THE ROLE OF THYROID FUNCTION IN PREGNANCY: ENSURING HEALTHY OUTCOMES FOR MOTHER AND CHILD

Abu Bakar Sarfaraz[1], Shamim Akram[2], Iram Arshad[3], Anam Arshad[4], Emaan Fatima[5], M. Zamir Ahmad[6], Muhammad Mustansar[7].

[1] *Asst Professor, Pakistan Red Crescent Medical College, Lahore.*

[2] *Associate Professor, RLKU Medical College, Lahore.*

[3] *Asst Professor, RLKU Medical College, Lahore.*

[4] *Associate Professor, Central Park Medical College, Lahore.*

[5] *Medical Student, Aga Khan University, Karachi.*

[6] *Principal, Professor of Biochemistry, Islam Medical College, Sialkot.*

[7] *Professor, Head of Dept, Pakistan Red Crescent Medical College, Lahore.*

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Corresponding Author:
Abu Bakar Sarfaraz

Abstract

Introduction: Pregnancy induces significant physiological changes, including alterations in thyroid function, which can profoundly affect maternal and fetal health. The thyroid gland is essential in regulating metabolism, growth, and development, and its hormones are important for normal pregnancy progression. Disruptions in thyroid function can increase the risk of hindering the development of the baby's brain and raise the risk of issues like preterm birth, low birth weight, gestational hypertension, and preeclampsia. They may contribute to cognitive and developmental delays.

Objectives: The aim of the study is to investigate the effect of thyroid function on pregnancy, highlighting the mechanisms through which thyroid hormone imbalances affect maternal and fetal outcomes. It also discusses early detection, appropriate management, and the role of screening in improving pregnancy outcomes and long-term health for both mother and child.

Methodology: The study was conducted at Pak Red Crescent Teaching Hospital, Dina Nath, Pakistan, over 9 months. Samples were collected from women who were not pregnant and women with healthy pregnancies in their third trimester along with the cord blood of the newborn babies by these pregnant women. The samples were collected in the 3rd trimester from the pregnant women, and the biochemical analysis was performed on the serum.

Results: Pregnant women's Thyroid profile included Triiodothyronine (T_3), Thyroxine (T_4), Thyroid-Stimulating Hormone (TSH), Thyroglobulin Antibodies (Tg Ab, and V (TPO Ab); their averages were noticeably greater than those of the group that wasn't pregnant. Levels of T_3, T_4, and TSH in cord blood of 50 neonates were evaluated as well. Out of 50, three newborns were found to have low levels of T_3, T_4, and TSH.

Conclusion: This study highlights the importance of thyroid function during pregnancy, as imbalances can cause complications. Early detection, management, and routine screening are essential for improving outcomes and ensuring long-term health for both mother and child.

Introduction:

Pregnancy induces significant physiological changes, including alterations in thyroid function, which can have profound effects on both maternal and fetal health [1]. The thyroid gland is essential in regulating metabolism, growth, and development, and its hormones are essential for normal pregnancy progression [2]. Disruptions in thyroid function, such as hypothyroidism or hyperthyroidism, may hinder the development of the baby's brain and raise the risk of issues like preterm birth, low birth weight, gestational hypertension, and preeclampsia, which can contribute to cognitive and developmental delays [3].

The total T_4 and T_3 are increased as a result of increasing levels of TBG and decreased levels of TSH caused by the thyrotrophic action of hCG secreted by the placenta [4]. The thyroid produces triiodothyronine (T_3) and tetraiodothyronine (T_4). Thyroid-stimulating hormone (TSH), which is secreted by the pituitary gland, controls hormone production [5].

Negative feedback to the hypothalamus generates the releasing hormone, which controls the production of TSH. The majority of T_4 is converted to the active form, T_3, in target tissues. Thyroid-binding globulin (TBG) proteins bind most of the hormones in circulation. The only active hormone is the unbound free hormone [6].

During pregnancy, the thyroid gland's size grows by 10% to 40%. The iodine demand rises as a result of a 50% increase in T_3 and T_4 production. During pregnancy, the hypothalamic-pituitary-thyroid feedback mechanisms operate correctly [7]. T_3 and T_4 protein binding, however, has changed significantly. Furthermore, human chorionic gonadotropin (HCG), the hormone of pregnancy, functions on thyroid receptors similarly to TSH. Total and bound levels of T_3 and T_4 will rise as a result of the rise in TBGs during pregnancy, while free T_3 (FT_3) and free T_4 (FT_4) remain unaltered [8].

During the first 10 weeks, TSH is usually low when HCG is high, and it will rise after 10–12 weeks when HCG levels decline. As HCG levels are high, serum FT_4 is at its peak, and as they decline, it falls [9].

Thyroid physiology undergoes significant changes throughout pregnancy to ensure adequate thyroid hormone supply to both mother and child. Because the fetal thyroid only begins to produce a significant amount of thyroid hormone at 20 weeks gestation, this is crucial in the early stages of pregnancy. Thus, up until this point, the fetus is dependent on the mother's thyroid hormone supply [10].

