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Hypothyroidism in Pregnancy and Its Association with Feto-Maternal Outcomes: A Prospective Cohort Study

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Abstract

Background: Thyroid hormones play a vital role in maternal metabolic regulation and fetal neurodevelopment. Maternal hypothyroidism during pregnancy has been associated with adverse fetomaternal outcomes, though prevalence and impact vary across populations.

Methods: This prospective cohort study was conducted at MDM Hospital, Dr. S.N. Medical College, over seven months (June 2025–May 2026). A total of 100 pregnant women with singleton pregnancies and gestational age <20 weeks were recruited. Serum TSH levels were measured, and participants were categorized as euthyroid (86%), subclinical hypothyroid (11%), and overt hypothyroid (3%). Hypothyroid women received levothyroxine with dose titration every six weeks. Maternal outcomes (preeclampsia, GDM, abruption, PPH) and fetal outcomes (birth weight, APGAR scores, RDS, NICU admission) were recorded and followed until 24 hours post-delivery.

Results: The prevalence of hypothyroidism was 14.0% (11% subclinical, 3% overt). Mean gestational age at delivery was similar across groups (p=0.983). Mode of delivery showed significant association with thyroid status (χ^2=8.079, p=0.018), with LSCS in 90.9% of subclinical hypothyroid women vs. 46.5% of euthyroid women. Maternal complications occurred at higher frequencies in subclinical hypothyroidism (preeclampsia 18.2%, PPH 18.2%) but were not statistically significant. Neonatal outcomes showed trends toward lower APGAR scores and higher NICU admission in hypothyroid groups, with NICU indication significantly associated

with thyroid status (χ^2=20.250, p=0.009). All subclinical hypothyroid women achieved controlled TSH (<4.0 mIU/L) by delivery.

Conclusion: Maternal hypothyroidism during pregnancy shows associations with adverse fetomaternal outcomes and higher LSCS rates. Universal first-trimester screening, early levothyroxine initiation, and regular TSH monitoring are essential to optimize pregnancy outcomes.

Keywords: Hypothyroidism; Pregnancy Complications; Thyroid Hormones; Pregnancy Outcome; Thyrotropin

Introduction

The thyroid gland plays a crucial role in regulating metabolic processes through the secretion of thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3). These hormones influence cellular metabolism, thermoregulation, oxygen consumption, and the functional activity of multiple organ systems. The secretion of thyroid hormones is regulated by the hypothalamic–pituitary–thyroid axis, wherein the hypothalamus releases thyrotropin-releasing hormone (TRH), stimulating the anterior pituitary to secrete thyroid-stimulating hormone (TSH). TSH subsequently acts on the thyroid gland to promote synthesis and release of T3 and T4. The majority of circulating thyroid hormone is bound to plasma proteins, particularly thyroxine-binding globulin (TBG), while the unbound fraction represents the biologically active form responsible for metabolic activity at the cellular level.[1]

During fetal development and early childhood, thyroid hormones are particularly important for brain maturation and skeletal growth. Deficiency during these critical periods may result in irreversible cognitive impairment and growth retardation.

As pregnancy progresses, the maternal thyroid gland undergoes functional adaptations to meet the increased requirement for thyroid hormones. There is an approximate 30–50 percent increase in thyroid hormone production to maintain adequate maternal and fetal metabolic activity.[2]

