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Original Article

ASSESSMENT OF THYROID FUNCTIONS IN NEONATES WITH BIRTH ASPHYXIA

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ABSTRACT

Background: Perinatal asphyxia is a major cause of neonatal morbidity and mortality, often leading to multi-organ dysfunction. It can alter the hypothalamic-pituitary-thyroid axis, affecting thyroid hormones (T3, T4, TSH) crucial for neurodevelopment. This study aims to assess thyroid hormone levels in neonates with birth asphyxia at a tertiary care centre of southern Rajasthan.

Methods: A hospital-based case-control study was conducted in the NICU of RNT Medical College, Udaipur, over one year. Seventy-five term neonates with perinatal asphyxia (cases) and seventy-five healthy term neonates (controls) were enrolled. Cases were diagnosed based on clinical criteria and Apgar score <7 at 10 minutes. Serum T3, T4, and TSH levels were measured within 24 hours and at 72 hours of life using chemiluminescent immunoassay. Data were analysed using appropriate statistical tests, with $p < 0.05$ considered significant.

Results: Within the first 24 hours of life, mean serum levels of T3, T4, and TSH were comparable between asphyxiated and control neonates, with no statistically significant differences observed (T3: 2.39 ± 0.54 vs 2.31 ± 0.71 nmol/L, ; T4: 116.67 ± 24.18 vs 114.10 ± 21.59 nmol/L, TSH: 6.91 ± 1.46 vs 6.92 ± 1.35 mIU/L). By 72 hours, a significant decline in thyroid hormone levels was noted in asphyxiated neonates compared to controls. Mean T3 and T4 concentrations were markedly lower in cases (T3: 1.51 ± 0.73 vs 2.19 ± 0.69 nmol/L; T4: 76.82 ± 25.50 vs 117.27 ± 22.70 nmol/L). TSH levels also decreased significantly in cases relative to controls (5.97 ± 2.81 vs 7.46 ± 2.15 mIU/L), suggesting a blunted hypothalamic-pituitary-thyroid response.

Conclusion: Perinatal asphyxia significantly suppresses thyroid function by 72 hours of life, as evidenced by a marked reduction in T3, T4, and TSH levels in affected neonates compared to controls. These findings indicate a blunted hypothalamic-pituitary-thyroid response, highlighting the importance of monitoring thyroid status in asphyxiated term neonates for timely intervention.

Keywords: *Perinatal asphyxia; Thyroid hormones; Term neonates; Hypothalamic-pituitary-thyroid axis*

INTRODUCTION

According to the WORLD HEALTH ORGANISATION (WHO), perinatal asphyxia is the failure to initiate and sustain breathing (1). Perinatal asphyxia is the third major cause of neonatal mortality in India (2). According to the WHO, around 4 million babies develop birth asphyxia, and asphyxiated newborns may develop severe consequences such as epilepsy, cerebral palsy, developmental delay, and mental retardation. Furthermore, of 1.2 million neonatal deaths in India, 300,000-350,000 babies die due to perinatal asphyxia mostly within the first 3 days of life (3).

American Academy of Paediatrics (AAP) and American College of Obstetrics and Gynaecology define birth asphyxia as the presence of all of the following criteria:

a) Profound metabolic or mixed acidaemia (pH <7.00) in umbilical artery blood sample, if obtained
b) Persistence of Apgar score of 0–3 for longer than 5 min
c) Neonatal neurologic sequelae (e.g., seizures, coma, hypotonia), and multiple organ involvement (e.g., kidney, lungs, liver, heart, intestines). (4)

Birth asphyxia causes multiorgan dysfunction. The brain is commonly affected by Perinatal asphyxia because of hypoxic-ischemic encephalopathy but other organs or systems are frequently overlooked which also bear the consequences of hypoxic-ischemic insult. The other commonly affected organ systems in birth asphyxia involve kidneys in about 50% of neonates, cardiovascular system in 25%, pulmonary system in 23%. (5)

