Serum Vitamin D Levels in Term Neonates with Perinatal Asphyxia: A Case-Control Study.

Dr. Asgar Ali, Dr. Manmeet Kaur Sodhi, Dr. Jaspal Singh, Dr. NihalRehim, Dr. Ajaydeep.
Designation of Authors: Junior Resident, Professor & HOD, professor, junior resident, junior resident.
Institute: Department of Pediatrics, Government Medical College, Amritsar

Address for correspondence
Dr. Asgar Ali
Junior Resident, Department of Pediatrics, Government Medical College, Amritsar.
Email –debacktrack.aa@gmail.com
Mobile - 7006581859

Received : 06 JANUARY 2026; Accepted: 25 MAY 2026

Abstract

Background: Perinatal asphyxia (PA) continues to be a major contributor to neonatal morbidity and mortality, particularly in low- and middle-income countries. Its most severe neurological consequence, hypoxic-ischemic encephalopathy (HIE), often leads to lifelong neurodevelopmental impairments. Emerging evidence highlights a potential neuroprotective role of vitamin D, which regulates inflammatory pathways, oxidative stress, and neuronal survival. However, limited data exist on the association between vitamin D deficiency and neurological outcomes in asphyxiated term neonates in the Indian setting.

Objectives: To compare serum 25-hydroxyvitamin D [25(OH)D] levels between term neonates with perinatal asphyxia and healthy controls, and to evaluate the relationship between vitamin D deficiency and the severity of HIE, mortality, and early neurological outcomes.

Methods: A prospective observational case–control study was conducted in the NICU of Government Medical College, Amritsar, including 150 term neonates with PA and 150 healthy term controls. Clinical assessment, HIE staging (Sarnat and Sarnat), cranial ultrasonography, and developmental evaluation (TDSC, DDST) were performed. Serum 25(OH)D levels were measured within 24 hours of birth. Vitamin D deficiency was defined as <12 ng/mL. Data were analysed using chi-square and t-tests, with $p < 0.05$ considered significant.

Results: Asphyxiated neonates had significantly lower mean serum 25(OH)D levels than controls (13.06 ± 5.28 vs 19.71 ± 7.61 ng/mL, $p < 0.001$). Vitamin D deficiency (<12 ng/mL) was more prevalent among cases (52.7% vs 14%). Severe HIE (Stage II) was significantly more common in vitamin D–deficient infants (62.2% vs 19.7%, $p = 0.001$). Mortality was markedly higher in deficient neonates (32.9% vs 7.0%, $p = 0.004$). Developmental delay at 12 weeks correlated strongly with higher HIE stages.

Conclusion: Vitamin D deficiency is significantly associated with increased HIE severity, higher mortality, and poorer early neurological outcomes in term neonates with perinatal asphyxia. Routine screening and early correction of vitamin D may offer a simple, cost-effective strategy to improve neurodevelopmental outcomes.

How to cite this article:
Ali A, Sodhi MK, Singh J, Rehim N, Ajaydeep. Serum Vitamin D Levels in Term Neonates with Perinatal Asphyxia: A Case-Control Study. Indian J Dev Behav Pediatr. 2026;Vol 4(2):19-27. https://doi.org/10.5281/zenodo.20796566

Keywords:
- Vitamin D
- Perinatal Asphyxia
- Hypoxic-Ischemic Encephalopathy
- Neonatal Neurodevelopment
- Biomarker
- Neonatal Mortality

Introduction:

Perinatal asphyxia (PA) is characterised by impaired gas exchange in the foetus or newborn during the perinatal period, leading to challenges in initiating and sustaining breathing.[1] It remains one of the leading causes of neonatal and under-five mortality, accounting for about 23 % of neonatal deaths globally and 23–25 % in India.[2] According to the National Neonatal Perinatal Database, PA contributes to 3,00,000–3,50,000 of the 1.2 million annual neonatal deaths. In high-resource nations, two cases occur per 1000 live births, while in low-resource settings, the rate is nearly tenfold higher. [3] When the placenta or lungs can't exchange gases properly, not getting enough oxygen can cause hypoxaemia, hypercapnia, and lactic acidosis to get worse.

