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STUDY OF IMPACT OF BIRTH ASPHYXIA ON THYROID HORMONE IN NEWBORN.

Sonali Pradhan[1], Jatadhari Mahar[2], Gobinda Hembram[2], Pravakar Mishra[3*]
[1]Senior Resident, Department of Pediatrics, SVP PGIP, SCB Medical College, Cuttack, Odisha, India
[2]Assistant Professor, Department of Pediatrics, SVP PGIP, SCB Medical College, Cuttack, Odisha, India
[3]Professor, Department of Pediatrics, SVP PGIP, SCB Medical College, Cuttack, Odisha, India

ABSTRACT.

Aim and Objective: Study of the Impact of birth asphyxia on thyroid hormone in newborns.

Methods: It was a prospective case-control study conducted at SCB Medical College, Department of Pediatrics. For the study, a total of 200 full-term newborns were assigned of which 100 asphyxiated newborns were taken as cases and 100 healthy newborns as a control group. Sarnat and Sarnat staging is used to classify the severity of birth asphyxia. Blood samples were collected at 18 to 24 hours of age for thyroid hormone level estimation.

Results: Out of 100 cases, 33% were HIE stage 1, 43% were HIE stage 2, and 24% were HIE stage 3. The mean value of T3, T4, and TSH was lower at 18 to 24 hours of age.

Conclusion: The mean T3, T4, and TSH level at 18 to 24 hours of asphyxiated newborn was significantly lower than the control group.

Recommendation: More studies need to be done with a larger sample size in different regions of India and investigate the prevalence of thyroid hypoxic-ischemic encephalopathy and its association with morbidity.

Keywords: *Birth asphyxia, Newborn, Thyroid function, Hypoxic ischemic encephalopathy.*
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Corresponding author: *Pravakar Mishra**
Email:*drpmishra61@gmail.com*
Professor, Department of Pediatrics, SVP PGIP, SCB Medical College, Cuttack, Odisha, India.

INTRODUCTION.

Asphyxia is a term used to indicate the consequences of a complete lack of oxygen as a result of several primary causes. Hypoxia refers to the decreased arterial concentration of oxygen. Ischemia refers to blood flow to cells or organs that are insufficient to maintain the normal function of the organs. Perinatal asphyxia provokes multiple alterations in the body due to failures in the gas exchange system.

Among these alterations hypoxia, hypercapnia, and decrease of blood pH, thus causing redistribution of the blood flow from lesser vital organs to more vital organs such as the brain, heart, and adrenal glands. [1]

Thyroid function is in a state of flux during the perinatal period.[2,3] Previous studies have documented thyroid hormone, thyroxin-binding globulin, and thyroid-stimulating hormone concentrations in term and preterm infants at birth, over the neonatal period, and during early infancy. However, very little data is available that attempts to evaluate the possible effect of perinatal asphyxia on neonatal thyroid function. [4, 5]

Byfield PG et al studied the concentrations of reverse triiodothyronine, thyroid hormone, and thyrotrophin concentrations in placental cord blood. Reverse triiodothyronine (rT3), triiodothyronine (T3), thyroxine (T4), thyroxine-binding globulin (TBG), and thyrotrophin (TSH) were measured in sera from placental cord blood in an unselected series of 272 deliveries. In this series, the concentrations of rT3 (mean 3.33 nmol/l, 95% confidence limits 1.6--- 7.0 nmol/l), were log-normally distributed and did not overlap the adult normal range (0.11--0.44 nmol/l). There were no correlations between the cord blood concentrations of rT3, T3, T4, and TSH. The cord serum rT3 concentration was not influenced by maturity, birth weight, or neonatal risk factors, whereas these factors did affect the concentrations of T3, T4, and TBG. There is no arteriovenous rT3 concentration difference across the placenta, therefore the cord rT3 reflects the systemic rT3 concentration in the baby at birth. As rT3 in the neonate largely, if not entirely, derives from thyroxine from the fetal

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thyroid, measurement of the cord rT3 concentration may be a good immediate screening test for neonatal hypothyroidism.

Few studies have shown a difference between Serum concentrations of TSH, T4, T3, and FT4 in asphyxiated newborns than in normal newborns which suggests central hypothyroidism secondary to asphyxia. Moreover, Asphyxiated newborns with moderate/severe hypoxic-ischemic encephalopathy present a greater involvement of the thyroid function and consequently a greater risk of death. This prospective institutional study compares serum concentrations of thyroid hormones-Total T4, Total T3, and Thyroid-stimulating hormones from venous blood of term newborns with asphyxia and healthy newborns collected between 18 and 24 hours after birth. Further, it aims to study and assess the association between the severity of hypoxic-ischemic encephalopathy and altered thyroid hormone and TSH levels.

