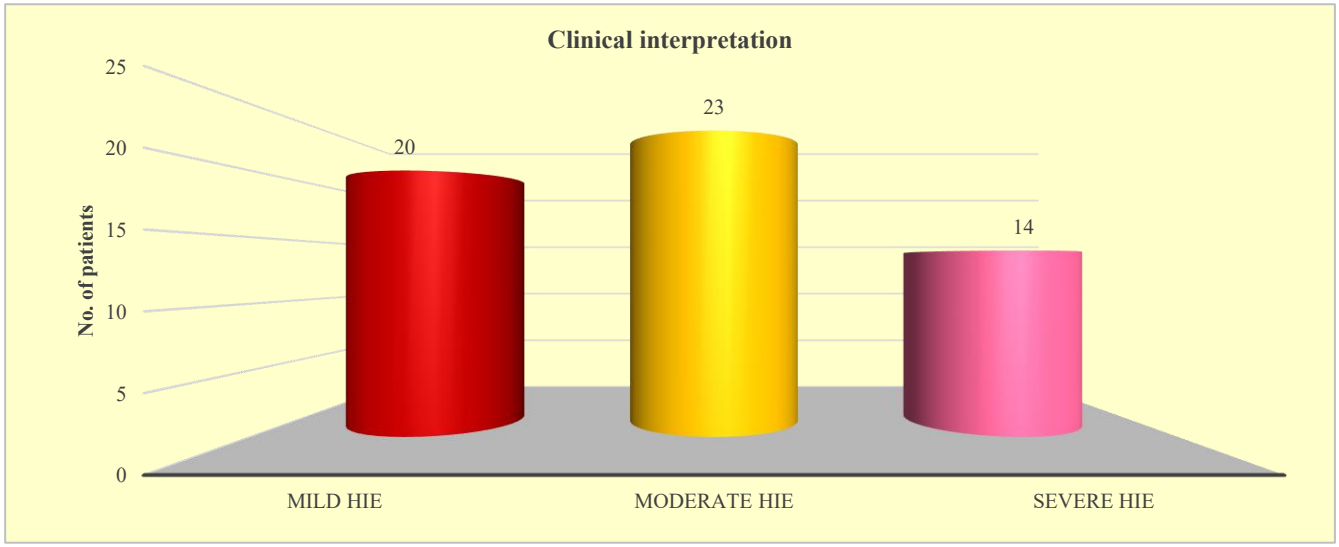


Figure 3: Distribution according to Sarnat and Sarnat grading of HIE.

Table 1: Cranial ultrasound findings at ≤36 and ≥72 hours of life, (n=57).

USG findings		N	Percentage
At ≤36 hours of life	GMH	15	26.3%
	PVL	14	24.6%
	Dilated ventricles	2	3.5%
	PVL + GMH	2	3.5%
	GMH + cerebral edema	1	1.8%
	GMH + dilated ventricle	1	1.8%
	Normal scan	22	38.6%
	Total abnormal findings	35	61.4%
At ≥72 hours of life	PVL	20	35.1%
	GMH	12	21.1%
	Dilated ventricles	2	3.5%
	PVL + GMH	2	3.5%
	GMH + dilated ventricle	1	1.8%
	Normal scan	20	35.1%
	Total abnormal findings	37	64.9%

Table 2: Association between HIE stage and abnormal cranial USG findings at ≤ 36 and ≥72 hours.

HIE stage	Abnormal USG findings	Normal USG findings	Abnormal	Abnormal USG findings	Normal USG findings	Abnormal
	At ≤36 hrs			**At ≥72 hrs**		
Stage 1	6	14	30.0%	6	14	30.0%
Stage 2	15	8	65.2%	17	6	73.9%
Stage 3	14	0	100.0%	14	0	100.0%
Total	35	22	-	37	20	-
X=17.26, p=0.0002*				X=19.09, p<0.0001*		

*Significant value.

Table 3: Association between birth weight and abnormal USG findings at ≤ 36 and ≥72 hours.

Birth weight	Abnormal USG findings	Normal USG findings	Abnormal	Abnormal USG findings	Normal USG findings	Abnormal
	At ≤36 hrs			**At ≥72 hrs**		
<1500 gm	14	4	77.8%	15	3	83.3%
1500-2500 gm	21	18	53.8%	22	17	56.4%
Total	35	22	-	37	20	-
X=2.976, p=0.0845				X=3.919, p=0.0477*		

*Significant value.

Table 4: Association between gestational age and specific USG abnormalities at ≤36 and ≥72 hours.

Gestational age (in weeks)	GMH	PVL	Both	Other	Total abnormal	GMH	PVL	Both	Other	Total abnormal
	At ≤36 hrs					**At ≥72 hrs**				
28-32 weeks	10	9	1	1	21	7	12	1	1	21
33-36 weeks	5	5	1	3	14	5	8	1	2	16
Total	15	14	2	4	35	12	20	2	3	37
X=2.510, p=0.4735						X=0.8057, p=0.9377				

DISCUSSION

Perinatal asphyxia is a major cause of neonatal morbidity and mortality, with the brain being particularly vulnerable to hypoxic-ischemic injury due to its high metabolic demands and immature autoregulation.[11] Preterm neonates are especially at risk, with injury patterns varying by gestational age-white matter and germinal matrix in preterms, and cortical/deep grey matter in terms. Early detection through CUS is crucial, as it offers a non-invasive, bedside tool for identifying ischemic and hemorrhagic changes.[11,12] This study aimed to evaluate