Perinatal asphyxia results in hypoxic-ischemic injury to various organs, including the kidneys, central nervous system (CNS), heart, lungs, and intestines. The outcome largely depends on the extent of organ dysfunction. [4] The most severe neurological manifestation is hypoxic-ischemic encephalopathy (HIE)—a spectrum of CNS injury leading to motor, sensory, cognitive, and psychological impairment. Around 15–20 % of neonates with HIE die in the neonatal period, while 25 % of survivors develop long-term sequelae such as cerebral palsy or global developmental delay. [4, 5]

The pathophysiology of HIE involves two major phases of neuronal injury. During the initial injury, diminished oxygen and blood flow result in energy depletion, ionic imbalance, lactic acidosis, and excitotoxicity mediated by glutamate accumulation. [6, 7] Subsequently, the brain and neurones experience secondary injury characterised by oxidative stress, inflammation, and reperfusion. [5,7] Although neurogenesis and synaptic remodelling occur, the restoration process is often insufficient, resulting in persistent neurological impairment.[7, 8] Clinically, HIE is graded as mild, moderate, or severe based on Sarnat and Sarnat criteria.[9]

Vitamin D is a fat-soluble hormone essential for numerous physiological processes. It undergoes hepatic and renal hydroxylation to produce its active form, 1,25-dihydroxyvitamin D (calcitriol), which interacts with vitamin D receptors (VDRs) to modulate gene expression. [10, 11] Vitamin D deficiency is a worldwide issue, impacting 60–80% of Indian neonates and 80–90% of expectant women in South Asia. [12, 13] Vitamin D receptors are present in brain regions associated with motor and cognitive functions, where they modulate neurogenesis, synaptic plasticity, and neuroprotection. [14] Experimental research demonstrates that vitamin D mitigates oxidative stress and inflammation through the regulation of cytokines including TNF-α, IL-6, and IL-1. [15] A deficiency of vitamin D may exacerbate HIE outcomes by increasing oxidative stress, disrupting calcium homeostasis, amplifying excitotoxicity, and compromising the integrity of the blood–brain barrier. [15] Considering its potential neuroprotective effects, assessing vitamin D levels in neonates with perinatal asphyxia may offer valuable insights into disease severity and prognosis.

Hence, the present prospective observational comparative study was undertaken to compare vitamin D deficiency [serum 25(OH)D < 12 ng/mL] in full-term neonates with perinatal asphyxia and healthy term controls, and to assess its predictive value for adverse neurological outcomes.

Materials and Methods:

Study Design and Setting:

A prospective, observational, case–control study was conducted in the Department of Paediatrics, Neonatal Intensive Care Unit (NICU), Government Medical College, Amritsar. The study enrolled full-term neonates (≥37 weeks gestation) admitted with a diagnosis of perinatal asphyxia, as well as healthy term controls. Ethical clearance was obtained from the Institutional Ethics Committee (Ethical Clearance No. 395/D-26/2022), and written informed consent was obtained from parents or legal guardians.

Study Population:

The study included **150 term neonates with perinatal asphyxia (cases)** and **150 healthy term neonates (controls)**. Cases were identified when two or more of the following criteria were present:

- **Clinical neurological signs:** irritability, lethargy, hypotonia, hypoactivity, or seizures.
- **Supportive parameters:** Apgar ≤ 5 at 5 minutes, need for positive-pressure ventilation> 1 minute, resuscitation beyond routine care, umbilical cord arterial pH < 7.0 or base deficit > 12 mmol/L, or evidence of multisystem involvement.

Inclusion Criteria:

Inclusion criteria included term neonates with gestational age ≥ 37 weeks, Apgar score < 3 at 5 minutes, evidence of hypoxic-ischemic encephalopathy on modified Sarnat staging, presence of neurological sequelae such as seizures, hypotonia or coma, evidence of multiorgan involvement, and availability of parental informed consent.

Exclusion Criteria:

Exclusion criteria included neonates with congenital anomalies, intrauterine growth restriction, sepsis, infants of diabetic mothers, chromosomal disorders, or those exposed antenatally to antiepileptics, opioids, or magnesium sulphate.

Neonatal sepsis was excluded on the basis of maternal and neonatal sepsis risk factors, clinical evaluation, septic screening parameters, and blood culture results whenever indicated.

Mode of delivery was not used as an inclusion or exclusion criterion. Eligible neonates were enrolled irrespective of route of delivery.