Rising levels of estrogen stimulate hepatic production of thyroxine-binding globulin, leading to increased binding of circulating thyroid hormones and a subsequent rise in total T3 and T4 concentrations. Human chorionic gonadotropin, produced by the placenta, also plays a significant role due to its structural similarity to thyroid-stimulating hormone. This hormone mildly stimulates the thyroid gland, particularly during the first trimester, resulting in increased thyroid hormone synthesis and a transient reduction in serum thyroid-stimulating hormone levels. These physiological changes reflect normal maternal adaptation and should be distinguished from pathological thyroid dysfunction.[3] Maternal thyroid hormones play a particularly important role in early gestation, as the fetal thyroid gland begins functioning only around 10–12 weeks of gestation and becomes fully functional by approximately 18–20 weeks. Prior to this time, the foetus relies heavily on maternal thyroid hormone supply, particularly for neurological development. Adequate maternal thyroid hormone availability is therefore essential during the first trimester, when critical processes such as neuronal migration, myelination, and synapse formation are actively occurring. Insufficient thyroid hormone levels during this period may lead to impaired neurodevelopment and long-term cognitive deficits.[4] In addition to neurological development, thyroid hormones also influence fetal growth, skeletal maturation, and placental function.

Table 1: Normal Thyroid Function Test Ranges in Pregnancy[5]

Parameter	First Trimester	Second Trimester	Third Trimester	Units
TSH	0.1 – 2.5	0.2 – 3.0	0.3 – 3.0–3.5	mIU/L
Free T4	0.8 – 1.53 (approx.)	0.7 – 1.20 (approx.)	0.7 – 1.20 (approx.)	ng/dL
Total T4	↑ 1.5 × non-pregnant value	↑ 1.5 × non-pregnant value	↑ 1.5 × non-pregnant value	µg/dL

Hypothyroidism is defined as a clinical condition characterized by deficient production or action of thyroid hormones, resulting in reduced metabolic activity. In pregnancy, hypothyroidism may present in two primary forms: overt hypothyroidism and subclinical hypothyroidism. Overt hypothyroidism is typically characterized by elevated serum TSH levels accompanied by decreased free thyroxine concentrations.[6] Subclinical hypothyroidism, in contrast, is defined by elevated TSH levels with normal circulating free T4 concentrations.

The diagnosis of hypothyroidism during pregnancy relies primarily on biochemical evaluation, with TSH being the most sensitive indicator of thyroid dysfunction. Pregnancy-specific reference ranges are often used because normal TSH values vary according to gestational age.[7] In general, lower TSH levels are observed during the first trimester due to the stimulatory effect of hCG, whereas TSH levels gradually increase during the second and third trimesters.[5]

Primary hypothyroidism arises from intrinsic dysfunction of the thyroid gland itself, whereas secondary or central hypothyroidism results from pituitary or hypothalamic disorders affecting TSH secretion. The most common cause of hypothyroidism during pregnancy in iodine-sufficient regions is chronic autoimmune thyroiditis, also known as Hashimoto's thyroiditis.[5] Regular monitoring of TSH levels is recommended to ensure that thyroid function remains within pregnancy-specific reference ranges.

In this context, the present study was conducted in order to evaluate the impact of maternal hypothyroidism on fetal and maternal outcomes during pregnancy.

Aim & Objectives

Aim: To evaluate the impact of maternal hypothyroidism on fetal and maternal outcomes during pregnancy

Objectives

- To estimate the proportion of pregnant females with hypothyroidism
- To compare the foetal outcomes in hypothyroid vs euthoyroid pregnant women.
- To compare the maternal outcomes in hypothyroid vs euthoyroid pregnant women.

Materials and Methods

Inclusion Criteria

Pregnant women fulfilling the following criteria were included in the study:

Age ≥ 18 years

Singleton pregnancy

Gestational age less than 20 weeks at the time of recruitment

Attending ANC clinic at MDM Hospital

Planning for delivery at MDM Hospital

Hypothyroidism defined as serum Thyroid Stimulating Hormone (TSH) level >4 mIU/L

Euthyroid defined as serum Thyroid Stimulating Hormone (TSH) level <4 mIU/L

Willingness to participate and follow-up until 24 hours post-delivery

Exclusion Criteria

Pregnant women with the following conditions were excluded from the study:

- Molar pregnancy
- Known chronic medical disorders such as hypertension, diabetes mellitus, and hypothyroidism

