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Association Between Cerebral Near-Infrared Spectroscopy and Magnetic Resonance Spectroscopy Findings in Neonates with Hypoxic-Ischemic Encephalopathy

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ABSTRACT

Background: Hypoxic-ischemic encephalopathy (HIE) is an important cause of neonatal neurological morbidity and mortality. Early identification of cerebral injury is essential for clinical management and prognostication. Near-infrared spectroscopy (NIRS) provides continuous bedside assessment of cerebral oxygenation, whereas magnetic resonance spectroscopy (MRS) provides information on cerebral metabolic abnormalities. This study evaluated the association between cerebral oxygenation measured by NIRS and metabolic abnormalities detected by MRS in neonates with HIE.

Methods: This prospective hospital-based observational study included 38 neonates with HIE (Sarnat stages I–III) admitted to the neonatal intensive care unit of Chhatrapati Shivaji Subharti Hospital, Meerut, between July 2024 and February 2026. Cerebral regional oxygen saturation (rSO_2) was measured using NIRS at baseline, during hemodynamic monitoring/resuscitative management, and after fluid resuscitation. MRI/MRS was performed between 28 and 72 hours of life, and lactate peak, lactate/N-acetylaspartate (NAA) ratio, and NAA status were assessed. Continuous variables were summarized as mean ± standard deviation and categorical variables as frequencies and percentages. Between-group comparisons were performed using independent-samples t-tests, and binary logistic regression was used to evaluate the association between mean cerebral rSO_2 and lactate peak status.

Results: Of the 38 neonates, 30 (78.9%) required resuscitation and 18 (47.4%) had Sarnat stage II HIE. Mean cerebral rSO_2 was 63.3 ± 4.75% at baseline, decreased to 54.8 ± 8.97% during hemodynamic monitoring/resuscitation, and increased to 73.7 ± 3.35% after fluid resuscitation. Lactate peak and reduced NAA were observed in 30 (78.9%) neonates. Compared with neonates without a lactate peak, those with a lactate peak had lower baseline rSO_2 (61.5 ± 3.33% vs. 70.3 ± 2.12%; $p<0.001$), lower rSO_2 during monitoring/resuscitation (50.7 ± 4.29% vs. 70.3 ± 2.12%; $p<0.001$), and lower mean rSO_2 (62.0 ± 3.74% vs. 71.4 ± 2.05%; $p<0.001$). Mean cerebral rSO_2 was associated with lactate peak status (OR=2.95, 95% CI: 1.21–7.17; p=0.017).

Conclusion: Cerebral NIRS parameters were associated with MRS-detected metabolic abnormalities in neonates with HIE. NIRS may have potential as an adjunctive bedside monitoring modality; however, larger studies are required to validate these findings and determine its prognostic utility.

Keywords: Hypoxic-Ischemic Encephalopathy; Near-Infrared Spectroscopy; Magnetic Resonance Spectroscopy; Cerebral Oxygenation; Neonatal brain injury, Lactate/NAA ratio.

INTRODUCTION

Hypoxic ischemic encephalopathy (HIE) is a serious neonatal neurological disorder caused by impaired cerebral blood flow and oxygen deprivation during the perinatal period, resulting in neuronal injury and long-term neurodevelopmental impairment. Therapeutic hypothermia has significantly improved survival and neurological outcomes; however, early prediction of injury severity and outcome assessment remain major clinical challenges. [1,2] Magnetic resonance spectroscopy (MRS) has emerged as an important metabolic imaging modality for evaluating cerebral injury in neonatal encephalopathy by measuring biomarkers such as lactate and N-acetylaspartate (NAA), which reflect anaerobic metabolism and neuronal integrity. [3]

