E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.48047/ijprt/16.01.484

Research Article

Correlation between Maternal Thyroid Biochemistry, Physiological Changes in Pregnancy, and Infant Developmental Milestones

Umair Farukh[1], Ali Sebtain[2], Arsala Rehman[3], Rahat Jan Wazir[4], Zara Fatima Alvi[5], Mohammad Haroon[6]

[1]Assistant Professor, Department of Physiology, Rai Foundation Medical College, Sargodha, Pakistan
[2]Assistant Professor, Department of Emergency Medicine, Lady Reading Hospital, Peshawar, Pakistan
[3]Lecturer, Department of Physiology, Khyber College of Dentistry, Peshawar, Pakistan
[4]Associate Professor, Department of Physiology, Gomal Medical College, Dera Ismail Khan, Pakistan
[5]MBBS Final Year Student, Yichun University, Yichun, China
[6]Associate Professor, Department of Biochemistry, Mohammad College of Medicine, Peshawar, Pakistan

Corresponding Author: Ali Sebtain, Assistant Professor, Department of Emergency Medicine, Lady Reading Hospital, Peshawar, Pakistan, Email: ali_sebtain8955@yahoo.com
Received: 02.02.26, Revised: 26.04.26, Accepted: 10.05.26

ABSTRACT

Background: Maternal thyroid hormones are important in pregnancy adaptation, fetal growth and early neurodevelopment. Mild thyroid dysfunction may have a role in the outcomes of the newborn and the developmental milestones of the infant, especially during periods of fetal brain development.

Objective: To determine the correlation between maternal thyroid biochemistry, physiological changes during pregnancy, and infant developmental milestones.

Methods: This is a prospective observational analytical study carried out at Lady Reading hospital Peshawar during the time period of January 2024 to January 2025. Non-probability consecutive sampling was used to recruit 77 pregnant women. Demographic, obstetric, physiological and biochemical parameters of thyroid function (TSH, free T4, free T3, anti-thyroid peroxidase antibody) were noted. Post- delivery pregnancy outcome and neonatal parameters were recorded. Follow-up assessment was conducted at a later age on the gross motor, fine motor, language, social and cognitive/adaptive developmental milestones. The data were analyzed using SPSS version 25. The following methods were used: chi square test, correlation analysis, and binary logistic regression. A p value of ≤0.05 was deemed to be statistically significant.

Results: The mean maternal age was 28.41 ± 4.92 years. Among 77 participants, 52 (67.5%) were euthyroid, while 25 (32.5%) had thyroid dysfunction. Subclinical hypothyroidism was the most common abnormality, observed in 15 (19.5%) women. Overall developmental delay was found in 17 (22.1%) infants. Developmental delay was significantly higher among infants born to mothers with thyroid dysfunction compared with euthyroid mothers (40.0% vs. 13.5%, p=0.009). Maternal TSH showed a significant negative correlation with infant developmental score (r=-0.346, p=0.002), while Free T4 showed a significant positive correlation (r=0.392, p=0.001). On logistic regression, maternal thyroid dysfunction remained an independent predictor of infant developmental delay (AOR=3.42, 95% CI: 1.12–10.47, p=0.031).

Conclusion: Maternal thyroid dysfunction during pregnancy was significantly associated with delayed infant developmental milestones. Raised TSH and reduced Free T4 were particularly linked with poorer developmental outcomes. Early thyroid screening, timely management, and developmental follow-up of infants born to mothers with thyroid dysfunction may help reduce preventable neurodevelopmental risk.

Keywords: Maternal thyroid dysfunction, pregnancy, TSH, Free T4, infant development, developmental milestones, hypothyroidism.

