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Analysis of Thyroid Hormones in Newborns with Hypoxic Ischaemic Encephalopathy

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Abstract

Introduction: Thyroid hormone plays an essential role in brain development and fetal, neonatal, and adult brain function and may influence neuronal recovery after hypoxia and traumatic brain injury. We aimed to see whether there is a relationship between the effects of hypoxia at the cellular level and the stages of the disease.

Materials and Methods: After ethics committee approval and informing the families, demographic characteristics, laboratory parameters, diffusion MRI images, and TSH and free T4 levels from thyroid function tests on postnatal days 5 and 21 were recorded retrospectively in newborns who were hospitalized with the diagnosis of hypoxic-ischemic encephalopathy in the neonatal intensive care unit of our hospital in the last two years. 81 HIE babies over 37 gestational weeks and 50 control babies of the same age participated in the study. Sarnat staging was performed according to the patient's clinical status.

Results: There were no statistically significant differences between HIE and healthy babies regarding gender, mode of delivery, birth weight, and birth week. According to Sarnat scoring, while healthy infants had lower TSH levels on postnatal day 5 compared to all types of hypoxic infants, moderate HIE newborns had higher TSH measurements compared with healthy, mild, and severe HIE neonates. fT4 level was statistically significantly higher in moderate HIE babies than in healthy, mild, and severe newborns.

Conclusion: We demonstrated that thyroid hormones, known to have significant effects on energy catabolism and central nervous system development, varied over time in HIE infants at various stages.

Key words: Hypoxia-ischemia; newborn; endocrine; thyroid.

Introduction

Perinatal asphyxia involves a disturbance in the exchange of respiratory gases (oxygen and carbon dioxide), leading to reduced oxygen levels (hypoxemia) and elevated carbon dioxide levels (hypercapnia), often accompanied by metabolic acidosis (1). This condition can result in brain injury or damage established as hypoxic-ischemic encephalopathy (HIE), which occurs due to a lack of oxygen in the brain during the perinatal period. Thyroid hormones, which are known to have significant effects on energy catabolism and central nervous system development, showed variability in different periods in HIE infants at different stages (2). In high-income countries, the occurrence is approximately 1.5 per 1,000 live births. However, in low- and middle-income countries, the rate is considerably higher, varying from 10 to 20 cases per 1,000 live births (3,4). According to the Centers for Disease Control and Prevention, in 2023, intrauterine hypoxia and birth asphyixia were the nineth leading cause of infant mortality in the United States, with an incidence of 10.2 per 100,000 live births (5). The fundamental mechanisms resulting in neuronal death following hypoxia-ischemia start with energy loss and the activation of glutamate receptors. This triggers an increase in cytosolic calcium, which in turn causes cellular injury (6).
Therapeutic hypothermia is the established standardised treatment for newborns with hypoxic-ischaemic encephalopathy. This therapy involves cooling the entire body to a core temperature of 33.5°C for 72 hours, beginning within 6 hours of birth (7-9). However, it does not provide comprehensive neuroprotection and is limited in effectiveness (10,11). The endocrine system is essential in managing respiratory, metabolic, and vasomotor reactions to hypoxia (12). Additionally, although not frequently, perinatal asphyxia can be linked to endocrine disorders such as imbalances in electrolytes and glucose, adrenal insufficiency, pineal gland damage, and thyroid hormone irregularities (13).

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Thyroid hormones regulate water and electrolyte balance, control thermogenesis, and promote brain growth and development (14). The link between thyroid function and neurological outcomes in HIE newborns remains poorly understood. Some studies suggest that serum levels of fT3 (free T3) and fT4 (free T4) after the third postnatal day could potentially predetermine brain damage in neonates with asphyxia (15). However, there are only a few researches examining the impact of perinatal asphyxia on thyroid hormones, and their limited findings may be due to differences in research methods (16,17). We aimed to evaluate thyroid function hormones as a potential prognostic indicator of neurological outcomes in neonates with perinatal asphyxia.