Near-infrared spectroscopy (NIRS) is a non-invasive bedside monitoring tool that continuously measures regional cerebral oxygen saturation and provides real-time assessment of cerebral perfusion and oxygen utilization in critically ill neonates. [4] Recent studies evaluating cerebral oxygenation during therapeutic hypothermia have demonstrated dynamic alterations in cerebral perfusion patterns associated with evolving brain injury. [5] In addition, altered cerebral metabolic rates following hypothermia have been associated with adverse neurodevelopmental outcomes, highlighting the importance of continuous cerebral monitoring in neonates with HIE. [6] Cerebral blood flow abnormalities occurring after hypoxic-ischemic injury have also been shown to correlate with severity and distribution of brain injury. [7]

Advanced neuroimaging modalities, particularly MRI and MRS, provide detailed structural and metabolic assessment of neonatal brain injury and remain important prognostic tools in neonatal encephalopathy. [8] However, these investigations are episodic, resource-intensive, and unsuitable for continuous bedside monitoring in hemodynamically unstable neonates. Continuous monitoring of cerebral oxygenation and perfusion has therefore gained increasing attention as an adjunctive neuroprotective strategy. [9] Serial perfusion imaging studies have demonstrated evolving cerebral hemodynamic changes following hypoxic injury, emphasizing the dynamic nature of neonatal brain injury. [10] Similarly, MRI-based studies evaluating cerebral oxygen metabolism during and after therapeutic hypothermia have demonstrated persistent metabolic disturbances despite clinical stabilization. [11] Alterations in cerebral oxygenation during rewarming after hypothermia further support the relationship between cerebral perfusion and metabolic recovery in neonatal HIE. [12]

Despite these advances, limited Indian data exist comparing cerebral NIRS monitoring with MR spectroscopy in neonates with hypoxic ischemic encephalopathy, particularly in Tier II tertiary NICU settings. Therefore, the present study was undertaken to evaluate the correlation between bedside cerebral oxygenation measured by NIRS and metabolic brain injury assessed by MR spectroscopy in neonates with HIE.

MATERIAL AND METHODS

Study Design: Prospective hospital-based observational study to correlate cerebral oxygenation by near infrared spectroscope (NIRS) and metabolic derangements by magnetic resonance spectroscopy (MRS) in

neonates with hypoxic ischemic encephalopathy (HIE).

Site and Period of Study: The study was conducted at Neonatal Intensive Care Unit (NICU) of Chhatrapati Shivaji Subharti Hospital, Meerut from July 2024 to February 2026.

Study Population: Neonates with clinical neurological dysfunction and a history of perinatal hypoxic-ischemic insult were assessed for HIE. Perinatal parameters recorded were need of resuscitation and Apgar score. Encephalopathy was graded according to Sarnat and Sarnat staging into Stage I (mild), Stage II (moderate) and Stage III (severe) based on neurological status.

Sample Size: A total of 38 neonates were enrolled by convenience sampling after obtaining written informed consent from the parent(s) or legally authorized guardian.

Inclusion Criteria:

- Neonates with HIE (Sarnat Stages I–III) admitted to the NICU.
- Neonates with associated hemodynamic compromise requiring clinical monitoring and/or resuscitative management.

Exclusion Criteria:

- Patients beyond the neonatal age group.
- Neonates with major congenital anomalies.
- Neonates with skin breakdown at the NIRS probe application site.

NIRS Monitoring Protocol: Cerebral regional oxygen saturation (rSO_2) was measured by an INVOS 5100 NIRS system, sensor on the forehead/scalp. Baseline rSO2 was taken before fluid correction/resuscitation. Serial monitoring was done during resuscitative management at about 1, 2, 4, 8, 12 and 24 hours and reassessment was done after hemodynamic stabilization.

Fluid-Resuscitation Protocol: Hemodynamically compromised neonates received isotonic crystalloids, either normal saline or Ringer's lactate, in documented volumes of 10–20 mL/kg according to clinical requirement. Response was assessed using heart rate, blood pressure, capillary refill time, SpO_2, peripheral perfusion, and cerebral rSO_2.

Therapeutic Hypothermia: Therapeutic hypothermia status was not captured as a separate study variable and therefore was not included in the analysis.