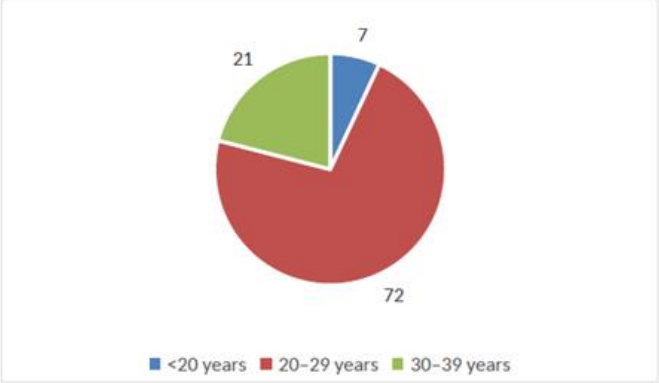
Study Procedure

The present study aimed to evaluate the impact of maternal hypothyroidism on fetal and maternal outcomes during pregnancy. Specific objectives included estimating the proportion of pregnant females with hypothyroidism and comparing fetal and maternal outcomes between hypothyroid and euthyroid pregnant women.

This prospective cohort study was conducted at the Department of Obstetrics and Gynecology, MDM Hospital, Dr. S.N. Medical College, over a period of seven months from June 2025 to May 2026. A total of 100 pregnant women with singleton pregnancies and gestational age less than 20 weeks attending the antenatal clinic were recruited using consecutive sampling after fulfilling inclusion criteria. Participants aged 18 years or above with no known chronic medical disorders were included, while those with molar pregnancy or known chronic conditions were excluded. Written informed consent was obtained from all participants. Routine antenatal investigations including complete blood count, blood grouping, liver and renal function tests, glucose challenge test, and serum TSH estimation were performed. Participants were categorized into euthyroid, subclinical hypothyroid, and overt hypothyroid groups based on TSH levels. Oral levothyroxine therapy was initiated for hypothyroid patients, and TSH levels were reassessed every six weeks with dose adjustment accordingly. Maternal outcomes including preeclampsia, gestational diabetes mellitus, abruption, and postpartum hemorrhage were recorded. Fetal outcomes including birth weight, APGAR scores, respiratory distress syndrome, NICU admission, and discharge status were assessed. All participants were followed until 24 hours post-delivery. Categorical variables were analyzed using the Chi-square test, and quantitative variables were analyzed using Student's t-test, with p-value <0.05 considered statistically significant.

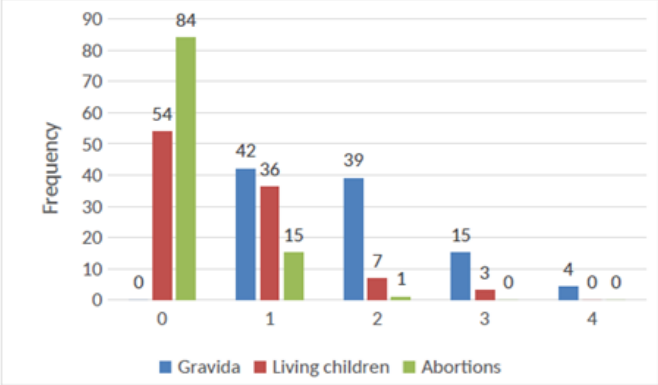
Results

Figure 1: Distribution of Study Participants According to Age of patients (years)



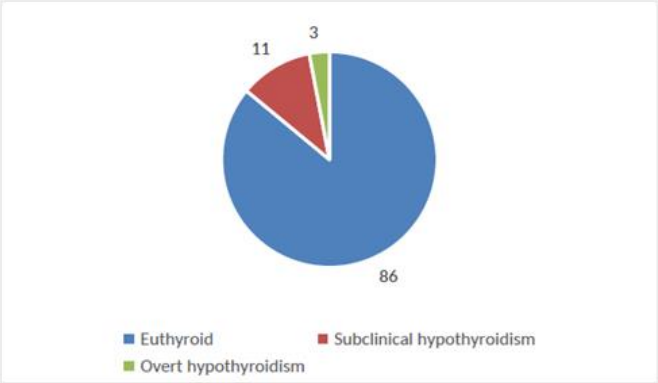
The majority of participants (72.0%) were aged 20–29 years, with a mean gestational age at booking of 13.58 ± 3.86 weeks and a mean BMI of 24.13 ± 3.62 kg/m².