Materyal and Methods

Newborns who were hospitalized in our neonatal unit with a diagnosis of hypoxic-ischemic encephalopathy (n=81) between 0-28 days in 2022 and 2023 were included in the study. The diagnosis of perinatal hypoxia was determined by the presence of an acute intrapartum/peripartum episode with a pH ≤7.00 or base deficit ≥12 mmol/L in the first hour after birth, or a blood gas from the umbilical cord, an Apgar score <5 at 5 to 10 minutes, or the need for prolonged resuscitation. Neonates with gestational age <37 weeks, congenital anomalies, congenital heart disease, cerebral anomalies, genetic diseases, sepsis, or lack of parental consent were excluded. After ethics committee approval and informing the families, demographic characteristics, laboratory parameters, and diffusion magnetic resonance images (MRI) were recorded retrospectively in newborns diagnosed with hypoxic-ischemic encephalopathy and hospitalized in the neonatal intensive care unit of our hospital. TSH (thyroid stimulating hormone) and fT4 levels on postnatal day 5 were also documented. In the neonatal outpatient clinic, healthy newborns (n=50) whose thyroid hormones were checked on day 5 as the control group. Both the HIE group and the control group in the study included newborns born at our hospital. Before any procedure, written informed consent was obtained from all parents. Sarnat stage was classified as HIE, and the stages I to III represented mild, moderate, and severe HIE, respectively (18). Hypothermia was administered to moderate and severe hypoxic infants with the Cool Star (CSM2020013-GMS Medical, Turkey) device within the first six hours postnatally for 72 hours, followed by a 6-hour rewarming period. A diffusion cerebral MRI was performed, and the results of blood samples for TSH and free T4 were recorded as thyroid function tests. The abnormal traces of amplitude-integrated electroencephalography (aEEG; Natus-Olympic Brainz Monitor) were recorded.

Ethical approval: The ethics committee approval was obtained by the Yuzuncu Yil University Hospital Clinical Research Ethics Committee with approval number 06-12-2023-09. all procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/ or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Statistical analysis: Descriptive statistics, including the mean and standard deviations of thyroid-related hormones such as TSH and free T4 measurements, were calculated for healthy and HIE newborns. The normal distribution of variables was assessed visually (using histograms and probability plots) and analytically (via the Shapiro-Wilk test).

Table 1: Demographic features of the HIE and control groups

Variables	HIE (n=81)	Control (n=50)	CI	p-value
Male*	46 (56.8%)	24 (48%)		0.32
Type of delivery (vaginally)*	56 (64.4%)	31 (62%)		0.401
Gestational age (weeks)**	38.26±3.2	38.90±2.1	-1.01, 0.49	0.50
Birth weight (g)**	3010.2±450.1	3027.6±441.6	-94.9, 223.1	0.42
Maternal age (years)**	30.29±6.8	29.40±8.1	-1.7, 3.5	0.49
APGAR score 1st minute***	3 (1)	8 (2)	-4.1, -2.9	0.001
APGAR score 5th minute***	6 (2)	9 (2)	-4.01, -3.07	0.001
APGAR score 10th minute***	6 (2)	9 (1)	-3.2, -2.3	0.001
Mortality (%)*	5(6.17%)	-		0.001

*****percent, Chi-square ******mean±stderr *******median (interquartile range) Independent T-test,

Categorical variables are presented as numbers and percentages. The independent t-test and ANOVA were used to compare continuous variables, with Tukey post hoc tests applied. The chi-square test, along with ratio comparisons where necessary, was used to determine relationships between categorical variables. Results were considered statistically significant at a p-value of 0.05. SPSS package (IBM SPSS Statistics for Windows, version 26.0, Armonk, NY: IBM Corp) was used for the calculations.

Results

The study included 81 HIE and 50 healthy newborns over 37 weeks of gestation. Table 1 displays the demographic features of the patient and control groups. There was no statistical significance between hypoxic babies and healthy newborns in terms of sex, delivery type, birth weight, gestational week, and maternal age. However, the Apgar scores of hypoxic babies were statistically lower at 1, 5 and 10 minutes. Patients were staged using the Sarnat classification according to their clinical presentation (Figure 1). All stage 3 infants had both diffusion MRI findings of restriction in different brain regions and abnormal EEG findings. In analyzing hypoxia-related pathological findings on diffusion MRI and EEG findings, no statistically significant correlation was found in thyroid hormones measured on postnatal day 5 in HIE neonates.