MRS Protocol: MRI/MRS was performed after clinical stabilization using a uMR780 3-Tesla system with T2/FLAIR, diffusion-weighted imaging (DWI), and apparent diffusion coefficient (ADC) sequences followed by proton MRS. MRS was performed between 28 and 72 hours of life. Spectroscopic assessment included lactate peak status (present/absent), Lactate/NAA ratio, and NAA status (normal/reduced) according to the documented MRS interpretation. Exact voxel location and predefined quantitative cut-offs for lactate peak and reduced NAA were not captured in the study dataset.

Ethical Considerations: Ethics committee approval was obtained before commencement of the study. Written informed consent was obtained from the parent(s) or legally authorized guardian, and confidentiality was maintained.

Statistical Analysis

Data were analyzed using appropriate descriptive and inferential statistical methods. Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The distribution of continuous variables was assessed before applying parametric tests. Differences in cerebral regional oxygen saturation (rSO_2) between neonates with and without a lactate peak on magnetic resonance spectroscopy (MRS) were evaluated using the independent-samples *t*-test. Changes in rSO_2 across the baseline, hemodynamic monitoring/resuscitation, and post-fluid-resuscitation measurements were assessed using an appropriate repeated-

measures analysis, with post-hoc pairwise comparisons where indicated. The association between cerebral rSO_2 and lactate peak status was evaluated using binary logistic regression, and odds ratios (ORs) with 95% confidence intervals (CIs) were reported. Receiver operating characteristic (ROC) curve analysis was used to assess the discriminatory performance of mean cerebral rSO_2 for lactate peak status, with the area under the curve (AUC), sensitivity, specificity, and accuracy reported. Two-sided *p* values <0.05 were considered statistically significant. Given the small sample size and imbalance between lactate-peak groups, regression and ROC findings were interpreted as exploratory and with caution.

RESULTS

Table 1. Distribution of Neonates According to Resuscitation Requirement (n = 38).

Variable	Category	Frequency (n)	% of Total
Resuscitation Required	No	8	21.1 %
	Yes	30	78.9 %

Table 1 shows the distribution of neonates according to resuscitation requirement. The study included 38 neonates of whom 30 (78.9%) required resuscitation and 8 (21.1%) did not require resuscitation. These results showed that majority of neonates had significant perinatal compromise.

Table 2. Distribution of Neonates According to Sarnat Stage (n = 38).

Variable	Category	Frequency (n)	% of Total
Sarnat Stage	I	8	21.1 %
	II	18	47.4 %
	III	12	31.6 %

Table 2 shows the distribution of neonates according to Sarnat stage. Among the 38 neonates, the majority were classified as Stage II HIE, 18 (47.4%), followed by Stage III, 12 (31.6%), and Stage I, 8 (21.1%), indicating predominance of moderate-to-severe HIE.

Table 3. Baseline Hemodynamic Profile of Neonates at Admission (n = 38)

Variable	Value
Heart rate (beats/min)	153.0± 15.31
Mean arterial pressure (mmHg)	41.74 ± 6.92
Capillary refill time (seconds)	3.32 ± 0.96
Oxygen saturation (SpO_2 %)	92.20 ± 4.22
Shock (Yes)	20(52.6 %)
Shock (No)	18 (47.4 %)

The baseline hemodynamic profile of the neonates at admission is shown in table 3. The mean heart rate was 153.0 ± 15.31 beats/min, mean arterial pressure 41.74 ± 6.92 mmHg and mean oxygen saturation 92.20 ± 4.22 %. Shock was observed in 52.6% neonates indicating significant hemodynamic compromise in the study population.

Table 4. Cerebral Near-Infrared Spectroscopy (NIRS) Monitoring Parameters in Neonates with HIE (n = 38).

