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**Original Research Article**

# A study on the effect of exogenous thyroxine on known hypothyroid and the newly diagnosed hypothyroid patients during first trimester of pregnancy

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## ABSTRACT

**Background:** Hypothyroidism complicates pregnancy and is associated with adverse maternal and fetal outcomes if inadequately treated. This study evaluated the effect of levothyroxine on thyroid function and pregnancy outcomes among women with known and newly diagnosed hypothyroidism during the first trimester.
**Methods:** A prospective observational study was conducted in an Obstetrics and Gynaecology department from September 2024 to October 2025. Fifty pregnant women diagnosed with hypothyroidism in the first trimester (25 known, 25 newly diagnosed) were enrolled via consecutive sampling. Thyroid function tests (TSH, free T3, free T4) were assessed at baseline, six weeks post-intervention, and during the second and third trimesters. Maternal complications, delivery mode, neonatal outcomes, and NICU admissions were recorded.
**Results:** Participants' mean age was 24.30±3.04 years. Subclinical hypothyroidism was present in 54% (n=27) and overt hypothyroidism in 46% (n=23). Women with overt hypothyroidism had significantly higher TSH and lower free T3 and free T4 levels throughout pregnancy compared to those with subclinical hypothyroidism (p=0.001). Dose adjustments were required in 92% of women at baseline, decreasing to 68% by the third trimester. Term normal vaginal deliveries occurred in 90% of women, while 10% underwent caesarean section. Complications included miscarriage (18%), gestational diabetes (12%), gestational hypertension (10%), postpartum haemorrhage (10%), and abruptio placentae (4%). NICU admission was required for 19.5% of live births. Mean birth weight was 2.67±0.23 kg, and mean APGAR score was 8.62±1.12.
**Conclusions:** Early diagnosis, regular monitoring, and timely levothyroxine adjustments improve thyroid control and foster favorable maternal and neonatal outcomes. Continuous surveillance remains essential, particularly for overt cases.

**Keywords:** Hypothyroidism, Levothyroxine, Pregnancy, Thyroid-stimulating hormone, Maternal outcome, Neonatal outcome, First trimester

## INTRODUCTION

Hypothyroidism represents one of the most prevalent endocrine disorders encountered during gestation, affecting approximately 2% to 5% of pregnant women globally. In developing nations such as India, the burden is disproportionately higher due to regional variations in nutritional iodine intake, environmental goitrogens, and varying degrees of screening coverage.[3,4] The disease manifests across a spectrum ranging from subclinical hypothyroidism defined as an elevated thyroid-stimulating hormone (TSH) with normal free thyroxine (fT4) levels-to overt hypothyroidism, where elevated TSH is accompanied by subnormal fT4 concentrations.[2,5] Both

entities, if unrecognized or inadequately optimized, pose substantial risks to both maternal stability and fetal development.[6,7] Pregnancy triggers marked maternal physiological alterations that place increased metabolic demand on the thyroid axis.[8] Rising concentrations of human chorionic gonadotropin (hCG) during early gestation directly stimulate maternal TSH receptors, while elevated estrogen levels induce a twofold rise in circulating thyroxine-binding globulin (TBG).[8,9]

Furthermore, enhanced renal glomerular filtration accelerates iodide clearance, and transplacental transfer of iodine and thyroxine to the developing fetus further depletes maternal pools.[9,10] In euthyroid women, the thyroid gland responds by increasing thyroid hormone output by 30–50%; however, women with pre-existing impaired functional reserve or subclinical dysfunction fail to adapt, resulting in relative or absolute maternal hypothyroxinemia.[2,11]

During the critical first trimester, the fetal neurodevelopmental apparatus relies entirely on the transplacental supply of maternal thyroxine, as the fetal thyroid architecture does not achieve functional maturity until approximately 12 to 14 weeks of gestation.[12,13] Maternal thyroid hormones act through nuclear receptors in the fetal brain to regulate neuronal migration, myelination, and cortical architecture formation.[13,14]

Consequently, unmanaged maternal thyroid deficiency during early organogenesis increases the incidence of early pregnancy loss, spontaneous miscarriage, recurrent gestational loss, placental abruption, gestational hypertension, pre-eclampsia, and anemia.[1,15,16] From a neonatal perspective, persistent uncorrected maternal hypothyroxinemia correlates with increased risks of fetal growth restriction, preterm delivery, low birth weight, elevated neonatal intensive care unit (NICU) admission rates, and subtle long-term neurocognitive or intellectual deficits in childhood.[17,18]