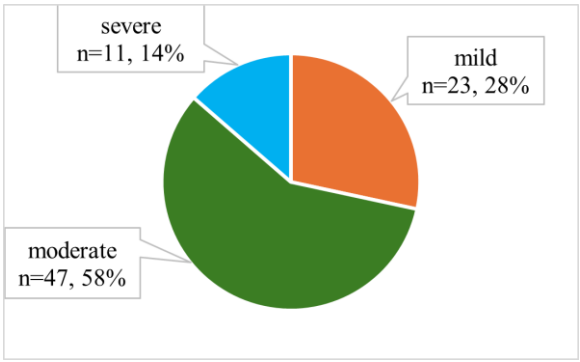

Figure 1: Severity distribution of HIE patients

According to Sarnat scoring, while healthy infants had lower TSH levels on postnatal day 5 compared to all types of hypoxic infants, moderate HIE newborns had higher TSH measurements compared with healthy, mild, and severe HIE neonates (p=0.001). The fT4 measurement was statistically significantly grater in moderate HIE babies than in healthy, mild, and severe newborns (p=0.001) (Table 2, Figure2). The overall model predicting TSH levels was not statistically significant ($F(5,75)=1.823$, $p=0.119$), indicating that sex, Sarnat stage, and their interaction together did not explain a significant proportion of variance in TSH levels ($R^2=0.108$, adjusted $R^2=0.049$). Neither sex ($F(1,75)=0.001$, $p=0.978$), Sarnat stage ($F(2,75)=1.464$, $p=0.238$), nor their interaction ($F(2,75)=1.974$, $p=0.146$) showed significant individual effects on TSH concentrations. In contrast, the model predictin.

Table 2: Postnatal fifth-day TSH and fT4 hormone measurements

Hormones	Healthy (n=50)	HIE severity			η^2	p-value
		Mild (n=23)	Moderate (n=47)	Severe (n=11)		
TSH	2.57±1.05a	6.53±5.6bc	8.88±6.9c	5.64±4.9b	0.91	0.001
fT4	1.08±0.09a	1.22±0.17ab	1.63±0.29c	1.27±0.55b	0.76	0.001

ANOVA Tukey posthoc analysis, groups labeled with different letters are significantly different from each other ($p < 0.05$), while those sharing the same letter are statistically similar.

free T4 levels was statistically significant ($F(5,75)=6.464$, $p<0.001$), explaining approximately 30.1% of the variance in st4 levels ($R^2=0.301$, adjusted $R^2=0.255$). The main effect of Sarnat stage was significant ($F(2,75)=14.598$, $p<0.001$), indicating that ft4 levels differed significantly across severity stages of hypoxic-ischemic encephalopathy. Neither sex ($F(1,75)=0.001$, $p=0.997$) nor the interaction between sex and Sarnat stage ($F(2,75)=0.574$, $p=0.565$) were significant predictors of st4 levels. Only moderate and severe patients received hypothermia treatment, while none of the mild infants were treated with hypothermia. Multiple regression indicated that hypothermia significantly increased fT4 levels ($B=0.35$, $\beta=0.43$, $t=4.17$, $p<0.001$). Sex ($p=0.549$) and the hypothermia × sex interaction ($p=0.939$) were not significant. In contrast, TSH showed a non-significant increase under hypothermia ($B=1.93$, $t=1.22$, $p=0.225$), with no evidence that sex influences this effect. The interaction between sex and Sarnat stage was not statistically significant ($F(6,148)=0.996$, $p=0.430$), suggesting that the effect of HIE severity on the dependent variables does not differ significantly between males and females.