INTRODUCTION

The physiological and endocrine changes during pregnancy are significant changes, that facilitate fetal growth, the function of the placenta and maternal metabolic adaptation. Thyroid hormone regulation is especially significant

amongst them since thyroid hormone hormones play an important role in fetal growth, maturation of nervous system, and early brain development in the baby. In the first trimester, fetal thyroid requirements for thyroid hormone are met almost exclusively by the supply from mother until the fetal thyroid starts to function. Thus early pregnancy thyroid biochemistry imbalance may have clinical implications for pregnancy outcome and development of the foetus. Thyroid hormones are known to play a role in brain development and maturation, and clinical management of thyroid disorders during pregnancy focuses on judicious interpretation and management of thyroid function during pregnancy (1-3).

Estrogen and elevated thyroxine binding globulin, elevated renal iodine clearance, and stimulation of the thyroid by human chorionic gonadotropin, all contribute to natural changes in maternal thyroid function during pregnancy. Such changes in these physiological parameters may affect the levels of thyroid stimulating hormone and thyroid hormones in circulation. These adaptive mechanisms do not always happen in women, leaving them with thyroid dysfunction including subclinical hypothyroidism, overt hypothyroidism, or hyperthyroidism (rarely). Certainthorium recommends that thyroid function tests must be interpreted with consideration to pregnancy, since there are changes in thyroid physiology during pregnancy (4-6).

Thyroid dysfunction in pregnant women is associated with poor maternal and fetal outcomes such as anemia, hypertension, low birth weight, preterm birth and impaired fetal growth. Besides birth outcomes, it has become more important to address the potential role of the imbalance of thyroid hormones during pregnancy on early neurodevelopment. The maternal thyroid hormones play a role in neuronal migration, myelination, synaptogenesis and cortical development. Abnormal maternal thyroid function has been suggested to affect neurodevelopment in previous studies and reviews, but the association has been shown to differ by population, timing of measurement of thyroid function, iodine status, treatment status, and developmental tools used (7-9).

The infant developmental milestones offer a useful clinical tool for early neurodevelopment. The milestones illustrate the development of gross motor skills, fine motor skills, language development, social and adaptive functioning. Achievement of milestones may be delayed due to a number of maternal and neonatal factors such as low birth weight, anemia, poor nutrition, and maternal illness or endocrine abnormalities during pregnancy. Thyroid biochemistry should be examined in addition to maternal and neonatal factors since the thyroid function may be influenced by physiological changes during pregnancy (10-12).

Local data from Pakistan regarding maternal thyroid biochemistry and infant developmental milestones remain limited. In tertiary-care settings, pregnant women may present with variable nutritional status, anemia, delayed antenatal booking, and limited routine thyroid screening. These contextual factors may increase the clinical importance of studying thyroid dysfunction during pregnancy. Therefore, the present study was conducted at Lady Reading Hospital, Peshawar, to determine the correlation between maternal thyroid biochemical parameters, physiological changes during pregnancy, and infant developmental milestones.

METHODOLOGY

The present study was a prospective observational analytical study carried out at Lady Reading Hospital, Peshawar from January 2024 to January 2025. The objective of the study was to evaluate the relationship between maternal thyroid hormone levels, physiological changes of pregnancy and infant developmental status. The study adopted non-probability, consecutive sampling and 77 pregnant women who met the study's criteria were included in the study.

A sample size was determined with the WHO sample size calculator for correlation based studies using a 95% confidence level, 80% power, and a moderate correlation between maternal thyroid hormone and infant developmental outcomes that was anticipated. 77 participants were retained for the last analysis. The pregnant women were screened for eligibility and enrolled in the study upon informed written consent obtained from them at the antenatal clinic of Lady Reading hospital.

In this study, the women were included who were pregnant in the age group 18-40 years, who have singleton pregnancy and who are willing to follow up. Women who had known chronic medical conditions, such as diabetes mellitus, chronic hypertension, renal disease, liver disease, epilepsy, autoimmune disease other than thyroid disease, multiple pregnancy, congenital fetal anomaly, or drug intake that is known to suppress thyroid function were excluded. Women who were missed at follow-up and whose laboratory or infant developmental information was missing were also not included in the final analysis.