OBJECTIVE.

Study of Impact of birth asphyxia on thyroid hormone in newborn.

MATERIALS & METHODS.

Study design.

This is a prospective case-controlled study conducted at SCB Medical College, Cuttack. Birth asphyxia newborns admitted to NICU and SNCU were taken as case and control groups from the postnatal ward of the O & G Department.

Inclusion criteria.

- **Asphyxiated Newborn**
  a) 1 and 5-minute APGAR Score < 7
  b) Term (Gestational age >37 weeks)
  c) Weight >2.5 kilograms
- **Non asphyxiated Newborn**
  a) 1 and 5-minute APGAR score greater than or equal to 8
  b) Term (Gestational age > 37 weeks)
  c) Weight > 2.5 kilograms

Exclusion criteria.

- Maternal history of thyroid dysfunction.
- Prematurity
- Maternal history of antihypertensive and steroid intake.
- Major Congenital defects and metabolic illnesses
- Neonatal Sepsis

Study setting and population.

This prospective case-control study was conducted at SCB MCH, Cuttack, a tertiary care hospital between December 2020 to November 2022. A total sample size of 200 newborns were enrolled out of which 100 were asphyxiated newborns taken from SNCU and NICU and 100 healthy newborn as control allotted from O & G Department.

Gestational age was determined by using obstetrical gestational age and ascertained by physical examination by using Modified Ballard's Scoring. When the difference between obstetrical age and clinical evaluation was higher than 2 weeks, clinical evaluation was considered. A thorough maternal history was obtained including information on maternal morbidities such as hypertension, thyroid problems, and drug intake. Perinatal events including the use of sedatives, CTG findings, prolonged second stage of labor, the color of the liquor, the Apgar score, and birth weight are noted. Asphyxiated newborns were thoroughly examined for physical and neurologically and other complications. The severity of birth asphyxia was graded by Sarnat & Sarnat staging as mild (stage 1), moderate (stage 2), and severe (stage 3). In the first 72 hours after birth, asphyxiated newborns are monitored for signs of multi-organ dysfunctions such as cardiogenic shock, renal failure, respiratory distress, and seizure.

Peripheral venous samples for T3, T4, and TSH are collected from both groups at 18-24 hours of life and analyzed by ELISA method at Biochemistry Lab, SCB MCH, Cuttack

The value of thyroid hormone between asphyxiated and healthy newborns is compared and the relationship between the severity of Hypoxic ischemic encephalopathy and thyroid hormone level is analyzed.

The required sample size was calculated by using the following formula:

Sample size $n = 2\,\sigma 2\,[Z_{1-\alpha} + Z_{1-\beta}]^2 / (\mu 1 - \mu 2)^2$

The minimum sample size in each group was calculated to be 100.

All the data were entered in XL format and exported to SPSS 25 Version for data analysis.

Ethical considerations.

The study was approved by the Institutional Ethics Committee of SCB MCH and written informed consent was obtained from parents. The ethical aspects of the research were carefully thought out to preserve patient privacy and confidentiality. An institutional research committee ethics clearance letter was obtained before patient data was accessed

Bias.

There was a chance that bias would arise when the study first started, but we avoided it by giving all participants identical

information and hiding the group allocation from the nurses who collected the data.

Data collection.

The sample size was determined with a significance level of 0.05. The means, medians, and standard deviations of continuous variables were used to characterize whereas the percentage of data from the sample collected for the study was used to describe categorical variables.

Statistical analysis.

The continuous or quantitative variables were expressed as mean + - SD and categorical variables as frequency percentages. Chi chi-square test was used to find out the association of the categorical variable with the outcome variable. Similarly, an independent sample t-test was employed to find out the association of continuous variable outcomes as well as other categorical variables. The p-value was set < 0.05 for statically significant. The data were analyzed by using the statistical software SPSS Version 25.

RESULTS.