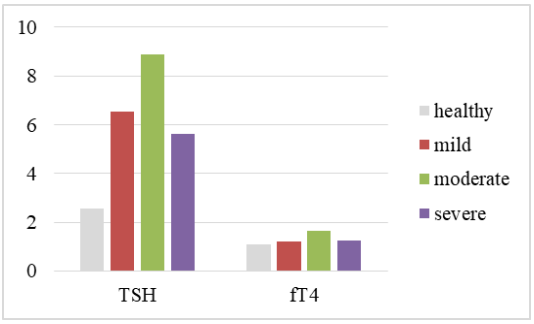

Figure 2: Postnatal TSH and fT4 hormone measurements

The results showed no significant effect of seizure presence on free T4 levels ($F(1,56)=0.006$, $p=0.941$), indicating that free T4 concentrations were similar regardless of seizure status. Likewise, TSH levels were not significantly different between groups with or without seizures ($F(1,56)=0.347$, $p=0.558$). These findings suggest that the occurrence of seizures detected by aEEG does not significantly influence thyroid hormone levels in the studied neonatal population.

Table 3: Postnatal 21th day TSH and fT4 hormone second measurements (n=40)

Hormones	HIE severity			p-value
	Mild (n=12)	Moderate (n=18)	Severe (n=10)	
Second TSH	1.92±0.42a	3.22±3.01a	1.50±2.02a	0.116
Second fT4	1.30±0.13a	1.28±0.22a	1.23±0.61a	0.952

ANOVA Tukey posthoc analysis, groups labeled with different letters are significantly different from each other ($p < 0.05$), while those sharing the same letter are statistically similar

For TSH, the regression coefficient was not statistically significant (B= –0.002, p=0.827), and the 95% confidence interval (–0.019 to 0.015) included zero, indicating no reliable association. Similarly, fT4 was not a significant predictor of MRI findings (B= –0.023, p=0.877), with its confidence interval (–0.323 to 0.276) also encompassing zero. In both models, the standardized coefficients were close to zero (TSH β=–0.025; fT4 β=–0.018), suggesting negligible effect sizes. Among the babies who showed abnormal hormone results in the day-5 tests, when their tests were evaulated on postnatal day 21, no statistically significant differences were observed between the stages (Table 3).

Discussion

Perinatal asphyxia is a major contributor to neonatal mortality and morbidity, impacting nearly all organs and systems due to oxygen deprivation at the tissue level (1). Several studies have explored the effects of perinatal asphyxia on thyroid function (13,14,16,14,19). However, the mechanisms underlying the thyroid hormone response to asphyxia in neonates remain unclear.
This condition is commonly associated with non-thyroidal illness syndrome, which is characterized by low T3 and T4 levels, elevated reverse T3, and low-normal or suppressed TSH concentrations. These alterations reflect an adaptive response to hypoxia and systemic stress, rather than intrinsic thyroid dysfunction. The severity of non-thyroidal illness syndrome correlates with the extent of hypoxic-ischemic encephalopathy (HIE) and may be linked to poorer neurological outcomes (13). The underlying mechanisms include dysregulation of the hypothalamic-pituitary-thyroid axis, altered deiodinase activity, and impaired thyroid hormone uptake at the tissue level (13). Inadequate oxygenation of the hypothalamus and/or pituitary, along with elevated cytokine levels in the context of asphyxia, may disrupt the release of hypothalamic and pituitary hormones such as Thyroid regulating hormone and TSH (20). Therapeutic hypothermia, a standard treatment for moderate to severe HIE, may further influence thyroid hormone dynamics and occasionally produce patterns resembling central hypothyroidism. Although non-thyroidal illness syndrome typically resolves spontaneously as the acute phase subsides, thyroid function monitoring is recommended—particularly in neonates undergoing therapeutic hypothermia. Thyroid hormone replacement is usually not required unless persistent primary or central hypothyroidism is demonstrated (13). Almost two decades ago, cord blood TSH contents were significantly rised in the low Apgar score newborns group compared to the controls, and cord blood T4 and fT4 measurements were significantly decreased. At the same time, there was no difference in TBG, T3, and rT3 levels between the groups (21). In a study involving a small sample size, no significant differences were determined in the mean cord blood measurements of TSH, T3, T4, and fT4 between the asphyxiated