Perinatal asphyxia affects thyroid hormone equilibrium by way of depressing the central secretion of the thyroid stimulating hormone levels. Lower thyroid hormone levels have been associated with increase in morbidity and mortality. For the development of central nervous system thyroid hormone plays a pivotal role. (6)

Hypoxia leads to activation of deiodinase type 3(D3) in turn inactivates the peripheral conversion of T4 to T3. In hypoxic ishchemic injury, HIF-1[Hypoxia Inducible Factor 1] reduces local thyroid hormone signalling through induction of D3. Firstly, hypoxia impairs cellular metabolism and energy production, leading to oxidative stress and damage to thyroid follicular cells responsible for producing thyroid hormones. This damage can result in decreased synthesis and secretion of thyroid hormones. Secondly, the stress response triggered by hypoxia induces the release of stress hormones, such as cortisol, which can inhibit the hypothalamic-pituitary- thyroid (HPT) axis. This can suppress the release of thyrotropin-releasing hormone (TRH) and thyroid-stimulating hormone (TSH), reducing thyroid hormone production. Finally, hypoxia can disrupt the regulation of iodine uptake by the thyroid gland, leading to decreased hormone synthesis. There is a significant negative correlation between the severity of asphyxia to the level of thyroid hormone at 24- 36 hours after birth hence, the present study was undertaken to compare thyroid hormone profiles (T3, T4, and TSH) at <24 hours and at 72 hours of life in asphyxiated and non-asphyxiated term neonates (7)

MATERIALS AND METHODS

A hospital-based case-control study was conducted over one year in the NICU of the Department of Paediatrics, RNT Medical College, Udaipur, after obtaining ethical clearance. The study's sample size was calculated based on a previous study Prabhakar N et al (8). A total of 150 term neonates were enrolled: 75 cases with perinatal asphyxia and 75 controls who were healthy term neonates. Enrolment followed a consecutive sampling approach based on eligibility at the time of admission/birth. Cases were defined as term newborns requiring resuscitation or with a 10-minute APGAR ≤5. Controls were healthy term neonates with APGAR ≥7 at 1 and 5 minutes. Both groups were matched for gestational age, birth weight, sex, and mode of delivery.

Inclusion criteria: Term neonates (≥37 weeks) with APGAR ≤5 at 10 minutes, need for assisted ventilation, umbilical cord pH ≤7.0, base deficit >16 mmol/L, or lactate ≥10 mmol/L.

Exclusion criteria: Preterm infants, congenital anomalies, sepsis, metabolic disorders, or maternal use of thyroid-related drugs.

All NICU admissions and inborn deliveries were screened. Eligibility was assessed by the on-call pediatric resident using predefined criteria. Written informed consent obtained from a parent/legally authorized representative in their preferred language. A detailed history including sociodemographic profiles, maternal details, antenatal course, obstetric and medical complications were noted in predesigned proforma. Neonatal birth history, need and type of resuscitation required were noted and detailed examination was performed at the time of admission to the NICU. Appropriate management was begun as per unit protocol. Neonates were categorised into three groups based on the Sarnat & Sarnat HIE staging (9)

Blood samples for T3, T4, and TSH were collected within 24 hours of birth and repeated at 72 hours. Blood obtained via peripheral venipuncture using aseptic technique. A volume of 1.5–2.0 mL per time point was collected into serum separator tubes; allowed to clot (20–30 min), centrifuged at 3000 rpm for 10 min, serum aliquoted into pre-labelled cryovials, and analysed within 4 hours or stored at 2–8°C (≤24 h). Longer storage (if any) at −20°C with single freeze–thaw cycle. Hormone levels were measured by chemiluminescence immunoassay (CLIA). Primary outcome was measured between-group differences (cases vs. controls) in serum T3, T4, and TSH at <24 h and at 72 h.

Secondary outcomes were measured within-group change in T3/T4/TSH from 24 h to 72 h.

- Association of hormone levels with HIE stage (I/II/III).
- Correlation between resuscitation intensity/duration and hormone levels.
- Proportion with transient hypothyroxinemia or TSH surge abnormalities across groups.