Clinical Assessment:

All enrolled neonates underwent detailed antenatal ,intrapartum and perinatal history assessment, physical examination, and neurological assessment including tone, activity, reflexes, and seizure activity. Although advanced fetal surveillance parameters such as non-stress testing, biophysical profile and Doppler studies were not uniformly available for all participants, clinically significant antenatal hypoxia was assessed through available obstetric records and neonatal evaluation. The possibility of subclinical antenatal hypoxia cannot be completely excluded.In asphyxiated newborns, the severity of hypoxic-ischemic encephalopathy (HIE) was assessed utilising the Sarnat and Sarnat classification [9]. Neurological outcomes were evaluated at discharge and at 12 weeks of age utilising the Trivandrum Developmental Screening Chart (TDSC) and the Denver Developmental Screening Test (DDST)[16,17]. Neonates who did not achieve age-appropriate milestones in one or more domains were classified as screen-positive for developmental delay.

Laboratory Investigations:

Under aseptic precautions, **3 ml of venous blood** was collected within the first 24 hours of life for complete blood count, renal function tests, and serum 25-hydroxyvitamin D [25(OH)D] estimation. Vitamin D levels were measured by **chemiluminescence immunoassay**. Vitamin D status was classified as:

- **Deficient:**< 12 ng/mL
- **Insufficient:** 12–20 ng/mL
- **Sufficient:**> 20 ng/mL

All neonates admitted with perinatal asphyxia received standard NICU care including monitoring and correction of hypoglycemia, electrolyte disturbances and other metabolic abnormalities according to institutional protocols. These variables were not analysed independently because the primary objective was evaluation of vitamin D levels and neurological outcomes.

Statistical Analysis:

Data were entered in SPSS version 22 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation and compared using the independent t-test. Categorical variables were presented as frequency and percentage and analyzed using the Chi-square test. The proportion of vitamin D deficiency and mean vitamin D levels were compared between groups. Statistical significance was defined at $p< 0.05$.

Results:

The baseline demographic variables—gender, birth weight, and gestational age—exhibit no significant differences between cases and controls, so verifying the comparability of both groups at baseline ($p > 0.05$). However, serum 25(OH)D levels were considerably lower in asphyxiated newborns than in healthy controls ($p < 0.001$). More than half (52.7%) of the asphyxiated newborns were lacking in vitamin D, while only 14% of the controls were. Clinically, 80% of individuals progressed to some stage of HIE, with the largest percentage observed in HIE II (34.1%). Mortality rates were significantly elevated in HIE III (16.7%) and were also present in HIE II (4%), demonstrating a robust correlation between low vitamin D levels, the severity of HIE, and mortality.(Table 1)

A strong and statistically significant link ($p < 0.001$ for all measurements) can be made between the stage of HIE and neurological disorders. The severity of HIE was significantly correlated with abnormal cranial ultrasonography findings, impaired higher neurological functions, unusual motor and reflex responses, and developmental delays at 12 weeks. HIE II babies showed the highest issues, with 93.8% having more neurological disorders and 75.5% having developmental delays. Infants without HIE had minimal abnormalities, so validating the predictive relevance of HIE staging for long-term neurodevelopment.(Table 2). Neonates with vitamin D deficiency (<12 ng/mL) demonstrated significantly increased mortality rates (32.9% versus 7.0%, $p = 0.004$) and more severe manifestations of hypoxic-ischemic encephalopathy (HIE). HIE II was much more common in vitamin D deficient neonates (62.2% vs 19.7%, $p = 0.001$), although non-deficient infants showed a notably greater prevalence of cases classified as No HIE (40.9%). The deficient group had a higher incidence of abnormal neurological examinations at discharge; however, this disparity was not statistically significant ($p = 0.074$). These data indicate that vitamin D deficiency is significantly associated with adverse clinical outcomes and increased severity of HIE.(Table 3)

Table 1. Baseline Characteristics, Vitamin D Status, and Clinical Outcomes of Study Population