and controls. However, in arterial blood samples taken 18 to 24 hours following birth, the asphyxiated group showed significantly lower mean contentss of TSH, T3, T4, and fT4 compared to the controls. Neonates with moderate to severe HIE had substantially reduced mean levels of TSH, T3, T4, and fT4. Mortality was found to correlate with fT4 levels; none of the asphyxiated babies with fT4 ≤ 2.0 ng/dl survived, while all those with fT4 > 2.0 ng/dl survived. The study also examined the connection between the HIE severity, altered thyroid hormone measurements, and the relationship between mortality and fT4 contents in newborns (16). Thyroid binding globulin, TSH, fT4, T4, T3, and rT3 levels were measured in cord blood and hypoxic infants. In infants with asphyxia born through emergency cesarean section, the findings reveal the occurrence of temporary hypothyroxinemia at birth. The clinical significance of these datas was that in some infants with perinatal asphyxia and related central nervous system injury, thyroid hormone tests may be suggestive of hypopituitary hypothyroidism. Also, in those newborn screening plans that report infants with low T4-normal TSH, such neonates will induce false-positive events (14). Thyroid function was declared to be lower post-birth in HIE babies, particularly in those with moderate or severe HIE (15,16). In terms of seizures, it was found that none of those with high fT4 (>1.84 ng/dL) had seizures (19). In a retrospective study of babies born over 34 gestational weeks diagnosed with HIE, when capillary TSH, serum TSH, and free T4 levels of babies who received and did not receive therapeutic hypothermia were examined, they showed that hypothermia may modulate thyroid hormones. Researchers also found a higher capillary TSH in the first 4 days of life (17). Recently, researchers noted that fT4 levels were negatively correlated with length of hospitalization, duration of intubation, duration of ventilatory support, and time to full oral feeding. They also reported high fT4 concentrations in newborns with HIE treated with hypothermia, which might be preventive in terms of neurological difficulties (19). In our study, since moderate and severe infants received therapeutic hypothermia while none of the mild cases underwent hypothermia, an interaction effect on free T4 levels was observed in infants treated with hypothermia, independent of sex. However, as there were no pre-hypothermia or cord blood thyroid hormone measurements available, it was not possible to evaluate the direct impact of hypothermia on hormone levels. A recent study investigated transient tachypnea of the newborn (TTN) and thyroid hormone levels in hypoxic neonates at different stages on postnatal day five. The results showed that serum TSH and fT4 levels were lower in neonates with stage 3 HIE compared to those with stage 2 HIE. Although serum TSH concentrations were higher in neonates with HIE than in those with TTN, there were no significant differences in serum fT4 levels between the cohorts. When compared to reference values, hypothermic HIE was associated with a greater increase in TSH levels compared to TTN, with this difference being more prominent in stage 2 hypothermic HIE. Stage 3 hypothermic HIE was linked to higher frequencies of low fT4 levels than TTN, although this was not the case for stage 2 hypothermic HIE (22).

Study limitations: This study has limitations, including its retrospective analysis, small sample size, lack of T3/fT3 measurements, and potential effects of inotropic agents on thyroid function in severe HIE. Coexisting Euthyroid Sick Syndrome may alter thyroid hormones, particularly by lowering T3. Additionally, elevated cortisol as a stress response may influence TSH and T4 via hypothalamus–pituitary–thyroid axis modulation. The imbalance in group sizes especially in small "severe HI" subgroup can also affect the precision of estimated effects and the robustness of variance assumptions in group comparisons. These factors were not assessed but are noted as limitations and suggested for future research.

Conclusion

In our study, we found that hypoxia may affect thyroid hormone levels in newborn infants at different stages of hypoxia in the first days of life and show variability in the following days. Future prospective multicenter studies with large data sets are required to definitively establish the role of thyroid hormone levels as prognostic biomarkers in hypoxic-ischemic encephalopathy.

Ethical approval: The ethics committee approval was obtained by the Yuzuncu Yil University Hospital Clinical Research Ethics Committee with approval number 06-12-2023-09. all procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/ or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Author contributions: Conception or design of the work (NA, EY), Data collection (NA, MB), Data analysis and interpretation (SK, OT), Drafting the article (NA, EY, DCD), Critical revision of the article (OT, NA), Study supervision, fundings (OT, DCD)

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