Data on perinatal history, clinical examination, and staging of hypoxic-ischemic encephalopathy (HIE) were recorded. Cases were managed as per standard NICU protocols. Statistical analysis was performed using SPSS v20. Categorical

variables were compared using the Chi-square test, and continuous variables by Student's t-test. A p-value <0.05 was considered significant.

RESULTS

In this study, 75 neonates with birth asphyxia and 75 healthy term neonates were included. The sex distribution was comparable between cases and controls (p=0.161). Gestational age between 37–38 weeks was significantly more common in cases (77.3%) than controls (54.7%), while 39–40 weeks was more frequent among controls (p=0.007). Low birth weight (<2.5 kg) was more frequent in cases (21.3% vs. 6.7%), though the difference was not statistically significant (p=0.094).

Vertex presentation was observed in 92.0% of controls compared to 70.7% of cases, with breech and shoulder presentations significantly higher in cases (p<0.001). Caesarean section was more common among cases (57.3%) than controls (30.7%), while normal vaginal delivery was higher in controls (p<0.001). Prolonged labor was also significantly associated with cases (45.3% vs. 12.0%; p<0.001). Meconium-stained liquor and fetal distress were more frequent in cases, though the overall perinatal events difference was not significant (p=0.605). **(Table 1)**

Table 1: Distribution of Study Participants According to Neonatal and Perinatal Characteristics

Variable	Category	Cases (n=75)	Control (n=75)	P value
Sex of Baby	Male	47 (62.7%)	55 (73.3%)	0.161
	Female	28 (37.3%)	20 (26.7%)	
Gestational Age	37–38 weeks	58 (77.3%)	41 (54.7%)	0.007
	39–40 weeks	13 (17.3%)	30 (40.0%)	
	≥41 weeks	3 (4.0%)	4 (5.3%)	
Birth Weight	< 2.5 kg	16 (21.3%)	19 (25.3%)	0.094
	2.5 – 3.0 kg	49 (65.3%)	37 (49.3%)	
	≥ 3.0 kg	10 (13.3%)	19 (25.4%)	
Presentation of Fetus	Vertex	53 (70.7%)	69 (92.0%)	<0.001
	Breech	13 (17.3%)	7 (9.3%)	
	Shoulder	9 (12.0%)	0 (0.0%)	
Type of Delivery	Normal Vaginal Delivery	32 (42.7%)	52 (69.3%)	<0.001
	LSCS	43 (57.3%)	23 (30.7%)	
Course of Delivery	Uneventful	41 (54.7%)	66 (88.0%)	<0.001
	Prolonged Labor	34 (45.3%)	9 (12.0%)	
Perinatal Events †	Meconium-Stained Liquor	14 (18.7%)	5 (6.7%)	0.605
	Fetal Distress	24 (32.0%)	6 (8.0%)	

The mean thyroid hormone levels of cases and controls within 24 hours of birth are presented in Table 2. The mean T3 level was 2.39 ± 0.54 nmol/L in the case group and 2.31 ± 0.71 nmol/L in controls, with no statistically significant difference (p=0.461). Similarly, mean T4 levels were 116.67 ± 24.18 nmol/L in cases and 114.10 ± 21.59 nmol/L in controls, which was also not significant (p=0.493). The mean TSH level was 6.91 ± 1.46 mIU/L among cases and 6.92 ± 1.53 mIU/L among controls, with no significant difference between the groups (p=0.991). Thus, no significant difference was observed in T3, T4, or TSH levels between asphyxiated and healthy neonates on Day 1 of life **(Table 2).**

Table 2: Mean Thyroid Hormone Levels on Day 1 among Cases and Controls

Hormonal Parameter	Group	Mean ± SD	p-value	Interpretation
T3 (nmol/L)	Case Group	2.39 ± 0.54	0.461	Not Significant
	Control Group	2.31 ± 0.71		
T4 (nmol/L)	Case Group	116.67 ± 24.18	0.493	Not Significant
	Control Group	114.10 ± 21.59		
TSH (mIU/L)	Case Group	6.91 ± 1.46	0.991	Not Significant
	Control Group	6.92 ± 1.35		