Figure 2: Distribution of Study Participants According to Obstetric Profile



The obstetric profile revealed that 42.0% were primigravida, 39.0% were gravida 2, 54.0% had no living children, and 84.0% had no history of abortion.

Figure 3: Distribution of Thyroid Status Among Study Participants (n = 100)



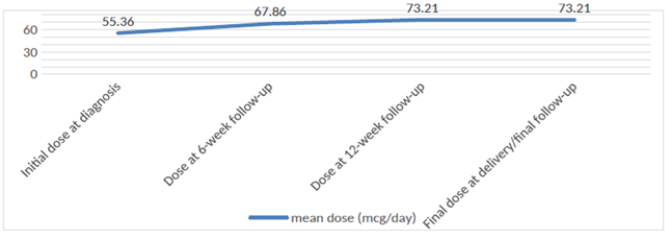
86.0% of participants were euthyroid. Subclinical hypothyroidism was present in 11.0%, while overt hypothyroidism was present in 3.0%.

Table 1: Distribution of Levothyroxine Therapy Among Study Participants (n = 100)

Levothyroxine (LT4) therapy initiated	Frequency (n)	Percentage (%)
No	86	86.0
Yes	14	14.0

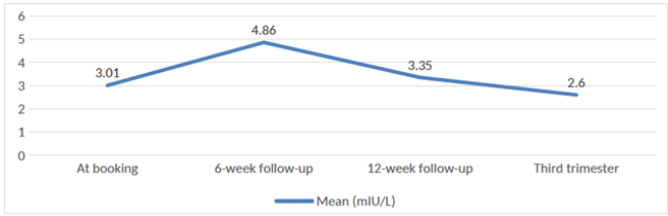
Levothyroxine therapy was initiated in 14.0% of participants, while 86.0% did not receive levothyroxine therapy.

Figure 4: Levothyroxine Dose Titration Among Hypothyroid Pregnant Women (n = 14)



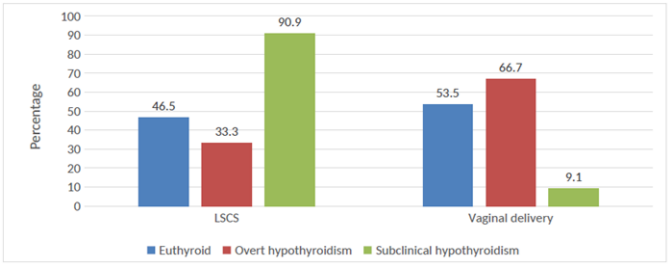
Levothyroxine dose titration among hypothyroid pregnant women. The mean initial dose was 55.36 ‡ 28.04 mcg/day, increasing to 67.86 ‡ 38.52 mcg/day at 6 weeks and 73.21 ‡ 47.50 mcg/day at 12 weeks. The final dose at delivery or final follow-up remained 73.21 ‡ 47.50 mcg/day.

Figure 5: Serial Thyroid Stimulating Hormone (TSH) Levels During Pregnancy Among Hypothyroid Women Receiving Levothyroxine Therapy



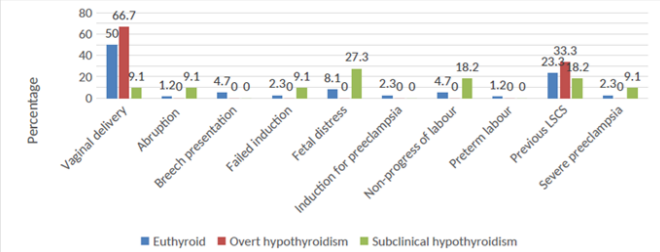
The mean TSH was 3.01 + 2.57 mIU/L at booking, 4.86 ‡ 2.77 mIU/L at 6-week follow-up, 3.35 + 2.08 mlU/L at 12-week follow-up, and 2.60 = 1.57 mIU/L in the third trimester.