Variable	N	Mean ± SD	Min–Max
Baseline Cerebral rSO_2 (%)	38	63.3 ± 4.75	56 – 73
rSO_2 during hemodynamic monitoring/resuscitation (%)	38	54.8 ± 8.97	44 – 73
rSO_2 After Fluid Resuscitation (%)	38	73.7 ± 3.35	68 – 82
Δ rSO_2 (Post – Baseline)	38	10.4 ± 3.72	3 – 14
Mean Cerebral rSO_2 (%)	38	64.0 ± 5.18	56.0 – 74.0

Table 4 presents cerebral NIRS parameters in the 38 neonates. Mean baseline cerebral rSO_2 was 63.3 ± 4.75%. During hemodynamic monitoring/resuscitative management, mean rSO_2 was 54.8 ± 8.97%, and following fluid resuscitation it increased to 73.7 ± 3.35%. Clinical shock was present in 20 (52.6%) neonates; however, NIRS monitoring was performed in all 38

neonates during the hemodynamic assessment/resuscitative period.

Table 5. Distribution of Neonates According to Improvement in Cerebral rSO_2 Following Fluid Resuscitation (n = 38)

Variable	N	Mean ± SD	Min–Max
Improvement in rSO_2	No	6	15.8 %
	Yes	32	84.2 %

Table 5 shows the distribution of neonates according to improvement in cerebral rSO_2 following fluid resuscitation. Among 38 neonates, improvement in cerebral oxygen saturation was observed in 32 (84.2%) neonates, while 6 (15.8%) showed no improvement, suggesting a favorable cerebral oxygenation response after hemodynamic stabilization.

Table 6. Magnetic Resonance Spectroscopy (MRS) Profile of Neonates (n = 38).

Variable	Category	N	Mean ± SD	Min–Max
Lactate/NAA Ratio	—	38	1.54 ±0.861	0.280 – 3.30
Timing of MRS (hours)	—	38	48.8 ± 11.3	28 – 72
Lactate Peak	Absent	8	21.1 %	
	Present	30	78.9 %	
NAA Status	Normal	8	21.1 %	
	Reduced	30	78.9 %	

Table 6 presents the MR spectroscopy profile of neonates with HIE. The mean lactate/NAA ratio was 1.54 ± 0.861, and MRS was performed at a mean age of 48.8 ± 11.3 hours. Lactate peak and reduced NAA levels were observed in 78.9% neonates, indicating significant cerebral metabolic injury.

Table 7. Comparison of Cerebral NIRS Parameters According to Lactate Peak Status on MR Spectroscopy (n = 38)

Variable	Lactate Peak Absent (n=8) Mean ± SD	Lactate Peak Present (n=30) Mean ± SD	t-value	p-value
Baseline Cerebral rSO_2 (%)	70.3 ± 2.12	61.5 ± 3.33	7.0224	<0.001
rSO_2 during hemodynamic monitoring/resuscitation (%)	70.3 ± 2.12	50.7 ± 4.29	12.3931[a]	<0.001
rSO_2 After Fluid Resuscitation (%)	73.6 ± 1.92	73.7 ± 3.67	−0.0801[a]	0.937
Mean Cerebral rSO_2 (%)	71.4 ± 2.05	62.0 ± 3.74	6.7878[a]	<0.001

Table 7, Association of cerebral NIRS parameters according to lactate peak status on MR spectroscopy. Neonates with lactate peak demonstrated significantly lower baseline cerebral rSO_2, rSO_2 during hemodynamic monitoring/resuscitation, and mean cerebral rSO_2 compared to those without lactate peak ($p<0.001$). However, post-resuscitation rSO_2 values were comparable between the two groups.