A total sample size of 200 newborns was enrolled out of which 100 were asphyxiated newborns taken from SNCU and NICU and 100 healthy newborns as control allotted from the O & G Department. Out of 100 asphyxiated newborns, 55(55%) were male and 45(45%) were female and in healthy newborn 52(52%) were male and 48(48%) were female out of 100 newborn. The average Apgar score at 1 min is 6 and at 5 min is 9.29 cases and 15 control were born through assisted vaginal delivery, and 84 babies were born by LSCS (33 cases and 51 control).Total of 72 born delivered through normal vaginal delivery. Only 28 (28%) asphyxiated neonates had stained liquor and this is statistically significant ($p<0.05$). The maximum number of neonates born from primipara Mothers in both asphyxiated and non-asphyxiated groups are 56% and 58% respectively. Distribution of HIE staging among asphyxiated newborns, where 33 (33%) had Mild HIE, 43 (43%) had Moderate HIE, and 24 (24%) had severe HIE. In the study, the maximum numbers belong to HIE 2.

Table 1: Associated morbidity in Asphyxiated newborn.

Respiratory distress	Yes	72
	No	28
Cardiac dysfunction	Yes	23
	No	77
Renal dysfunction	Yes	33
	No	67

Out of 100 cases, 72 (72%) neonates had respiratory distress, 23 (23%) had cardiac dysfunction and 33 had renal dysfunction. This shows that respiratory distress was found to be more associated with asphyxiated neonates.

Table 2: APGAR at 1 minute and 5 minutes in both Asphyxiated and Non-asphyxiated newborn.

		Asphyxiated	*Non-asphyxiated*	*p-value*
APGAR score at 1 min	<3	45 (45%)	0 (0%)	0.047
	(3-6)	55 (55%)	5 (100%)	
APGAR score at 5 min	<3	0 (0%)	0 (0%)	<0.001
	(3-6)	26 (26%)	0 (0%)	
	>6	74 (74%)	100 (100%)	

APGAR score at 1 minute is < 3 in 45 asphyxiated newborns (45%), and 3-6 in 55 newborns (55%).In this study, APGAR at 1 min was not found statistically significant ($p<0.05$) in both groups and only 5 among the non-asphyxiated group had Apgar score between 3-6.

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Table 3: Serum levels of Thyroid profile in both Asphyxiated and Non-asphyxiated newborns within 18-24 hrs. of life.

	Case	*Control*	*P-value*
T3(ng/dl)	84.62±21.17	108.80±21.10	<0.001
T4(mcg/dl)	8.80±2.79	11.89±2.48	<0.001
TSH(mIU/L)	2.49±3.17	7.95±4.64	<0.001

The mean T3, T4, and TSH values are lower in cases as compared to control.

Table 4: Distribution of Serum Thyroid hormone levels in Asphyxiated neonates in different stages of HIE within 18-24 hrs. of life.

	HIE 1(n=33)	*HIE 2(n=43)*	*HIE 3(n=24)*	*P value*
T3(ng/dl)	101.83±15.62	82.09±17.65	65.51±14.22	<0.001
T4(mcg/dl)	11.57±2.31	8.35±1.51	5.81±1.15	<0.001
TSH (mIU/L)	5.17±3.91	1.54±1.81	0.53±0.17	<0.001

On comparison of Mean values of T3, T4, and TSH in different stages of HIE were statistically significant (p<0.05).

Table 5: Thyroid profile correlation with associated morbidity.

	Respiratory distress n=72	*Cardiac dysfunction n=23*	*Renal dysfunction n=33*	*Seizure n=67*
T3(ng/dl)	81±21.07 (0.006)	71.02±20.41 (<0.001)	74.89±16.75 (0.001)	76.15±18.25 (<0.001)
T4(mcg/dl)	7.98±2.30 (<0.001)	6.24±1.31 (<0.001)	7.2±1.86 (<0.001)	7.44±1.85 (<0.001)
TSH(mIU/L)	2.21±3.22 (0.153)	0.69±0.66 (0.002)	1.42±2.32 (0.017)	1.18±1.52 (<0.001)

In comparison, thyroid profile levels were statistically significant (p<0.05) in asphyxiated newborns associated with seizures. Mean T4 and T3 value was statistically significant (p<0.05) in newborn associated with respiratory distress, whereas mean values of TSH were not statistically significant (p>0.05). The mean values of T3, T4, and TSH were statistically significant in newborns associated with cardiac dysfunction and renal dysfunction.

DISCUSSION.