CUS findings in preterm neonates with birth asphyxia and their correlation with clinical severity, gestational age, and birth weight. Perinatal asphyxia is a major cause of early neonatal morbidity and mortality, especially in low-and middle-income countries like India, where perinatal care may be inadequate.[13] Preterm neonates are particularly vulnerable due to immature cerebral vasculature and fragile germinal matrix, making them highly susceptible to hypoxic-ischemic.[13,14] In this study, all 57 preterm neonates had severely depressed Apgar scores, indicating significant hypoxia at birth. Shukla et al and Kuruvilla et al similarly reported high rates of HIE and neurosonographic abnormalities in preterm infants.[11,12] Our study showed a mild male predominance (56.1%), aligning with the known "male disadvantage" in neonatal outcomes. A key finding was the significantly higher incidence of abnormal USG findings in neonates with birth weight <1500 g (83.3 %) compared to those ≥1500 g (56.4 %; p=0.0477). This is consistent with Sherwani et al, Laha et al and Al-Mashhadani et al who reported low birth weight as a major risk factor for GMH, IVH, and PVL.[15-17] Egesa et al also found strong associations between low birth weight, poor Apgar scores, and abnormal cranial USG, underscoring the need for early imaging in VLBW neonates.[18] In our study, 61.4% of preterm neonates showed abnormal cranial USG within ≤36 hours, with GMH (26.3%) and PVL (24.6%) as the most common findings. This aligns with reports by Yasmin et al and others though the latter noted higher rates likely due to inclusion of extremely preterm infants.[19-21] Liu al emphasized USG's utility for early detection, and similar findings were noted by Susmita et al and Omar et al reinforcing the need for prompt cranial screening in high-risk preterms.[22-24] By the 72-hour scan, our study showed a shift from GMH (21.1%) to PVL (35.1%), indicating progression from hemorrhagic to ischemic lesions. Similar patterns were observed by Shukla et al and Yasmin et al who emphasized the importance of repeat scans.[11,19] Serial USG revealed evolving findings in studies by Sherwani et al and others, with CUS abnormalities rising with HIE severity.[15,16,25] Konan et al also confirmed that delayed imaging enhances detection of PVL and cystic changes, supporting the role of dynamic cranial monitoring.[26] The present study found a strong correlation between HIE severity (Sarnat staging) and abnormal cranial USG findings. At ≤36 hours, abnormalities were seen in 100% of stage 3, 65.2% of stage 2, and 30% of stage 1 neonates; this trend intensified by 72 hours (p=0.0002 and p<0.0001, respectively), indicating disease progression. Similar associations were reported by Ganesan et al and others confirming that ultrasound findings parallel HIE stage and can aid in early risk stratification and prognosis.[25,27] Although the correlation between gestational age and lesion type was not statistically significant (p=0.4735 at ≤36 hours; p=0.9377 at ≥72 hours), a clinical trend was evident. GMH and PVL were more frequent in preterms (28-32 weeks), while cerebral edema predominated in late preterms and near-term neonates. This aligns with Laha et al and Yasmin et al who reported similar lesion distributions based on gestational age.[16,19] The trend reflects known anatomical vulnerabilities and warrants further investigation in larger cohorts. Cranial ultrasound remains the primary imaging tool for neonatal brain assessment due to its safety and bedside utility, though it may miss subtle or deep lesions. MRI serves as a valuable adjunct, especially in ambiguous or progressive cases. Laha et al showed MRI detected 49 abnormalities versus 33 on USG, including more basal ganglia and thalamic lesions.[16] Rani et al and Zhu et al confirmed MRI's superiority, particularly DWI, in detecting early ischemic and deep grey matter injuries.[28,29] Given MRI's limited availability, a tiered approach using serial USG with selective MRI is recommended. This study reinforces the key role of cranial ultrasound in managing preterm neonates with perinatal asphyxia. Significant associations with HIE severity and low birth weight support routine neurosonographic screening at ≤36 and ≥72 hours to detect evolving lesions like PVL. Konan et al showed USG's utility even in term neonates with high-risk deliveries.[26] Liu L al emphasized that early imaging and intervention reduce neonatal mortality and disability.[22] Structured USG protocols in NICUs enable timely diagnosis, informed counseling, and early referrals, improving short- and long-term outcomes. This study confirms that CUS is a practical, non-invasive tool for early detection and monitoring of cerebral injury in preterm neonates with perinatal asphyxia. USG abnormalities significantly correlated with HIE severity and birth weight, with a noted shift from early GMH to later PVL. Though gestational age showed no statistical association, injury patterns varied with maturity. These findings support integrating CUS into NICU protocols. While MRI remains superior for deep lesions, USG's bedside utility, especially in low-resource settings, makes it essential for early diagnosis, monitoring, and prognostication.

CONCLUSION

Overall, the study demonstrates that CUS is a sensitive, accessible, and effective tool for the early identification of brain injury in preterm neonates with perinatal asphyxia. It not only correlates well with clinical severity but also allows for dynamic monitoring of lesion progression. While MRI remains superior for detailed structural evaluation, especially in detecting deep grey matter and cortical injuries, CUS offers irreplaceable utility in initial screening, particularly in resource-limited settings. Integration of serial CUS at standardized intervals into routine neonatal intensive care protocols can enable timely intervention, improved prognostication, and ultimately better neurodevelopmental outcomes in this high-risk population. However, this study was limited by a relatively small sample size, a single-center design, and the lack of MRI correlation in all cases, which may have led to an underestimation of deeper brain injuries. Moreover, the absence of long-term neurodevelopmental follow-up restricts the evaluation of

clinical outcomes. Therefore, future research should focus on larger multicentric studies with broader populations, include MRI in moderate to severe cases, and incorporate long-term follow-up to better assess the prognostic utility of early cranial ultrasound findings.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Hussain S, Mahaldar A, Vahab M, Lokesh BN. Ultrasonography findings of the brain of the of neonatal preterm babies with birth asphyxia. Int J Contemp Pediatr 2026;13:2434-40.