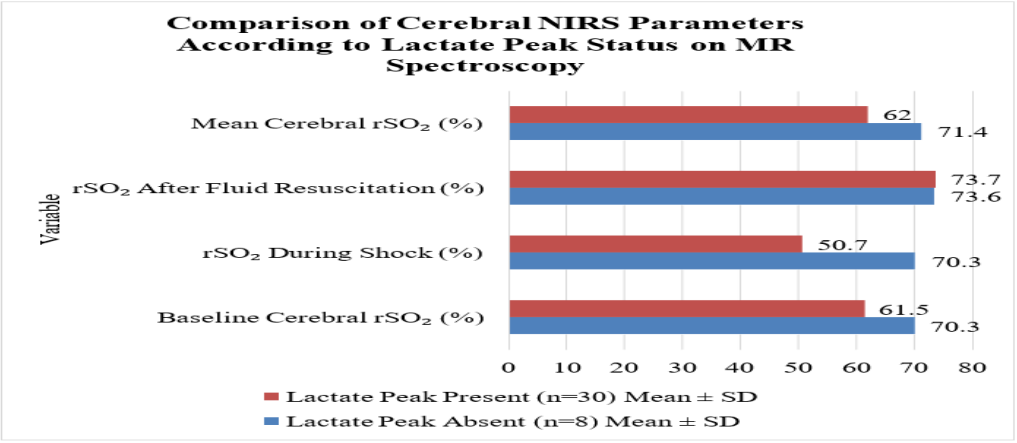

Figure 1: Comparison of cerebral NIRS parameters according to lactate peak status on MR spectroscopy.

Table 8. Binomial Logistic Regression of Mean Cerebral rSO_2 (%) for Predicting Lactate Peak Absent vs Present (n = 38).

A. Model Fit Measures

Model Fit Measure	Value
Deviance	8.72
AIC	12.7
McFadden R^2	0.777

B. Model Coefficients

Predictor	Estimate (β)	SE	Z	p-value	Odds Ratio (OR)
Intercept	−74.50	31.237	−2.38	0.017	4.44×10^{-33}
Mean Cerebral rSO_2 (%)	1.08	0.454	2.38	0.017	2.95 (95% CI: 1.21–7.17)

Model note: Estimates represent the log odds of Lactate Peak Absent (coded 1) versus Lactate Peak Present (coded 0).

C. Predictive Performance (Cut-off Probability = 0.5)

Measure	Value
Accuracy	0.947
Sensitivity	0.875
Specificity	0.967
AUC	0.992 (95% CI: –)

Table 8 demonstrates a significant association between mean cerebral rSO_2 and lactate peak status on MR spectroscopy (p=0.017). Higher cerebral rSO_2 values were associated with increased odds of absence of lactate peak (OR=2.95 (95% CI: 1.21–7.17)). The model showed high apparent discrimination, with 94.7% accuracy, 87.5% sensitivity, 96.7% specificity, and an AUC of 0.992 (95% CI: –). These estimates should be interpreted cautiously because the analysis included only 38 neonates, including 8 without a lactate peak.

DISCUSSION

Distribution of resuscitation requirement and Sarnat staging showed that 78.9% of neonates required active resuscitation at birth, of which 47.4% were Stage II HIE and 31.6% were Stage III HIE. Similar results were reported by Lally et al. [3] who found that moderate to severe encephalopathy was higher in neonates who underwent therapeutic hypothermia and metabolic neuroimaging. Goswami et al. [2] and Liu et al. [1] also reported poor perinatal adaptation with severe neurological compromise and need for increased resuscitation support. Severe HIE is associated with increased neurologic injury and abnormal cerebral oxygenation patterns Alderliesten et al. [5]. The lower proportion of mild HIE is probably related to the inclusion of clinically unstable neonates in need of intensive care monitoring.

The baseline hemodynamic profile indicated considerable circulatory compromise with a mean heart rate of 153.0 ± 15.31 beats/min, mean arterial pressure of 41.74 ± 6.92 mmHg, capillary refill time of 3.32 ± 0.96 seconds and 52.6% of neonates in shock. Similarly, Tierradentro-García et al. [7] obtained similar results and pointed out that systemic hypoperfusion and alterations in cerebral blood flow are aggravating factors of hypoxic-ischemic brain injury. Reduced cerebral oxygen metabolism, an indirect marker of circulatory instability, during the period of therapeutic hypothermia was associated with severe HIE. Conversely, Proisy et al. [10] reported that cerebral blood flow was increased during early reperfusion despite the persistence of the neurological injury. Shetty et al. [11]