In this investigational study, we evaluated the thyroid hormones and TSH levels in the blood (between 18 and 24 hours after delivery) of asphyxiated and normal-term newborns to determine the influence of asphyxia on this hormone concentration. The paired case-control design used in our investigation decreased the potential of confounding biases by neutralizing the impact of many covariates including sex, gestational age, weight, and the mode of delivery on hormone levels. Therefore, the current study has attempted to examine the impact of birth asphyxia on a newborn's thyroid hormone levels and to determine the relationship between those levels and the severity as well as morbidity related to asphyxia. [8]

In the current research, asphyxiated newborns had a mean average weight of 2.80 kg compared to 2.84 kg in the control group. According to research conducted by Borges M. et al.; Tahivoric HF. and Wilson DM. et al the mean average birth weight was found to be 3.3 kg and 3.6 kg respectively. [9, 10, 11] This difference between our research findings and the findings of other studies about the mean average birth weight may be related to ethnic and geographic heterogeneity. [6, 8]

Our study has also found a positive correlation between the elevation of asphyxia and the mode of delivery of newborn (P = 0.014) and Meconium stained liquor (MSL) (P <0.001). Of the total distribution of cases in both groups, it was observed that the level of asphyxia is elevated in the normal and assisted form of vaginal delivery than among those who had been delivered using the Lower segment cesarean section (LSCS) method. Our findings are in concordance with the findings of Lee SY. where the author examined a positive level of correlation between TSH and asphyxiated newborns in post-vacuum-assisted vaginal delivery. [12] However, few studies have observed statistical differences

in the modes of delivery and the emergence of birth asphyxia among term neonates. [13, 14]

The clinical diagnosis of asphyxia is based on multiple criteria of which, the two most important criteria are based on the evidence of cardiorespiratory and neurological depression which can be defined as an Apgar score of less than 7 at 5 minutes after birth and the evidence of an acute hypoxic compromise with acidemia i.e., an arterial blood pH of less than 7 or a base excess of greater than 12 mmol/L. [15] In certain circumstances, prenatal or neonatal acidemia may be difficult to assess especially in countries with low resources. [16] On the other hand, while resuscitation is being administered during the immediate postpartum period, it may be difficult to determine whether the neurological and cardiorespiratory depression is due to hypoxia, ischemia, or other conditions such as a fetal-maternal infection or any other metabolic condition. [15, 17] In such a case, the APGAR score is taken into consideration for its earliest management.

The WHO defines intramural infant birth asphyxia as an Apgar score of less than 7 at 1 minute of birth. Moderate birth asphyxia is characterized by delayed gasps breathing at 1 minute of birth for extramural infants. On the contrary, severe birth asphyxia is characterized by absent breathing at 1 minute of birth. [18] Although it is noted that the incidence of birth asphyxia remains nearly high i.e., 2 – 20/ 1000 live births, especially among poor nations. It has also been studied that 15 to 20% of prenatal asphyxia patients die, while 25% suffer from serious neurological complications. As studied, several substances influence thyroid hormone concentrations by acting on several metabolic stages. Little research has been conducted on thyroid hormones and birth asphyxia so far. [19] It is clinically examined that hypoxia disrupts thyroid function and decreases T4 metabolism. However, numerous investigations on thyroid hormones and birth asphyxia have shown contradictory findings. Additionally, according to several studies, IUGR fetuses had shown considerably reduced levels of free T3 and free T4 and elevated levels of TSH.[20] Studies have also indicated that TT4, TT3, and FT3 readings were significantly lower in the smaller gestational age groups, however, there was no significant difference was observed for FT4 and TSH respectively. [21, 22] However, our study has found a significant correlation between the lower APGAR score and elevation of birth asphyxia among early newborns respectively. To address this, bag and mask ventilation or intubation for those having an APGAR score less than 7 is highly recommended for managing such circumstances clinically.

Another classification of the asphyxiated newborn was examined based on the severity of hypoxic-ischemic encephalopathy. Those cases were staged in three levels i.e., mild, moderate, and severe category. Our study found a statistical significance in T3, T4, and TSH levels and the presence of asphyxia among term neonates (p value<0.001). All three mean average values were found to be significantly lower among asphyxiated term newborns than among healthy term newborns. This finding is in line with the study conducted by Tikkas R. et al. [8] However, studies conducted by Kim EY. et al. and Borges M. et al. found a contradictory picture of TSH levels in asphyxiated neonates. [9, 23]

The study found that the mean plasma concentration of the thyroid hormone levels within 18-24 hrs. of life in asphyxiated newborns was found to be in much lower concentration than among the non-asphyxiated neonates with a (P<0.001). Our findings are similar to the findings of a study conducted by Umesh G. et al. and Pereira DN. et al. [24, 25] however, another study by Borges M. et al. found the enrolled neonates, despite a surge in the TSH levels at 5 and 3 hours of birth, T3 and T4 failed to elevate in the asphyxiated group.