Parameter	Category / Level	Cases (n = 150) N (%) / Mean ± SD	Controls (n = 150) N (%) / Mean ± SD	*p*-value
Gender	Female	66 (44.0 %)	70 (46.7 %)	0.643
	Male	84 (56.0 %)	80 (53.3 %)	
Birth Weight (kg)	< 2.5 kg	37 (24.7 %)	40 (26.7 %)	0.600
	2.5 – 3.0 kg	78 (52.0 %)	82 (54.7 %)	
	> 3.0 kg	35 (23.3 %)	28 (18.7 %)	
	Mean ± SD	2.79 ± 0.33	2.77 ± 0.32	
Gestational Age (weeks)	37	49 (32.7 %)	52 (34.7 %)	0.722
	38	59 (39.3 %)	55 (36.7 %)	
	39	25 (16.7 %)	29 (19.3 %)	
	40	16 (10.7 %)	14 (9.3 %)	
	41	1 (0.7 %)	0 (0.0 %)	
	Mean ± SD	38.07 ± 0.99	38.03 ± 0.95	

Serum 25(OH)D (ng/mL)	< 12 (Deficient)	79 (52.7 %) 9.31 ± 1.82	21 (14.0 %) 10.49 ± 1.07	0.005 *
	12 – 20 (Insufficient)	55 (36.7 %)	69 (46.0 %)	
	> 20 (Sufficient)	16 (10.7 %)	60 (40.0 %)	
	Mean ± SD (overall)	13.06 ± 5.28	19.71 ± 7.61	<0.001
HIE Outcome	No HIE	30 (20.0 %)	–	–
	HIE I	43 (28.7 %)	–	–
	HIE II	52 (34.1 %)	–	–
	HIE III	25 (16.1 %)	–	–
	Mortality (HIE II)	6 (4.0 %)	–	–
	Mortality (HIE III)	25 (16.7 %)	–	–
	Total	150 (100 %)	150 (100 %)	

* $p < 0.05$ p**< 0.01** *p**< 0.001**

This table presents the baseline demographic characteristics, vitamin D status, and major clinical outcomes among term neonates with perinatal asphyxia (cases) compared with healthy term neonates (controls).

Table 2. Correlation of Clinical Outcomes and Neurological Parameters in Term Neonates With Perinatal Asphyxia (n = 119 Survivors)

Outcome (HIE Stage)	Cranial USG Abnormal n (%)	Higher Neurological Function Abnormal n (%)	Motor System Abnormal n (%)	Reflexes Abnormal n (%)	Developmental Delay Present n (%)	Abnormal 12-Week Outcome n (%)
No HIE (n = 30)	2 (2.9 %)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)	1 (1.6 %)
HIE I (n = 43)	25 (35.7 %)	3 (6.3 %)	25 (39.1 %)	23 (37.1 %)	13 (24.5 %)	23 (37.7 %)
HIE II (n = 46)	43 (61.4 %)	45 (93.8 %)	39 (60.9 %)	39 (62.9 %)	40 (75.5 %)	37 (60.7 %)
HIE III (n = 0)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)
Total (n = 119)	70 (100 %)	48 (100 %)	64 (100 %)	62 (100 %)	53 (100 %)	61 (100 %)
p-value	0.001 ***	0.001 ***	0.001 ***	0.001 ***	0.001 ***	0.001 ***

This table describes the relationship between HIE staging and major neurological outcomes among surviving neonates with perinatal asphyxia (n = 119). Increasing HIE severity is associated with progressively worsening neurological abnormalities and developmental outcomes.

No HIE III neonates survived to undergo neurological assessment and developmental follow-up.

Table 3: Clinical Outcomes Between Vitamin D Deficient (<12 ng/mL) and Non-Deficient (>12 ng/mL) Neonates

Variables	Vitamin D Deficient (<12 ng/mL) (n = 79)	Vitamin D Non-Deficient (>12 ng/mL) (n = 71)	*p*-value
Mortality n (%)	26 (32.9)	5 (7.0)	0.004*
Abnormal neurological examination at discharge n (%)	32 (60.3)	29 (43.9)	0.074
HIE I n (%)	12 (22.6)	31 (47.0)	
HIE II n (%)	33 (62.2)	13 (19.7)	0.001*
HIE III n (%)	0 (0.0)	0 (0.0)	
No HIE n (%)	3 (5.7)	27 (40.9)	

***p < 0.05 significant

This table compares major clinical outcomes between vitamin D–deficient neonates (<12 ng/mL) and those with adequate vitamin D levels (>12 ng/mL) among infants with perinatal asphyxia.