Figure 6: Association of Mode of Delivery with Thyroid Status



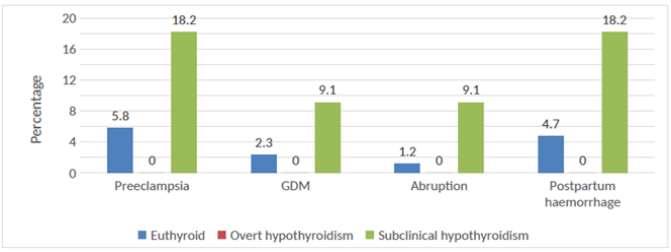
There was a statistically significant association between mode of delivery and thyroid status (P=0.018). LSCS was reported in 46.5% of euthyroid women, 33.3% of women with overt hypothyroidism, and 90.9% of women with subclinical hypothyroidism

Figure 7: Association of Indication for LSCS Delivery with Thyroid Status



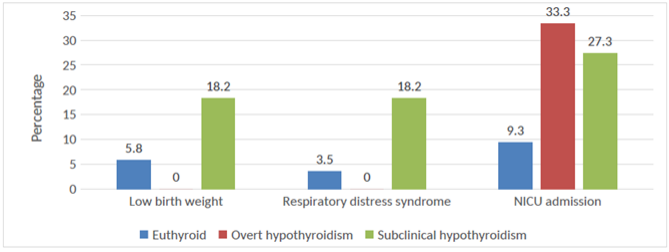
Previous LSCS was the most frequent indication overall, accounting for 23.0%, followed by fetal distress at 10.0%. The association between LSCS indication and thyroid status was not statistically significant (p = 0.420).

Figure 8: Association of Maternal Outcomes with Thyroid Status



Preeclampsia occurred in 7.0%, Gestational Diabetes Mellitus in 3.0%, Abruption in 2.0%, and Postpartum haemorrhage in 6.0% overall. None of these outcomes showed a statistically significant association with thyroid status: preeclampsia (p = 0.283), GDM (p = 0.443), abruption (p = 0.203), and postpartum haemorrhage (P= 0.186).

Figure 9: Association of Neonatal Outcomes with Thyroid Status



Low birth weight was present in 7.0%, respiratory distress syndrome in 5.0%, and NICU admission in 12.0% overall. No statistically significant association was observed between thyroid status and low birth weight (p = 0.283), respiratory distress syndrome (p =0.100), or NICU admission (p = 0.116).

Table 2: Association of NICU Indication with Thyroid Status

NICU indication	Euthyroid n=86 n (%)	Overt hypothyroidism n=3 n (%)	Subclinical hypothyroidism n=11 n (%)	Total n=100 n (%)	χ^2 value	p-value
Not admitted	78 (90.7)	2 (66.7)	8 (72.7)	88 (88.0)	20.250	0.009
Prematurity	3 (3.5)	1 (33.3)	0 (0.0)	4 (4.0)		
Fetal Growth Restriction	2 (2.3)	0 (0.0)	1 (9.1)	2 (2.0)		
RDS	3 (3.5)	0 (0.0)	2 (18.2)	5 (5.0)		

There was a statistically significant association between NICU indication and thyroid status (χ^2 = 20.250, p = 0.009). Overall, 88.0% of neonates were not admitted to the NICU. Prematurity accounted for 4.0% of NICU indications, fetal growth restriction for 3.0%, respiratory distress syndrome for 5.0%.