On day 3, the mean T3 level was significantly lower in the case group (1.51 ± 0.73 nmol/L) compared to controls (2.19 ± 0.69 nmol/L; p<0.001). Similarly, the mean T4 level was markedly reduced among cases (76.82 ± 25.50 nmol/L) as against controls (117.27 ± 22.70 nmol/L; p<0.001) **(Figure 1).** The mean TSH level was also significantly lower in asphyxiated neonates (5.97 ± 2.81 mIU/L) compared to controls (7.46 ± 2.15 mIU/L; p<0.001) **(Figure 2)**. Thus, by 72 hours of life, neonates with perinatal asphyxia demonstrated a statistically significant reduction in serum T3, T4, and TSH levels when compared with healthy controls, indicating suppression of thyroid function in the asphyxiated group. **(Table 3)**

Table 3: Mean Thyroid Hormone Levels on Day 3 among Cases and Controls

Hormonal Parameter	Group	Mean ± SD	p-value	Interpretation
T3 (nmol/L)	Cases	1.51 ± 0.73	< 0.001 **	Significant
	Controls	2.19 ± 0.69		
T4 (nmol/L)	Cases	76.82 ± 25.50	< 0.001 **	Significant
	Controls	117.27 ± 22.70		
TSH (mIU/L)	Cases	5.97 ± 2.81	< 0.001 **	Significant
	Controls	7.46 ± 2.15		