The maternal demographic and obstetric data collected on a structured proforma after enrolment. These included maternal age, residency, education, socioeconomic status, gravida, parity, gestational age at enrollment, body mass index, previous miscarriage and family history of thyroid disease. Physiological parameters related to pregnancy were also obtained, such as maternal weight gain, hemoglobin level, blood pressure, heart rate, anemia, gestational hypertension, gestational diabetes, and intrauterine growth restriction and preterm delivery (if applicable).

Venous blood was drawn from each one of the participants using aseptic precautions for thyroid biochemical assessment. Standard laboratory methods that were available in the hospital lab were used to measure serum thyroid stimulating hormone (TSH), free triiodothyronine (Free T3), free thyroxine (Free T4) and anti-thyroid peroxidase antibodies (Anti-TPO). Thyroid status was determined by trimester-specific thyroid reference ranges where available as euthyroid, subclinical hypothyroidism, overt hypothyroidism, subclinical hyperthyroidism or overt hyperthyroidism. Where values for the individual trimesters were not available, standard pregnancy adjusted reference ranges are used.

Normal maternal physiological changes were monitored during pregnancy by regular antepartum monitoring. Anemia was determined by hemoglobin level and blood pressure was measured to detect hypertensive disorders of pregnancy. Laboratory data and hospital diagnosis were used to determine the presence of gestational diabetes. Data on gestational age at delivery, mode of delivery and pregnancy outcome were obtained post-delivery.

Neonatal data included infant sex, birth weight, gestational age at delivery, APGAR score at 1 and 5 minutes, low birth weight, preterm birth, admission to the neonatal intensive care unit and any clinically suspected neonatal complication. Low birth weight was defined as being less than 2.5 kg, and preterm birth defined as delivery prior to 37 completed weeks of gestation.

Follow-up assessments used age appropriate clinical milestone assessment to assess infant developmental milestones. Domains of development assessed were: gross motor, fine motor, language, social, and cognitive/adaptive milestones. Infants were classified as normal if they met the expected age milestones and as delayed if the infant did not meet one or more expected age milestones. The overall developmental delay was considered to be delay in any domain during follow-up.

The infant developmental milestone delay was the primary outcome variable. The factors of primary interest were maternal thyroid biochemical values, particularly TSH, Free T4, Free T3 and Anti-TPO antibody status. Other factors were maternal age, BMI, parity, gestational age, hemoglobin level, pregnancy complications, birth weight and gestational age at delivery.

The SPSS version 25 software was used to enter and analyse the data. Maternal age, BMI, gestational age, TSH, Free T3, Free T4, Anti-TPO level, hemoglobin level, birth weight and developmental score were presented as mean and standard deviation. Other qualitative variables like residence, parity, thyroid status, anemia, gestational hypertension/pregnancy induced hypertension, gestational diabetes, preterm delivery, low birth weight, admission to NICU and developmental delay were presented as frequency and percentage. The association of maternal thyroid status with infant developmental delay was examined using a chi square test. The mean values were compared between groups when appropriate using independent sample t-test. For normally distributed continuous variables, Pearson correlation was used and for non-normally distributed continuous variables, Spearman correlation was utilized. Potential confounding factors (maternal age, BMI, anemia, gestational age at delivery, and birth weight) were also taken into account in a binary logistic regression to identify predictors of infant developmental delay. A p value of ≤ 0.05 was considered statistically significant.

RESULTS

There were 77 pregnant women in total. The mean maternal age was 28.41 ± 4.92 years and majority of the respondents aged between 26 and 30 years. The mean gestational age when the thyroid assessment was completed was 18.63 ± 5.21 weeks. The majority of the women were multigravida (58.4%) with 32 (41.6%) being primigravida. The mean maternal BMI was 25.76 ± 3.84 kg/m².