In the pregnant mother, Euthyroidism is important for healthy fetal growth and development. When T_4 levels are low and TSH levels are high, a diagnosis of primary hypothyroidism is made. About two to three percent of pregnant women have hypothyroidism [11]. It raises the risk of low

birth weight, low APGAR score, preterm delivery, placental abruption, perinatal mortality, and pregnancy loss [12].

High levels of T_4 and/or free T_3 in the blood lead to hyperthyroidism. Graves' disease is the most frequent cause of hyperthyroidism during pregnancy [13]. Thyroid storm, maternal congestive heart failure, intrauterine growth retardation, low birth weight, miscarriages, gestational hypertension, and stillbirth are among the risks associated with hyperthyroidism, which affects 0.2% of pregnancies overall [14].

In pregnancy, screening for thyroid disease can help identify patients with thyroid dysfunction who require treatment and eventually decrease the rates of complications [15].

This study explores the effect of thyroid functioning on pregnancy, highlighting the mechanisms through which thyroid hormone imbalances influence maternal and fetal outcomes. Early detection, proper management, and screening are the keys to improving pregnancy outcomes and long-term health for both mother and child.

Materials and Methods

Whole blood was collected in EDTA-containing tubes, from women who were non-pregnant (n=50) and women with pregnancy in their 3rd trimester (n=50) for evaluation of thyroid profile. After the delivery of the pregnant woman, cord blood samples were collected for the thyroid profile.

The study was conducted at Pak Red Crescent Teaching Hospital, Dina Nath, Pakistan. The non-pregnant (married) women's (n=50) blood samples were collected from colleagues, family members, and research fellows. Samples from all participants were collected after giving written and informed consent. The serum samples were separated from the blood and analyzed for the thyroid profile.

The "Research and Ethics Committee" at the Institute of Molecular Biology and Biotechnology (IMBB), The University of Lahore, Lahore, Pakistan, consented to this research work and biochemical analysis (IRB Approval No.: UOL/IMBB/CRIMM-1389/88/01).

Pregnant females, primigravida /multigravida, in the last trimester, were incorporated in the present study. Known cases of pregnancy with hypothyroidism or hyperthyroidism on treatment were also included.

The age groups of non-pregnant, and pregnant women ranged between 25 and 35 years. Newborns with one day of age were included to see the development of hypothyroidism from the thyroid profile of the cord blood. Multiple pregnancies, chronic hypertension, diabetes mellitus, and bad obstetric history were excluded.

The participants with medications for psychosis, depression, metabolic syndrome, chronic infection, and malnutrition syndrome were also excluded from this study.

Results:

Table I shows the demographic characteristics of non-pregnant and pregnant women. Mean age, weight, and BMI were comparable between the groups, and no statistically significant differences were observed ($p > 0.05$).

Variable	Non-Pregnant (n=50)	Pregnant (n=50)	p-value
Weight (kg)	70.88 +/- 5.29	82.26 +/- 2.29	0034
Age (years)	29.29 +/- 3.11	33.26 +/- 3.88	0.124
BMI (kg/m^2)	23.79 +/- 2.23	26.35 +/- 3.17	0.038

Table 1: Demographic profile of pregnant and non-pregnant women

Table 2 shows the thyroid profile of non-pregnant and normal pregnant women. Mean serum T4, TSH, TgAb, and TPOAb levels were higher in pregnant women than in non-pregnant women, whereas T3 levels were slightly lower. However, none of these differences were statistically significant ($p > 0.05$).

Variable	Non-Pregnant women	Pregnant women	p-value
T4 (Pmol/L)	8.225 +/- 1.060	13.626 +/- 4.59	0.067
T3 (pg/mL)	4.02 +/- 3.04	3.92 +/- 2.56	0.531
TSH (mIU/L)	4.98 +/- 0.09	5.23 +/- 0.02	0.510
TgAB (ng/mL)	48.33 +/- 13.14	66.26 +/- 4.26	0.514
TPO Ab (IU/mL)	10.16 +/- 9.78	102.26 +/- 4.26	0.532

Table 2: Thyroid profile of non-pregnant, normal pregnant women

Table 3 shows the demographic characteristics and thyroid profile of healthy neonates and neonates with congenital hypothyroidism. Cord blood samples from 50 neonates were analyzed, of whom 3 were diagnosed with congenital hypothyroidism. Neonates with congenital hypothyroidism had lower mean T3, T4, and TSH levels than healthy neonates; however, these differences were not statistically significant ($p > 0.05$).