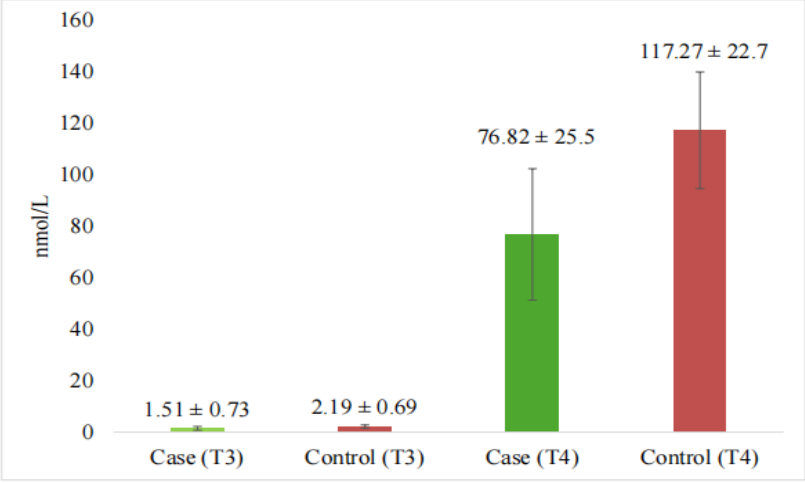

Figure 1: Mean Thyroid Hormone Levels on Day 3 among Cases and Controls

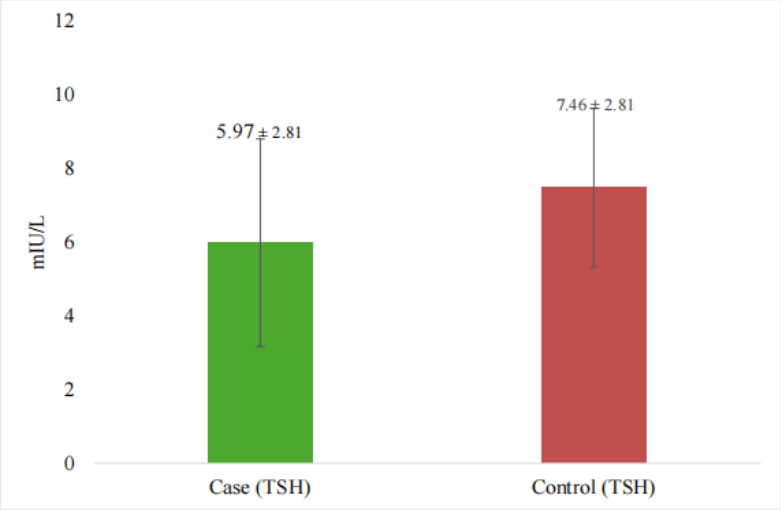

Figure 2: Mean TSH levels on Day 3 among Cases and Controls

On day 1, the mean T3 levels were 1.80 ± 0.65 nmol/L in Stage I, 1.82 ± 0.75 nmol/L in Stage II, and 2.23 ± 0.74 nmol/L in Stage III. Although there appeared to be a trend of higher T3 levels with increasing HIE severity, the difference did not reach statistical significance (p=0.059).The mean T4 levels were 114.61 ± 33.31 nmol/L, 109.96 ± 28.95 nmol/L, and 114.94 ± 33.01 nmol/L in Stages I, II, and III respectively, with no significant difference among groups (p=0.825).Similarly, mean TSH levels were 9.45 ± 2.95 mIU/L in Stage I, 8.32 ± 3.28 mIU/L in Stage II, and 9.37 ± 3.09 mIU/L in Stage III. The difference was not statistically significant (p=0.370). Thus, no significant correlation was observed between thyroid hormone levels (T3, T4, and TSH) and HIE staging on Day 1 of life. **(Table 4)**

Table 4: Correlation of Thyroid Hormone Levels among cases on Day 1 with SARNAT Scoring

Hormonal Parameter (Day 1)	SARNAT Stage			p-value (ANOVA)
	Stage I	Stage II	Stage III	
T3 (nmol/L)	1.80 ± 0.65	1.82 ± 0.75	2.23 ± 0.74	0.059
T4 (nmol/L)	114.61± 33.31	109.96 ± 28.95	114.94 ± 33.01	0.825
TSH (mIU/L)	9.45 ± 2.95	8.32 ± 3.28	9.37 ± 3.09	0.370

On Day 3, thyroid hormones showed a significant decline with increasing HIE severity. Mean T3 decreased from 1.84 ± 0.77 nmol/L (Stage I) to 1.25 ± 0.66 nmol/L (Stage III) (p = 0.017), T4 from 93.17 ± 28.03 to 61.81 ± 10.82 nmol/L (p < 0.001) **(Figure 3)**, and TSH from 7.08 ± 2.82 to 4.83 ± 2.69 mIU/L (p = 0.014) **(Figure 4)**. Post-hoc analysis confirmed significant differences mainly between Stage I and Stage III. Thus, a clear inverse relationship was observed between thyroid hormone levels and HIE severity on Day 3. **(Table 5)**

Table 5: Correlation of Thyroid Hormone Levels on Day 3 among cases with SARNAT staging

Hormonal Parameter	Stage I (n = 22)	Stage II (n = 25)	Stage III (n = 28)	p-value	Post Hoc (Tukey)
T3 (nmol/L)	1.84 ± 0.77	1.52 ± 0.72	1.25 ± 0.66	0.017	Stage I > Stage III *
T4 (nmol/L)	93.17 ± 28.03	79.24±25.93	61.81 ± 10.82	< 0.001 **	Stage I > III, II > III **
TSH (mIU/L)	7.08 ± 2.82	6.28 ± 2.60	4.83 ± 2.69	0.014	Stage I > Stage III *