Table 1. Baseline Maternal Demographic and Obstetric Characteristics

Variable	Frequency / Mean	Percentage / SD
Age, years	28.41	± 4.92
BMI, kg/m²	25.76	± 3.84
Gestational age at thyroid assessment, weeks	18.63	± 5.21
Gravida		
Primigravida	32	41.6%
Multigravida	45	58.4%
Residence		
Urban	43	55.8%
Rural	34	44.2%
Education status		
No formal education	14	18.2%
Primary/Middle	21	27.3%
Matric/Intermediate	27	35.1%
Graduate or above	15	19.5%
Socioeconomic status		
Low	28	36.4%
Middle	39	50.6%
High	10	13.0%
Family history of thyroid disease	9	11.7%
Previous miscarriage	11	14.3%

The mean serum TSH level was 3.18 ± 1.74 mIU/L, mean Free T4 was 1.07 ± 0.24 ng/dL, and mean Free T3 was 2.91 ± 0.51 pg/mL. 52 (67.5%) women had a normal thyroid biochemistry, and 25 (32.5%) had thyroid dysfunction. The most common abnormality was subclinical hypothyroidism (15, 19.5%).

Table 2. Maternal Thyroid Biochemical Profile During Pregnancy

Thyroid Parameter	Mean ± SD	Minimum–Maximum
TSH, mIU/L	3.18 ± 1.74	0.21–8.90
Free T4, ng/dL	1.07 ± 0.24	0.61–1.62
Free T3, pg/mL	2.91 ± 0.51	1.81–4.10
Anti-TPO antibody, IU/mL	28.64 ± 21.37	5.00–118.00
Thyroid Status	**Frequency**	**Percentage**
Euthyroid	52	67.5%
Subclinical hypothyroidism	15	19.5%
Overt hypothyroidism	5	6.5%
Subclinical hyperthyroidism	3	3.9%
Overt hyperthyroidism	2	2.6%
Anti-TPO positive	16	20.8%

Physiological changes and pregnancy outcomes: The mean maternal hemoglobin level was 10.82 ± 1.21 g/dL. 31 (40.3%) women had anemia. Nine (11.7%) encountered gestational hypertension and seven (9.1%) had gestational diabetes. The average gestational age at delivery was 37.84 ± 1.62 weeks. There were 12 (15.6%) cases of preterm delivery.

Table 3. Pregnancy-Related Physiological Changes and Birth Outcomes

Variable	Frequency / Mean	Percentage / SD
Hemoglobin, g/dL	10.82	± 1.21
Maternal weight gain, kg	9.34	± 3.12
Systolic blood pressure, mmHg	116.52	± 11.43
Diastolic blood pressure, mmHg	74.18	± 8.26
Gestational age at delivery, weeks	37.84	± 1.62
Birth weight, kg	2.83	± 0.46
Anemia	31	40.3%
Gestational hypertension	9	11.7%
Gestational diabetes mellitus	7	9.1%
Preterm delivery	12	15.6%
Low birth weight	14	18.2%
NICU admission	10	13.0%
APGAR score <7 at 5 minutes	6	7.8%

Follow-up was used to determine infant developmental milestones. Overall, 60 (77.9%) infants reached age-appropriate development milestones and 17 (22.1%) infants were delayed in one or more developmental milestones. Gross motor delay was the most commonly observed delay, reported in 12 (15.6%) infants, followed by language delay in 10 (13.0%) infants.

Table 4. Infant Developmental Milestone Status

Developmental Domain	Normal	Delayed
Gross motor milestone	65 (84.4%)	12 (15.6%)
Fine motor milestone	69 (89.6%)	8 (10.4%)
Language milestone	67 (87.0%)	10 (13.0%)
Social milestone	70 (90.9%)	7 (9.1%)
Cognitive/adaptive milestone	68 (88.3%)	9 (11.7%)
Overall developmental status	60 (77.9%)	17 (22.1%)

An infant thyroid condition was associated with higher rates of developmental delay when compared to healthy mothers. Of the euthyroid mothers, 7 (13.5%) mothers had delayed milestones, while 10 (40.0%) mothers with thyroid dysfunctions had delayed milestones. This difference was significant (p = 0.009).