All neonates with HIE stage III expired during hospitalization and therefore were not represented in the comparison of HIE stages according to vitamin D status.

Discussion:

Perinatal asphyxia remains a major cause of neonatal morbidity and mortality, particularly in developing countries. Its most serious sequel, hypoxic-ischemic encephalopathy (HIE), contributes substantially to long-term neurological disability. Emerging evidence indicates that vitamin D plays an important neuroprotective role through antioxidant, anti-inflammatory, and anti-apoptotic mechanisms that enhance neuronal resilience against hypoxic-ischemic injury.

The present prospective comparative study, *Serum Vitamin D Levels in Term Neonates With Perinatal Asphyxia*, evaluated 150 term neonates with asphyxia and 150 healthy controls admitted to the NICU of Government Medical College, Amritsar. Baseline demographic characteristics—including gestational age, birth weight, and gender—were comparable between the two groups, minimizing confounding factors.

Although congenital infections, endocrine disorders and rare inborn errors of metabolism may mimic hypoxic-ischemic encephalopathy, strict inclusion and exclusion criteria were employed to minimise these potential confounders. Universal TORCH screening, thyroid function testing and metabolic screening were not routinely performed in all participants and therefore their influence cannot be completely excluded.

Hyperbilirubinemia was not included as a predefined study variable because the primary objective was evaluation of vitamin D status in perinatal asphyxia. Neonates developing jaundice during hospitalization were managed according to standard neonatal guidelines and no cases of clinically evident bilirubin encephalopathy were documented during follow-up.

Our findings demonstrated significantly lower mean serum 25(OH)D levels among asphyxiated neonates (13.06 ± 5.28 ng/mL) compared to controls (19.71 ± 7.61 ng/mL; $p = 0.001$). In the deficient range (< 12 ng/mL), values were significantly lower in cases (9.3 ng/mL) compared to controls (10.5 ng/mL; $p = 0.005$). In the sufficient range (> 20 ng/mL), controls exhibited higher concentrations (27.6 ng/mL) than cases (24.6 ng/mL; $p = 0.032$). These results parallel those of **Saleh et al. (2019)** [18], who observed a high prevalence of vitamin D deficiency among HIE neonates, and of **Batra et**

al. (2023)[19], who reported significantly lower vitamin D levels in neonates with severe perinatal depression compared with healthy peers (7.5 ± 3.53 vs 20.23 ± 12.70 ng/mL; $p< 0.001$). **Elgayar et al. (2024)**[20] further demonstrated a progressive decline in vitamin D concentration with increasing HIE severity, supporting its role as a modifiable neuroprotective factor.

In the present study, 70 % of affected neonates developed HIE, distributed as Stage I (28.7 %), Stage II (34.7 %), and Stage III (16.7 %), while 20 % had no encephalopathy. Mortality correlated strongly with disease severity: 4 % of deaths occurred in HIE II and 16.7 % in HIE III. Comparable gradients of mortality with advancing HIE stage were reported by **Elgayar et al. (2024)**[20] (0 % – 13.3 % – 36.6 %) and **Alshtewi et al. (2021)**[21], who identified both vitamin D deficiency and advanced HIE as predictors of neonatal death. The significantly higher prevalence of vitamin D deficiency among neonates with severe HIE in our study reinforces the link between low 25(OH)D levels and poor survival, possibly due to impaired regulation of inflammatory cytokines and mitochondrial protection during hypoxia.

HIE severity in the present study was classified using the validated Sarnat and Sarnat staging system. EEG was not routinely performed in all enrolled neonates and therefore EEG findings could not be correlated with vitamin D status or neurological outcomes.

Cranial ultrasound abnormalities were observed in 70 of 119 (58.8 %) neonates with perinatal asphyxia, predominating in HIE II (61.4 %) and HIE I (35.7 %) with a strong statistical association ($p = 0.001$). Similar trends were noted by **Elgayar et al. (2024)**[20] and **Alshtewi et al. (2021)**[21], who found that neonates with low vitamin D levels had more extensive neuroimaging abnormalities. The significant association between cranial ultrasonography abnormalities and adverse neurological outcomes further supports the utility of cranial ultrasound as a readily available bedside tool for risk stratification. Significant intracranial pathology, including hemorrhagic lesions when present, would have been identified through neuroimaging evaluation.These findings highlight the utility of cranial USG as a rapid, non-invasive adjunct in grading hypoxic brain injury and underscore the biochemical–radiological concordance of vitamin D deficiency with cerebral insult severity.