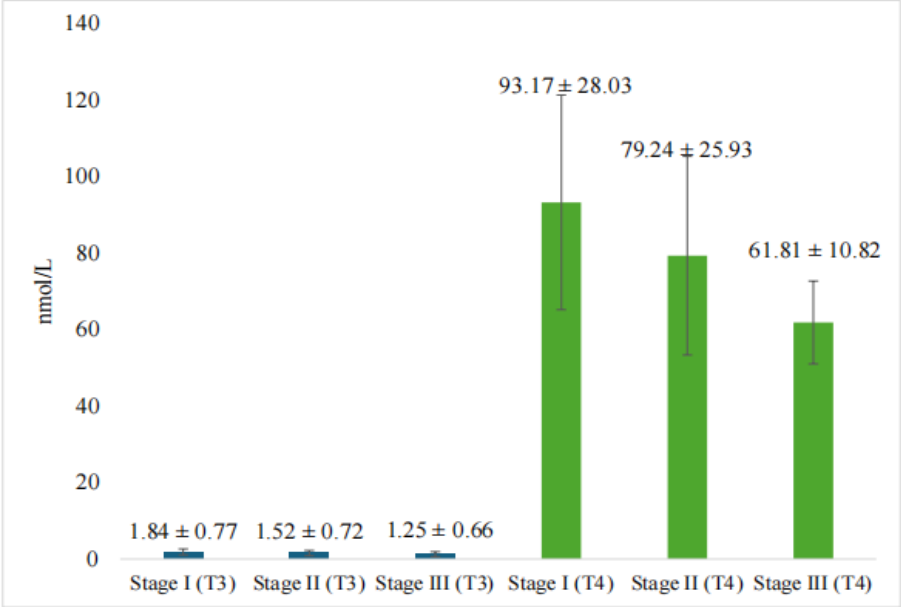

Figure 3: Correlation of Thyroid Hormone Levels on Day 3 among Cases with SARNAT Scoring

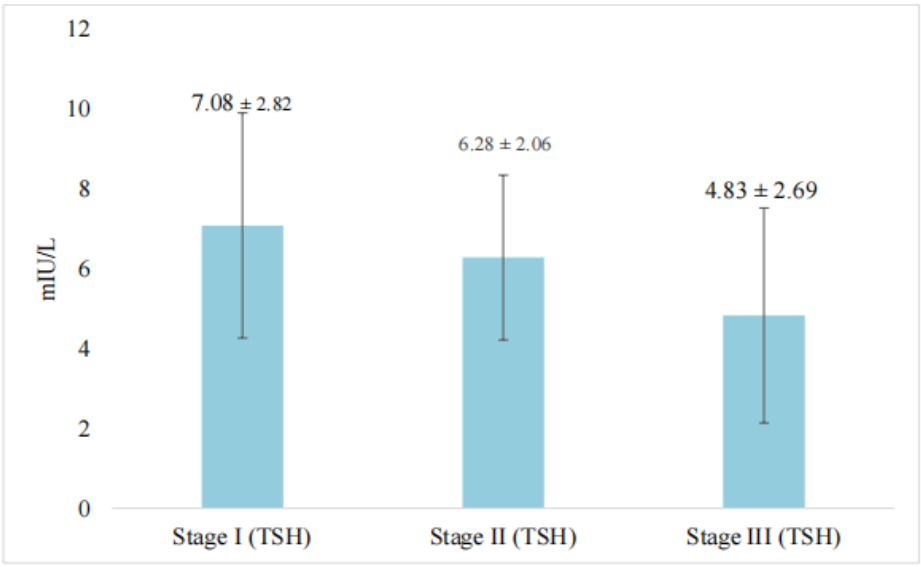

Figure 4: Correlation of TSH Levels on Day 3 among Cases with SARNAT Scoring

DISCUSSION

This hospital based case control study was conducted with 75 neonates as cases and 75 neonates as controls at the Neonatal Intensive Care Unit of a tertiary care centre. The babies were stratified based on the severity of HIE into three stages using Sarnat and Sarnat HIE staging (9). The effect of birth asphyxia was studies on thyroid hormone levels on day 1 and day 3 of life.

In this study, table 1 describes the different variables of study participants showing early-term delivery (37–38 weeks) was significantly associated with perinatal asphyxia, consistent with Lee et al. (10), who reported increased neonatal morbidity in early-term births. Low birth weight was more frequent among cases but not statistically significant, similar to Ilah et al. (11). Non-vertex presentations were markedly higher in cases, echoing Sultana et al. (12), who linked malpresentation to obstructed labor and asphyxia. Cesarean delivery was more common among cases, reflecting emergency interventions, as noted by Dabrowski et al. (13). Prolonged labor and fetal distress were significantly associated with cases, in line with Mwaniki et al. (14). Meconium-stained liquor was more frequent in cases but not significant. These findings emphasize the importance of intrapartum monitoring and timely intervention.