Table 5. Association between Maternal Thyroid Status and Infant Developmental Delay

Maternal Thyroid Status	Normal Development	Delayed Development	p-value
Euthyroid, n=52	45 (86.5%)	7 (13.5%)	
Thyroid dysfunction, n=25	15 (60.0%)	10 (40.0%)	0.009

There was a significant negative correlation between maternal TSH and infant developmental score (r = -0.346, p = 0.002), such that a higher maternal TSH was related to a lower infant developmental score. Within the physiological range of thyroid function, maternal Free T4 exhibited a significant positive correlation with infant developmental score (r = 0.392, p = 0.001) indicating improved performance of the infant in relation to increased maternal Free T4 levels. There was also a weak negative correlation with the anti-TPO antibody level and the developmental score (r = -0.241, p = 0.035).

Table 6. Correlation between Maternal Thyroid Biochemistry and Infant Developmental Score

Maternal Thyroid Variable	Correlation Coefficient	p-value
TSH	-0.346	0.002
Free T4	0.392	0.001
Free T3	0.174	0.131
Anti-TPO antibody level	-0.241	0.035
Birth weight	0.288	0.011
Gestational age at delivery	0.263	0.021

A binary logistic regression was used to determine predictors of infant developmental delay. Maternal thyroid dysfunction was associated with infant developmental delay after adjusting for maternal age, BMI, anemia, gestational age at delivery and birth weight. There was a 3.42-fold increased risk of developmental delay for infants with mothers with thyroid dysfunctions relative to infants with mothers with euthyroid status (AOR = 3.42,

95% CI: 1.12–10.47, p = 0.031). Developmental delay was also found to be significantly associated with low birth weight (AOR = 2.89, 95% CI: 1.01–8.91, p = 0.048).

Table 7. Logistic Regression Analysis for Predictors of Infant Developmental Delay

Predictor Variable	Adjusted Odds Ratio	95% CI	p-value
Maternal thyroid dysfunction	3.42	1.12–10.47	0.031
Raised TSH	2.76	1.03–7.86	0.044
Low Free T4	3.18	1.08–9.72	0.036
Anti-TPO positivity	2.21	0.76–6.43	0.143
Maternal anemia	1.69	0.58–4.91	0.336
Low birth weight	2.89	1.01–8.91	0.048
Preterm delivery	2.37	0.79–7.13	0.123