Neurological evaluation at discharge revealed a graded relationship between HIE stage and impairment of higher functions, motor activity, and reflexes. In HIE I, 6 % showed abnormal higher function, 39 % motor abnormality, and 37 % abnormal reflexes; the figures escalated to 94 %, 61 %, and 63 %, respectively, in HIE II ($p < 0.001$). No survivors were available in HIE III for assessment. These findings mirror the progression of hypoxic injury and align with reports by **Saleh et al. (2019)**[18] and **Batra et al. (2023)**[19], who documented severe neurodevelopmental deficits among vitamin D–deficient asphyxiated neonates.

Developmental follow-up at 12 weeks showed that 53 (44.5 %) infants exhibited delay, predominantly among HIE II (75.5 %) and HIE I (24.5 %) cases; none were from the no-HIE group. The relationship between developmental delay and HIE stage was highly significant ($p = 0.001$). Similar observations were made by **Elgayar et al. (2024)**[20] and **Batra et al. (2023)**[19], who found delayed milestone attainment in vitamin D–deficient neonates with moderate HIE, suggesting that low vitamin D may exacerbate early neurodevelopmental impairment.

Vitamin D deficiency was also strongly linked with mortality and adverse neurological outcomes. Among vitamin D–deficient neonates (< 12 ng/mL), mortality was 32.9 %, compared with 7.0 % in the non-deficient group ($p = 0.004$). Although the prevalence of abnormal neurological examination at discharge was higher in the deficient group (60.3 % vs 43.9 %), the difference was not statistically significant. However, severe encephalopathy (HIE II) predominated in vitamin D–deficient neonates (62.2 % vs 19.7 %; $p = 0.001$), whereas no HIE was

far more frequent among non-deficient infants (40.9 % vs 5.7 %). These findings demonstrate a clear association between hypovitaminosis D and worsening encephalopathy, emphasizing its potential role as a prognostic biomarker. Comparable conclusions were reached by **Mutlu et al. (2016)**[22], who observed higher neurological complication rates in vitamin D–deficient neonates, and by **Hagag et al. (2019)**[23], who reported improved neurodevelopmental outcomes following vitamin D supplementation in HIE cases.

Overall, the present study supports the growing consensus that vitamin D deficiency is significantly associated with greater severity of HIE, increased mortality, and poorer neurological performance. The **biologic plausibility** is underpinned by vitamin D's regulation of calcium homeostasis, neurotrophic factors, and anti-inflammatory pathways that mitigate hypoxic-ischemic injury. Early identification and correction of deficiency may therefore offer an adjunctive avenue for improving neurodevelopmental outcomes in at-risk neonates.

Limitations:

The study was conducted at a single tertiary-care center with neurodevelopmental follow-up limited to 12 weeks. Universal screening for congenital infections, thyroid dysfunction, and inborn errors of metabolism was not performed in all participants, and advanced antenatal fetal surveillance data were not uniformly available. Furthermore, routine EEG monitoring and advanced neuroimaging modalities such as MRI were not available for all neonates. These limitations should be considered while interpreting the observed association between vitamin D status and neurological outcomes.

Conclusion:

This study demonstrates a significant association between vitamin D deficiency and adverse outcomes in term neonates with perinatal asphyxia. Serum 25(OH) vitamin D levels were markedly lower in asphyxiated neonates than in healthy controls. Among affected infants, 78% developed hypoxic-ischemic encephalopathy (HIE), with disease severity correlating with both mortality and neurodevelopmental delay. Vitamin D–deficient neonates (<12 ng/mL) showed higher rates of HIE stages II–III, increased mortality (32.9% vs 7.0%), and more frequent neurological and developmental abnormalities.

These findings suggest that vitamin D deficiency may aggravate hypoxic-ischemic brain injury by enhancing oxidative stress and inflammation. Routine screening and correction of vitamin D deficiency in neonates with perinatal asphyxia may help mitigate neurological sequelae. Further interventional studies are warranted to explore its potential therapeutic and preventive role.