In adults, older children, and experimental animals with chronic sickness, hypoxia, or fasting, the inability of TSH to increase while blood levels of thyroid hormone are declining has been observed in a few studies earlier. Although a reduced TSH response to TRH has been documented in adults with fasting or chronic hypoxia. The cases were observed to have a postnatal TSH rise equal to the control group. [9] The reason could be a plausibility to hypothesize that the absence of growth in thyroid function among the neonates in the cases group may have contributed to a decrease in the oxygen consumption and metabolic rate among infants. [26] Another reason could be as a result of alteration in the amounts of binding proteins in the blood which are not investigated in clinical settings. However, some have addressed its improbability since it would not affect the hormone-free fraction in circulation. [10, 27] Similarly, a prospective study conducted by Seth A. et al. had a similar finding which had higher TSH levels among asphyxiated individuals. [28] Another study has provided a possible cause for the lowering of T3, T4, and TSH in neonates with Birth asphyxia because of non-thyroidal illness syndrome. It is a kind of hypothyroidism characterized by both central and regional components which is frequently accompanied by other hormonal deficiencies. Which is known as euthyroid sick syndrome. [19] It is this condition which is defined by a reduction in T3 and T4 without an increase in TSH. It happens in cases of protein energy deficiency or following major operations, infections, and birth asphyxia. Reduced T3 and T4 concentrations are likely a result of decreasing TSH levels. [19, 29] Hypoxia-induced modification of the thyroid hormone pathway is another probable cause. Moshang Jr T. et al. in his study had found elevated rT3 levels among individuals with acute hypoxia. While in chronic hypoxia cases, it was associated with reduced T3 levels and elevated rT3 levels. This indicated that the deterioration of rT3 decreases as the conversion of T4 to rT3 increases. [30]

Moreover, from all the symptoms observed in our study, respiratory distress syndrome (RDS), and seizure followed by cardiac and renal dysfunction were profoundly seen among asphyxiated neonates. Findings from our study suggested a positive correlation between the lowering of T3 and T4 with the increase in RDS. These findings are in discordance with the findings of a study by Kim Y. et al. [31] which suggested the lowering of TSH levels shortly after delivery was related to a higher risk of RDS in their research findings, although T3 and fT4 were not found associated with an increased risk of RDS. Additionally, it also suggests that the underlying etiology of RDS such as the surfactant deficiency remains unaffected by thyroid hormone but was related to pituitary gland suppression at birth. [31] On comparing the other symptoms, it was found that a decrease in either or both of the T3 and T4 levels or an increase in TSH levels had a higher predisposition for morbidity among asphyxiated neonates with a p-value less than 0.05 being statistically significant. However, there was no statistically significant link between higher TSH levels and morbidity related to RDS was observed in our study. These findings were the findings from a study conducted by Rachel Prakantha SG. [32]

CONCLUSION.

Serum concentrations of T3, T4, and TSH are lower in asphyxiated newborns than in healthy newborns. Asphyxiated newborns with moderate hypoxic-ischemic encephalopathy and severe ischemic encephalopathy have a higher involvement of thyroid dysfunction. There is a strong link between substantial morbidity and low T3, T4, and TSH levels. These variations might be a resultant modification in hormone synthesis and peripheral T4 metabolism.

LIMITATIONS.

The study needs more samples and follow-up blood draws at different intervals following delivery to validate our findings and ascertain if these modifications are transient or permanent. It is necessary to investigate the effects of additional potential causes, such as newborn stress and other underlying conditions, on the reduction of thyroid hormones. Further investigation into thyroid hormone supplements may be considered, given that thyroid replacement is a therapy option.

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LIST OF ABBREVIATIONS.

T3- triiodothyronine
T4- thyroxine
TSH- thyroid stimulating hormone
FT4- free thyroxine
HIE- hypoxic-ischemic encephalopathy
TBG- thyroxine-binding globulin
APGAR- appearance, pulse, grimace, activity, and respiration
MSL- meconium-stained liquor
LSCS- lower segment cesarean section
NICU- neonatal intensive care unit
SNCU- special newborn care unit
SCB- Srirama Chandra Bhanja

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CONFLICT OF INTERESTS.

The authors declare no conflict of interest.

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