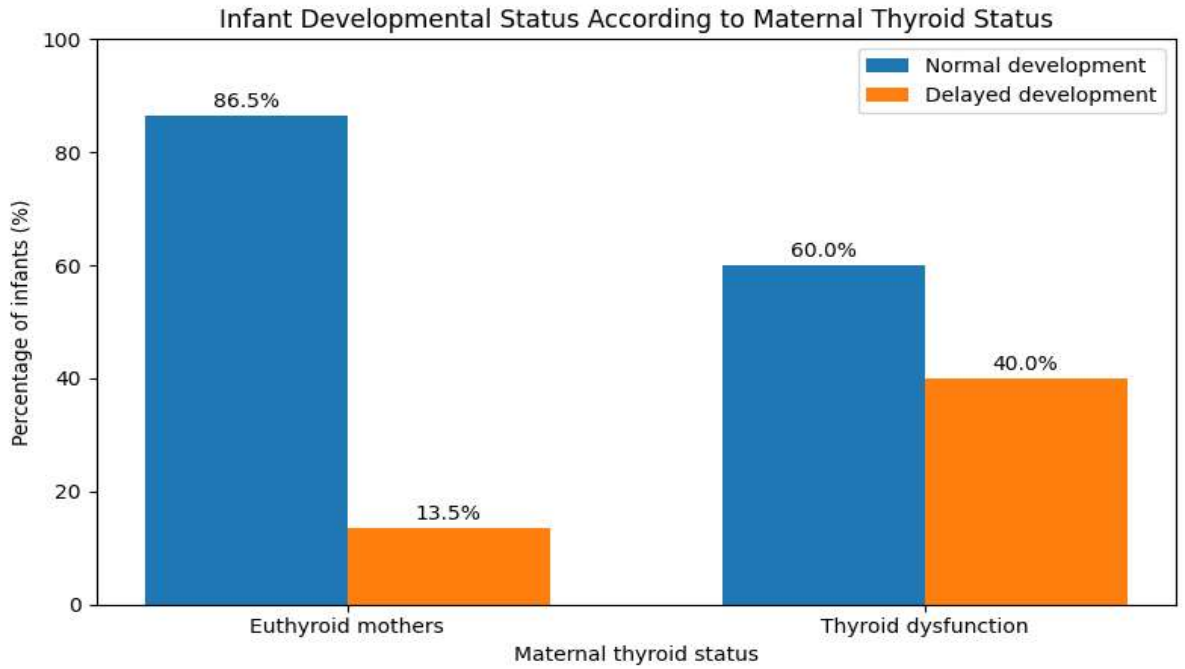

Figure 1. Infant Developmental Status According to Maternal Thyroid Status.

Developmental delay was more frequent among infants born to mothers with thyroid dysfunction compared with euthyroid mothers.

DISCUSSION

The present study evaluated the relationship between maternal thyroid biochemistry, pregnancy-related physiological changes, and infant developmental milestones among 77 pregnant women at Lady Reading Hospital, Peshawar. The findings showed that 32.5% of mothers had thyroid dysfunction, with subclinical hypothyroidism being the most frequent abnormality. Infant developmental delay was observed in 22.1% of cases and was significantly more common among infants born to mothers with thyroid dysfunction. This finding supports the biological role of maternal thyroid hormones in fetal and early infant neurodevelopment (13, 14).

Raised maternal TSH was significantly correlated with decreased infant developmental score and maternal Free T4 was significantly correlated with increased infant developmental score in this study. This suggests that the children of mothers with higher TSH and lower Free T4 had a greater risk of having poor performance in developmental areas. These results are biologically relevant, since thyroid hormones are known to play a role in fetal brain maturation, neuronal differentiation, and myelination. Previous studies have suggested that thyroid hormone deficiency during pregnancy can be linked to adverse neurodevelopmental outcome but the findings were not consistent because of study design, timing of testing and methods of neurodevelopmental assessment (15, 16).

In the present study, infants of mothers with thyroid dysfunction had a higher prevalence of developmental delay, as is generally believed in the clinic that maternal thyroid hormone imbalance may have an impact on fetal brain development in sensitive periods of gestation. Thyroid disease during pregnancy is a condition

that can affect the mother and her baby, and the American Thyroid Association insists on evaluating thyroid function during pregnancy. The current study noted developmental delay in the gross motor and language areas, which may indicate early vulnerability in the neurodevelopment areas; however, further studies would be needed in order to determine if these delays remain beyond infancy (17).

Maternal physiological factors also appeared to contribute to infant outcome. Low birth weight and lower gestational age at delivery showed significant associations with developmental score. This suggests that the pathway between maternal thyroid dysfunction and infant development may not be direct alone but may also operate through pregnancy complications, fetal growth restriction, preterm delivery, or neonatal vulnerability. WHO also emphasizes that maternal health during pregnancy and the postnatal period is closely linked with infant well-being and long-term outcomes (18). Therefore, thyroid screening should be considered as part of a broader maternal health approach rather than as an isolated laboratory investigation (19).

The role of maternal physiologic variables also seemed to be an influence in infant outcome. There was a significant association of weights of less than 5.5 lbs and gestational age less than 37 weeks with the developmental score. This indicates that the association of maternal thyroid dysfunction and infant development might not be direct but could also be mediated by pregnancy complications, fetal growth restriction, preterm delivery or neonatal vulnerability. WHO also highlights that maternal health in pregnancy and the postpartum stage is intimately connected to that of infants and their future development (20). Thus, the screening of the thyroid should be part of an overall maternal health plan rather than a stand-alone laboratory test.