Conflict of Interest: None

Funding: None

References

1. Technical Working Group WHO. Postpartum care of the mother and newborn: a practical guide. Birth. 1999;26:255-8.
2. Raj P. Clinical profile and neurobehaviour at discharge of term neonates with perinatal asphyxia: a prospective observational study. Int J Contemp Med Res. 2016;3:3073-6.
3. Victor S, Rocha-Ferreira E, Rahim A, Hagberg H, Edwards D. New possibilities for neuroprotection in neonatal hypoxic-ischemic encephalopathy. Eur J Pediatr. 2022;181:875-87.
4. Rees S, Azzopardi D, Luntamo M. Pathophysiology of neonatal brain injury: causes, mechanisms, and strategies for therapeutic intervention. Dev Neurosci. 2011;33:169-77.
5. Odd D, Heep A, Luyt K, Draycott T. Hypoxic-ischemic brain injury: planned delivery before intrapartum events. J Neonatal Perinatal Med. 2017;10:347-53.
6. Tetorou K, Sisa C, Iqbal A, Dhillon K, Hristova M. Current therapies for neonatal hypoxic-ischaemic and infection-sensitised hypoxic-ischaemic brain damage. Front Synaptic Neurosci. 2021;13:709301.

7. Cowan F, Barks JD, Shankaran S. Erythropoietin and other neuroprotective agents in neonatal hypoxic-ischemic encephalopathy. Neonatology. 2018;113:10-7.

8. Shankaran S, Pappas A, McDonald SA. Hypothermia for neonates with hypoxic-ischemic encephalopathy. N Engl J Med. 2015;372:1875-83.

9. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress: a clinical and electroencephalographic study. Arch Neurol. 1976;33:667-74.

10. Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357:266-81.

11. Saraf R, Morton S, Camargo C, Grant C. Global summary of maternal and newborn vitamin D status: a systematic review. Matern Child Nutr. 2016;12:647-68.

12. Sahu M, Muktan D, Kapoor S. Vitamin D deficiency in neonates: a study from India. Pediatr Res. 2009;65:182-8.

13. Rizvi SI, Jahan S. Vitamin D deficiency in pregnancy: impact on maternal and neonatal outcomes. J Matern Fetal Neonatal Med. 2016;29:1-7.

14. Rudolf MC, Smith CM. The neuroprotective effects of vitamin D and its role in neonates with hypoxic-ischemic encephalopathy. Neonatol Res. 2016;64:215-24.

15. Hochhauser M, Ziv E. Vitamin D deficiency and neonatal brain injury: review of the current literature. Pediatr Neurol. 2018;80:16-24.

16. Nair MKC, George B, Philip E. Trivandrum Developmental Screening Chart. Indian Pediatr. 1991;28:869-72.

17. Frankenburg WK, Dodds JB, Archer P, Shapiro H, Bresnick B. The Denver II: a major revision and restandardization of the Denver Developmental Screening Test. Pediatrics. 1992;89:91-7.

18. Saleh SM, Al-Sharkawi EA, Nasef ZA, Sabry NM. The status of vitamin D in neonates with hypoxic-ischemic encephalopathy. Ann Neonatol J. 2019;2:14-9.

19. Batra P, Singh P, Ahmed RS, Harit D. Serum vitamin D status in full-term neonates with severe perinatal depression. J Neonatal Perinat Med. 2023;16:257-64.

20. Elgayar AA, El-Sharkawy HM, El-Bindary AS, Nassar MA, Hamza MB. Evaluation of serum vitamin D level in full-term neonates with hypoxic-ischemic encephalopathy. Alex J Pediatr. 2024;37:8-13.

21. Alshtewi A, Khater N, AbdElhamed D, Hamed M. Assessment of 25(OH) vitamin D in neonates with hypoxic-ischemic encephalopathy. Egypt J Hosp Med. 2021;85:3503-8.

22. Mutlu M, Sarıaydın M, Aslan Y, Kader Ş, Dereci S, Kart C, et al. Status of vitamin D, antioxidant enzymes, and antioxidant substances in neonates with neonatal hypoxic-ischemic encephalopathy. J Matern Fetal Neonatal Med. 2016;29:2259-63.

23. Hagag AA, El-Frargy MS, Abd El-Latif AE. Vitamin D as an adjuvant therapy in neonatal hypoxia: is it beneficial? EndocrMetab Immune Disord Drug Targets. 2019;19:341-8.