Discussion

The present prospective cohort study evaluated the effect of maternal hypothyroidism on maternal and neonatal outcomes among pregnant women screened before 20 weeks of gestation. The overall prevalence of hypothyroidism was 14%, with subclinical hypothyroidism accounting for the majority of cases. Although women with hypothyroidism, particularly those with subclinical disease, experienced higher

frequencies of adverse maternal and neonatal outcomes, most associations were not statistically significant. However, cesarean section was significantly more common among women with subclinical hypothyroidism, and serial levothyroxine therapy resulted in improved thyroid function during pregnancy.

The prevalence observed in the present study is comparable with previous Indian studies reporting rates between 10% and 24%. Justin SA et al. and Khawale R et al. reported prevalence rates similar to our findings, whereas Sharma DV et al. documented a higher prevalence and Kunwar R et al. reported lower rates. These variations may be attributed to differences in iodine sufficiency, study population, diagnostic criteria, and trimester-specific TSH cut-off values. The predominance of subclinical hypothyroidism emphasizes the importance of routine antenatal screening, as affected women are often asymptomatic but remain at risk of pregnancy-related complications.

Women with hypothyroidism showed higher frequencies of preeclampsia, gestational diabetes mellitus, placental abruption, and postpartum hemorrhage compared with euthyroid women, although these differences were not statistically significant. Similar associations have been reported by Hizkiyahu et al. and Vamja et al., who demonstrated increased risks of hypertensive disorders, gestational diabetes, and postpartum hemorrhage among hypothyroid pregnancies. The lack of statistical significance in the present study is likely related to the relatively small sample size and the limited number of women with overt hypothyroidism. In addition, early diagnosis, levothyroxine therapy, and close antenatal monitoring may have reduced the occurrence of adverse maternal outcomes.

A significant association was observed between thyroid status and mode of delivery, with women having subclinical hypothyroidism showing the highest cesarean section rate. Fetal been reported by Khawale et al., suggesting that thyroid dysfunction may increase the likelihood of operative delivery. However, obstetric management depends on multiple maternal and fetal factors; therefore, the higher cesarean rate cannot be attributed solely to thyroid dysfunction.

Serial assessment of thyroid function demonstrated progressive improvement following levothyroxine dose adjustment. Most women with subclinical hypothyroidism achieved satisfactory biochemical control by delivery, whereas women with overt hypothyroidism were less likely to attain target TSH levels despite treatment. These findings highlight the importance of early diagnosis and periodic TSH monitoring throughout pregnancy, as physiological changes during gestation increase maternal thyroid hormone requirements. The reduction in mean TSH levels during follow-up indicates that individualized levothyroxine dose titration is effective in achieving adequate thyroid control.

Regarding neonatal outcomes, birth weight and APGAR scores were lower among hypothyroid women, particularly those with subclinical hypothyroidism, although the differences were not statistically significant. Respiratory distress syndrome and NICU admissions also occurred more frequently in hypothyroid pregnancies. While the overall NICU admission rate was comparable between groups, the indications for NICU admission differed significantly according to maternal thyroid status, suggesting a possible association between maternal thyroid dysfunction and neonatal morbidity. Similar observations have been reported by Mahadik et

al., Sharma DV et al., and Dubey et al., who described increased risks of low birth weight, respiratory complications, and NICU admission among infants born to hypothyroid mothers. The relatively favourable neonatal outcomes observed in the present study may be explained by timely initiation of levothyroxine therapy, regular thyroid monitoring, and comprehensive antenatal care.