Table 2 shows that within 24 hours, mean serum T3, T4, and TSH levels did not differ significantly between cases and controls (2.39 vs. 2.31 nmol/L, 116.67 vs. 114.10 nmol/L, and 6.91 vs. 6.92 mIU/L, respectively). Thus, perinatal asphyxia did not significantly alter thyroid hormone levels in the immediate postnatal period. Perrone et al. (15) similarly observed that early thyroid values may remain normal due to the postnatal surge, with suppression appearing after 48–72 hours. In contrast, Dilli et al. (16) reported lower early T3 and T4 in severe HIE, suggesting that timing and severity influence results. The absence of significant differences in our study may be due to early sampling before suppression, inclusion of mixed severity, and limited sample size. Kumar et al. (17) stressed the importance of serial hormone assessment.

Table 3 shows that at 72 hours, cases showed significantly lower mean T3, T4, and TSH levels compared to controls ($p < 0.001$ for all). This highlights suppression of the hypothalamic-pituitary-thyroid (HPT) axis and reduced peripheral conversion of T4 to T3 becoming evident after hypoxic insult. Similar findings were reported by Kumar et al. (17) and Dilli et al. (16), who noted that declines appear after 48–72 hours, especially in severe HIE. Mechanisms include reduced TRH/TSH secretion and impaired deiodinase activity, producing a "low T3 syndrome" or transient hypothyroxinemia. Although adaptive, this has been linked to poorer neurological outcomes. These findings underscore the need for serial hormone assessment beyond the immediate newborn period to detect and monitor thyroid dysfunction in perinatal asphyxia.

Table 4 shows on the first day, no significant differences were observed in mean T3, T4, or TSH across Sarnat stages I, II, and III ($p = 0.059$, 0.825, and 0.370, respectively). Although T3 levels appeared slightly higher in stage III, the differences were not significant. This suggests that in the immediate postnatal period, HIE severity does not markedly influence thyroid hormone levels. Perrone et al. (15) explained this by the physiological surge of TSH and thyroid hormones masking early suppressive effects. Dilli et al. (16) also observed minimal early differences, with significant declines emerging only after 48–72 hours in moderate to severe HIE. Our results highlight that single early measurements may not capture the effect of HIE severity on thyroid function.

Table 5 shows that by 72 hours of life , thyroid hormone levels showed a clear relationship with HIE severity. Mean T3 and T4 levels were highest in stage I, intermediate in stage II, and lowest in stage III. Similarly, TSH was significantly reduced in stage III compared to stage I. This stage-wise decline indicates that the severity of hypoxic injury directly influences suppression of the HPT axis and/or peripheral T4-to-T3 conversion. Comparable trends were reported by Dilli et al. (16) and Kumar et al. (17), where severe HIE was associated with pronounced and persistent hypothyroxinemia. Pathophysiology likely involves central suppression from hypothalamic-pituitary ischemia along with reduced deiodinase activity, leading to an adaptive but metabolically limiting low T3 state. These results suggest that monitoring thyroid function is especially important in moderate and severe HIE to identify infants at risk of persistent dysfunction and adverse outcomes.

Limitations: Being a single-center study with limited sample size, generalizability and subgroup analysis are restricted. Only basic thyroid hormones were measured, and long-term outcomes or confounders could not be fully assessed.

CONCLUSION

This study concludes that perinatal asphyxia significantly suppresses thyroid hormone levels in term neonates, with no difference on Day 1 but a marked decline in T3, T4, and TSH by Day 3, especially in moderate to severe HIE. Hormonal suppression correlated with higher Sarnat stage, prolonged hospital stay, greater need for intensive care, and higher mortality, being most pronounced in non-survivors. Low thyroid hormone levels may therefore serve as a prognostic marker. These findings highlight the need for serial thyroid function monitoring and suggest a potential role for hormone supplementation in future trials.

Declarations
Conflict of interest-None
Funding-None
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