Logistic regression analysis revealed that maternal thyroid dysfunction was still significantly associated with infant developmental delay regardless of maternal age, BMI, anemia, gestational age at delivery, or birth weight. This supports the hypothesis that thyroid dysfunction is a significant risk factor, independent of other factors, for poor developmental outcomes. The confidence interval was, however, large, perhaps because of the relatively small sample size. Care should be taken in interpreting these results and validate with larger multicenter studies to assess thyroid function in each trimester and to use the same developmental tools.

Some studies have reported weaker or non-significant associations between maternal thyroid function and child neurodevelopment, highlighting that this relationship is complex. For example, one cohort from a high fish-consuming population reported no clear association between maternal thyroid function and neurodevelopmental outcomes at 20 months. Such differences may be explained by variations in iodine intake, nutrition, timing of hormone measurement, maternal treatment status, infant age at assessment, and socioeconomic factors. In the present local setting, maternal anemia, nutritional limitations, and delayed antenatal care may have amplified the effect of thyroid dysfunction on infant outcomes (21).

However, some studies have found less strong or non-significant links between maternal thyroid function and neurodevelopment of children, indicating that this relationship is complex. For instance, a group with high fish consumption had no clear linkage between maternal thyroid function and neurodevelopmental results at 20 months. These disparities could be attributed to differences in iodine consumption, nutrition, when the hormone was measured, whether or not the mother was receiving treatment, whether or not the infant was assessed at birth or later, and socioeconomic factors. Anemia, malnutrition and poor attendance at ante-pregnancy checkups were issues that could have exacerbated the impact of thyroid dysfunction in the present local context on the outcomes of infants.

The strength of this study was the association of maternal biochemical parameters not just with thyroid status but also with pregnancy physiology and infant developmental assessment. However, there are some restrictions to be taken into account. The number of samples was small, and the study was performed in a single tertiary-care hospital, which will restrict the generalizability of the results. Developmental milestones were clinically evaluated; however the use of more detailed standardized neurodevelopmental scales may be useful to gain greater precision. Thyroid function was tested during the pregnancy, however, multiple trimester testing was not fully analyzed. In spite of these restrictions, the study offers local information that supports the value of the assessment of maternal thyroid status during pregnancy.

CONCLUSION

Maternal thyroid dysfunction during pregnancy was significantly associated with delayed infant developmental milestones. Raised TSH and

reduced Free T4 were correlated with lower infant developmental scores, while developmental delay was more frequent among infants born to mothers with thyroid dysfunction compared with euthyroid mothers. Low birth weight also contributed to developmental risk. These findings suggest that early identification and management of thyroid dysfunction during pregnancy may improve maternal and infant outcomes. Routine thyroid evaluation in antenatal care, especially among high-risk women, and developmental follow-up of exposed infants are recommended.