Gestational age at delivery was similar across all study groups, indicating that adequately treated hypothyroidism may not necessarily increase the risk of preterm birth. Likewise, maternal and neonatal discharge outcomes were favourable irrespective of thyroid status, suggesting that appropriate treatment and close surveillance can minimize adverse pregnancy outcomes. These findings support recent evidence demonstrating that optimal thyroid management during pregnancy can improve both maternal and neonatal prognosis.

monitoring of thyroid function, and evaluation of both maternal and neonatal outcomes. However, certain limitations should be acknowledged. The study was conducted at a single tertiary care centre with a relatively small sample size, particularly in the overt hypothyroid group, which may have limited the ability to detect statistically significant differences. In addition, thyroid autoantibody status, iodine status, medication adherence, and long-term neurodevelopmental outcomes of infants were not assessed. Future multicentre studies involving larger populations and long-term follow-up are required to better define the impact of maternal hypothyroidism on pregnancy and neonatal outcomes.

Limitations

The present study had several limitations that should be acknowledged when interpreting the findings. The sample size of 100 participants, although adequate for the primary objectives, was relatively small, which may have limited the statistical power to detect significant differences in maternal and fetal outcomes between the hypothyroid and euthyroid groups. The small number of overt hypothyroid cases (n=3) particularly constrained the ability to draw meaningful comparisons within this subgroup. The study was conducted at a single tertiary care center with a specific population demographic, which may limit the generalizability of the findings to other settings with different patient populations and healthcare practices. The study did not assess thyroid peroxidase antibody status, which is an important marker for autoimmune thyroid disease and could have provided additional insights into the etiology of hypothyroidism in the study population.

Conclusion

The present prospective cohort study was conducted to evaluate the impact of maternal hypothyroidism on fetal and maternal outcomes during pregnancy among 100 pregnant women attending a tertiary care center. The study found a prevalence of hypothyroidism of 14.0%, comprising 11.0% subclinical and 3.0% overt hypothyroidism, indicating a substantial burden of thyroid dysfunction in the antenatal population. Maternal outcomes including preeclampsia, gestational diabetes mellitus, abruption, and postpartum hemorrhage occurred at higher frequencies in the subclinical hypothyroidism group, although these associations did not reach statistical significance. Fetal outcomes including birth weight, APGAR scores, respiratory distress syndrome, and NICU admission also showed trends toward poorer outcomes in hypothyroid women without statistical significance. The mode of delivery demonstrated a statistically significant association with thyroid status, with a notably higher rate of lower

segment cesarean section (90.9%) in the subclinical hypothyroidism group compared to euthyroid women (46.5%). Serial TSH monitoring and levothyroxine dose titration were effective in achieving controlled TSH levels in the majority of hypothyroid women, with all subclinical hypothyroid patients achieving controlled status (<4.0 mIU/L) by delivery. Maternal and neonatal discharge status at 24 hours post-delivery was favorable in the majority of cases, with 94.0% of mothers and 88.0% of neonates being stable or discharged. The dysfunction during pregnancy, early initiation of levothyroxine therapy when indicated, and regular monitoring to optimize maternal and fetal outcomes. While the frequencies of adverse outcomes were generally higher in hypothyroid women, effective management appears to minimize the clinical impact of thyroid dysfunction during pregnancy. Larger multicenter studies with extended follow-up are recommended to further elucidate the relationship between maternal hypothyroidism and long-term outcomes in both mothers and their offspring. findings of this study underscore the importance of universal screening for thyroid.

References

1. Al-Suhaimi EA, Khan FA. Thyroid glands: physiology and structure. In: Emerging concepts in endocrine structure and functions. Singapore: Springer Nature Singapore; 2022. p. 133–160.
2. Pirahanchi Y, Toro F, Jialal I. Physiology, thyroid stimulating hormone.
3. Bagga AD, Johnson BP, Zhang Q. A minimal human physiologically based kinetic model of thyroid hormones and chemical disruption of plasma thyroid hormone binding proteins. Front Endocrinol (Lausanne). 2023;14:1168663.
4. Köhrle J. Local activation and inactivation of thyroid hormones: the deiodinase family. Mol Cell Endocrinol. 1999;151(1-2):103–119.
5. Forhead AJ, Fowden AM. Thyroid hormones in fetal growth and prepartum maturation. J Endocrinol. 2014;221(3):R87–103.