Variable	Healthy Neonates (n=47)	Congenital Hypothyroidism (n=3)	p-value
Weight (pounds)	6.34 +/- 0.49	6.16 +/- 0.31	0.025
Age	1 +/- 0	1 +/- 0	N/A

(days)			
Height (cm)	52.78 +/- 3.59	51.22 +/- 2.52	0.013
T_3 (pg/mL)	2.31 +/- 0.73	1.98 +/- 1.14	0.501
T_4 (ng/dL)	1.10 +/- 0.30	1.01 +/- 0.53	0.520
TSH (µU/mL)	12.74 +/- 3.56	6.77 +/- 1.93	0.540

Table 3: General demographic, and thyroid profile of healthy neonates and those with congenital hypothyroidism

In contrast, significant differences were observed in weight and height between the two groups ($p < 0.05$).

Discussion:

Thyroid hormones play a pivotal role in maternal health and fetal development during pregnancy. Physiological adaptations occurring during gestation significantly affect thyroid function, including increased production of thyroxine-binding globulin, enhanced renal iodine clearance, and stimulation of thyroid hormone production by human chorionic gonadotropin [16]. In the present study, thyroid parameters (T_3, T_4, TSH, TgAb, and TPOAb) were compared between pregnant and non-pregnant women. Although mean values of T_4, TSH, TgAb, and TPOAb were higher in pregnant women compared to non-pregnant women, these differences were not statistically significant ($p > 0.05$), whereas T_3 levels were slightly lower in pregnant women.

Maternal thyroid hormones are particularly critical during the first trimester when the fetal thyroid gland is not fully functional. Adequate maternal thyroxine supply is necessary for normal fetal neurodevelopment, especially for neuronal migration, myelination, and synaptogenesis. Adequate maternal thyroxine supply is necessary for normal fetal neurodevelopment, especially for neuronal migration, myelination, and synaptogenesis [17]. The present findings are partially consistent with previous studies reporting mild physiological increases in thyroid-binding activity and hormonal fluctuations during pregnancy without significant alteration in circulating free hormone levels in healthy pregnancies. Similar results were reported by Glinoer et al., who observed adaptive thyroid changes during pregnancy without overt dysfunction in euthyroid women [18].

Iodine sufficiency is another critical determinant of optimal thyroid function during pregnancy. The World Health Organization recommends increased iodine intake during pregnancy because maternal requirements rise substantially due to increased thyroid hormone synthesis and fetal needs. Even mild iodine deficiency may impair fetal brain development and increase the risk of maternal hypothyroxinemia [19].

Therefore, adequate iodine supplementation remains a key public health strategy, particularly in regions with marginal iodine intake.

However, some studies have reported significant reductions in free T4 levels during pregnancy, particularly in iodine-deficient populations, which was not observed in our cohort. The discrepancy may be attributed to differences in iodine nutritional status, sample size, and gestational age at sampling.

In the present study, thyroid autoantibodies (TgAb and TPOAb) were elevated in pregnant women compared to non-pregnant women, but without statistical significance. This finding aligns with reports suggesting that mild immunological modulation occurs during pregnancy; however, clinically significant antibody positivity is generally associated with autoimmune thyroid disease rather than normal pregnancy physiology [20].

Maternal hypothyroidism, whether overt or subclinical, has been associated with adverse neurocognitive outcomes in offspring, including reduced intelligence quotient (IQ) and impaired psychomotor development [21].

Regarding neonatal outcomes, congenital hypothyroidism cases demonstrated lower mean T3, T4, and TSH levels compared to healthy neonates; however, these differences were not statistically significant. This lack of significance is likely due to the very small number of affected neonates (n=3), which limits statistical power. Similar limitations have been reported in neonatal screening studies with low case numbers, where meaningful biochemical trends are observed but not statistically demonstrable.

Weight and height differences between healthy and affected neonates were statistically significant, although these findings should be interpreted cautiously due to the small sample size of the affected group.

Serum TSH remains the most sensitive screening marker for thyroid dysfunction in pregnancy, while FT4 provides additional diagnostic value in distinguishing overt and subclinical disease states [22]. Early identification of thyroid dysfunction is important due to its established association with adverse maternal and fetal outcomes, as supported by multiple studies [23].

Overall, although no statistically significant differences in thyroid hormones were observed between groups in this study, the observed trends highlight the physiological variation in thyroid function during pregnancy and the importance of continued monitoring.

Conclusion:

This study demonstrates physiological variations in thyroid hormone levels between pregnant and non-pregnant women, with no statistically significant differences observed in T3, T4, TSH, TgAb, and TPOAb levels. In neonates, differences in thyroid hormone levels between healthy and congenital hypothyroidism cases were observed, highlighting the clinical relevance of thyroid

screening in early life. Overall, the findings emphasize the importance of thyroid function assessment during pregnancy and neonatal screening for early identification of dysfunction.

Limitations:

The study provides useful preliminary data on thyroid profiles in pregnancy and neonates. However, subgroup analysis in neonates with congenital hypothyroidism was limited by the small number of cases identified. Future studies with larger, multicentre cohorts and longitudinal follow-up would further strengthen these findings and help establish clearer clinical correlations.

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