REFERENCES

1. Lucaccioni L, Ficara M, Cenciarelli V, Berardi A, Predieri B, Iughetti L. Long term outcomes of infants born by mothers with thyroid dysfunction during pregnancy. Acta bio-medica : Atenei Parmensis. 2020;92(1):e2021010.
2. Chen Y, Luo Z-C, Zhang T, Fan P, Ma R, Zhang J, et al. Maternal Thyroid Dysfunction and Neuropsychological Development in Children. The Journal of Clinical Endocrinology & Metabolism. 2023;108(2):339-50.
3. Xu D, Zhong H. Correlation Between Hypothyroidism During Pregnancy and Glucose and Lipid Metabolism in Pregnant Women and Its Influence on Pregnancy Outcome and Fetal Growth and Development. 2022;Volume 9 - 2022.
4. Murillo-Llorente MT, Llorca-Colomer F, Pérez-Bermejo MJN. Relationship between thyroid status during the first trimester of pregnancy and neonatal well-being. 2021;13(3):872.
5. Eng L, Lam LJN. Thyroid function during the fetal and neonatal periods. 2020;21(1):e30-e6.
6. Mégier C, Dumery G, Luton DJM. Iodine and thyroid maternal and fetal metabolism during pregnancy. 2023;13(5):633.
7. Anifantaki F, Pervanidou P, Lambrinoudaki I, Panoulis K, Vlahos N, Eleftheriades MJFin. Maternal prenatal stress, thyroid function and neurodevelopment of the offspring: A mini review of the literature. 2021;15:692446.
8. Zhou Y-L, Ren J-B, Ding R, Yu T-T, Fan J-X. Effects of maternal internal environment on early life growth and development. In Utero Pediatrics: Research & Practice: Springer; 2023. p. 49-78.
9. Lopes-Pereira M, Roque S, Costa P, Quialheiro A, Santos NC, Goios A, et al. Impact of iodine supplementation during preconception, pregnancy and lactation on maternal thyroid homeostasis and offspring psychomotor development: protocol of the IodineMinho prospective study. 2020;20(1):693.
10. Zdraveska N, Kocova MJCe. Thyroid function and dysfunction in preterm infants—Challenges in evaluation, diagnosis and therapy. 2021;95(4):556-70.
11. Salmeen KE, Block-Kurbisch IJ. Thyroid physiology during pregnancy, postpartum, and lactation. Maternal-Fetal and Neonatal Endocrinology: Elsevier; 2020. p. 53-60.
12. Marty S, Beekhuijzen M, Charlton A, Hallmark N, Hannas BR, Jacobi S, et al. Towards a science-based testing strategy to identify maternal thyroid hormone imbalance and neurodevelopmental effects in the progeny–part II: how can key events of relevant adverse outcome pathways be addressed in toxicological assessments? 2021;51(4):328-58.
13. Kolobarić A, Delić L, Raguž MJJAoB, Research C. Can the pathological conditions of the thyroid gland during pregnancy affect the course and outcome of pregnancy and the parameters of the newborn? 2023;2(2):108-18.
14. Deori R, Deori C, Das J, Kutum T. Metabolic changes and biochemical monitoring during pregnancy. Current Trends in the Diagnosis and Management of Metabolic Disorders: CRC Press; 2023. p. 29-47.
15. Moog NK, Entringer S, Heim C, Wadhwa PD, Kathmann N, Buss C. Influence of maternal thyroid hormones during gestation on fetal brain development. Neuroscience. 2017;342:68-100.
16. Thompson W, Russell G, Baragwanath G, Matthews J, Vaidya B, Thompson-Coon J. Maternal thyroid hormone insufficiency during pregnancy and risk of neurodevelopmental disorders in offspring: A systematic review and meta-analysis. Clinical endocrinology. 2018;88(4):575-84.
17. Alexander EK, Pearce EN, Brent GA, Brown RS, Chen H, Dosiou C, et al. 2017 Guidelines of the American Thyroid Association for the Diagnosis and Management of Thyroid Disease During Pregnancy and the Postpartum. Thyroid : official journal of the American Thyroid Association. 2017;27(3):315-89.
18. Souza JP, Day LT, Rezende-Gomes AC, Zhang J, Mori R, Baguiya A, et al. A global analysis of the determinants of maternal health and transitions in maternal mortality. 2024;12(2):e306-e16.
19. Miranda RA, de Moura EG, Lisboa PCJE. Adverse perinatal conditions and the

developmental origins of thyroid dysfunction—lessons from animal models. 2023;79(2):223-34.
20. Monaghan AM, Mulhern MS, Mc Sorley EM, Strain JJ, Winter T, van Wijngaarden E, et al. Associations between maternal thyroid function in pregnancy and child neurodevelopmental outcomes at 20 months in the Seychelles Child Development Study, Nutrition Cohort 2 (SCDS NC2). Journal of nutritional science. 2021;10:e71.
21. Anandappa S, Joshi M, Polanski L, Carroll PVJTaie, metabolism. Thyroid disorders in subfertility and early pregnancy. 2020;11:2042018820945855.