Cerebral NIRS monitoring showed mean cerebral rSO_2 decreased from baseline (63.3 ± 4.75%) to during hemodynamic monitoring/resuscitation (54.8 ± 8.97%) but improved to 73.7 ± 3.35% after fluid resuscitation; and 84.2% of neonates showed improved cerebral oxygenation. Similar findings were described by Mitra et

al. [12] who showed impaired cerebral oxygenation and metabolic disturbance during therapeutic hypothermia that normalized after stabilization. Jeon [4] also highlighted the potential of NIRS to provide continuous bedside monitoring of neonatal tissue perfusion and oxygenation. Therefore, the patterns of cerebral oxygenation were associated with the severity of neurological injury and neurodevelopmental outcome as described by Alderliesten et al. [5] and Sutin et al. [6] In some circumstances, however, hyperoxygenation with reperfusion may be a marker of reduced capacity of cerebral oxygen utilization. This was suggested by Costa et al. [9]

MR spectroscopy profile showed mean lactate/NAA ratio of 1.54 ± 0.861. In 78.9 % of the neonates a lactate peak and a decrease in NAA levels were observed. Similar findings were reported by Lally et al. [3] who found higher lactate and lower N-acetylaspartate concentrations to be good predictors of poor neurological outcome in neonatal encephalopathy. High lactate accumulation and low NAA level are also markers of severe metabolic brain injury in HIE, reported by Parmentier et al. [8]. Mitra et al. [12] found worsening of cerebral metabolic dysfunction with increasing lactate/NAA ratios during rewarming after hypothermia. MRS was performed relatively early and allowed the assessment of the evolving metabolic injury during the acute phase of HIE.

Neonates with lactate peak on MR spectroscopy had significantly lower cerebral NIRS parameters when compared to neonates without lactate peak at baseline, during hemodynamic monitoring/resuscitation and mean cerebral rSO2 ($p<0.001$). Similar findings were also reported by Mitra et al. [12] who reported a decrease in cerebral oxygenation with increasing lactate/NAA ratio in severe HIE. Severe neurological injury in neonates is associated with abnormal patterns of cerebral oxygen saturation (Alderliesten et al. [5]. Impaired cerebral metabolic recovery and abnormal cerebral oxygenation were related to worse outcomes (Sutin et al. [6]. rSO2 values in the brain were comparable between groups following resuscitation and stabilization of hemodynamics.

Mean cerebral rSO2 was significantly associated with lactate peak status by binomial logistic regression (OR=2.95, 95% CI: 1.21–7.17, p=0.017). Although the apparent discrimination is high, the results should be interpreted with caution given the small sample size (n=38), in particular the small number of neonates without a lactate peak (n=8), and need to be validated in larger cohorts. Similar findings were reported by Lally et al. [3] who demonstrated high prognostic accuracy of metabolic neuroimaging biomarkers in neonatal encephalopathy. Sutin et al. [6] also showed that the cerebral metabolic and oxygenation parameters were good predictors of neurodevelopmental outcomes. Liu et al. [1] further demonstrated the advantage of advanced neuroimaging and neurophysiological markers over traditional clinical assessment in prognosis prediction.

CONCLUSION

This single-centre observational study demonstrated a significant association between cerebral NIRS parameters and metabolic abnormalities identified on MR spectroscopy in neonates with hypoxic ischemic encephalopathy. Neonates with a lactate peak had lower cerebral rSO_2 values during hemodynamic monitoring/resuscitation. These findings suggest that NIRS may have potential as an adjunctive bedside monitoring modality; however, the study does not establish NIRS as a surrogate for MRS or demonstrate its prognostic value. Larger multicentre studies with clinical and neurodevelopmental follow-up are required to determine its role in risk stratification and prognostication.

Declaration

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Conflict of Interest: The authors declare no conflict of interest.

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