Current clinical management guidelines from the American thyroid association (ATA) and the Endocrine Society emphasize establishing trimester-specific reference intervals for TSH and free thyroid hormones to guide prompt therapeutic intervention.[2,19] Exogenous synthetic levothyroxine (L-T4) remains the gold standard treatment due to its proven safety profile, reliable gastrointestinal absorption, long half-life, and precise clinical efficacy in restoring systemic euthyroidism.[2,20]

Women entering pregnancy with established hypothyroidism almost universally require an immediate dose increment of 25–30% upon confirmation of conception to meet augmented physiological demands.[2,21] Conversely, women newly diagnosed during routine first-trimester screening require rapid initiation of levothyroxine alongside serial reassessment to titrate serum TSH into targeted gestational thresholds.[2,22] Despite well-defined international management frameworks, significant empirical knowledge gaps remain regarding thyroid disease dynamics in Indian clinical settings.[4,23] Indian pregnant women frequently present with distinct baseline clinical profiles, including variable iodine nutritional status, higher rates of subclinical disease, and distinct dietary habits.[4,24]

Furthermore, comparative clinical data evaluating treatment responses, serial dosage requirements, and fetomaternal outcomes between women entering pregnancy with pre-existing hypothyroidism and those newly diagnosed during first-trimester screening remain limited in the South Asian population.[21,24]

Therefore, the present prospective observational study was conducted to evaluate the response to exogenous levothyroxine therapy among pregnant women presenting in the first trimester with known versus newly diagnosed hypothyroidism.

By analyzing serial alterations in TSH, fT3, and fT4 alongside maternal and neonatal clinical outcomes, this study seeks to provide prospective clinical evidence supporting early screening, individualized levothyroxine dose titration, and rigorous antenatal surveillance to optimize pregnancy outcomes.

## METHODS

### *Study design and setting*

A prospective observational study was conducted in the Department of Obstetrics and Gynaecology, Sri Venkateshwaraa Medical College Hospital and Research Centre, Puducherry, India, during the study period from September 2024 to October 2025. The study was carried out after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants before enrolment.

### *Study population and sampling technique*

The study included pregnant women presenting during the first trimester who were either previously diagnosed with hypothyroidism or newly diagnosed with hypothyroidism during the current pregnancy.

A total sample size of 50 participants fulfilling the eligibility criteria was recruited consecutively during the study period using a non-probability convenience consecutive sampling technique. All eligible pregnant women presenting to the antenatal clinic during the study period were enrolled until the sample size was achieved.

### *Inclusion criteria*

Pregnant women in the first trimester (gestational age ≤13 weeks) who were either previously diagnosed with hypothyroidism receiving levothyroxine therapy or newly diagnosed with hypothyroidism during the current

pregnancy, and who were willing to provide written informed consent and comply with the follow-up schedule, were included in the study.

### *Exclusion criteria*

Conversely, women with hyperthyroidism or other endocrine disorders affecting thyroid function, multiple pregnancies, pre-existing chronic renal disease, chronic liver disease, significant cardiac disease, autoimmune disorders other than autoimmune thyroid disease, or those with incomplete follow-up or missing laboratory investigations were excluded from participation.

### *Study procedure*

A detailed obstetric and medical history was obtained from all participants, followed by a complete general and obstetric examination. Baseline demographic variables including maternal age, parity, BMI, and hypothyroidism status (known or newly diagnosed) were recorded. Laboratory investigations included serum TSH, fT3, and fT4. Additional routine antenatal investigations such as haemoglobin estimation and random blood glucose were performed according to institutional protocol.

Participants received oral levothyroxine therapy according to standard obstetric and endocrinology guidelines. Women with pre-existing hypothyroidism underwent dose adjustment whenever indicated, while newly diagnosed women were initiated on appropriate levothyroxine therapy. Dose modifications were guided by serial thyroid function tests with the objective of maintaining trimester-specific TSH targets.

Thyroid function tests were repeated at four predefined intervals: baseline (first trimester), six weeks after initiation or dose adjustment of levothyroxine, during the second trimester, and during the third trimester. Dose adjustment was performed whenever thyroid function values were outside the recommended trimester-specific reference range.

### *Outcome measures*

The primary outcome measured serial changes in serum TSH, fT3, and fT4 following levothyroxine therapy across the predefined intervals. Secondary maternal outcomes evaluated miscarriage, gestational diabetes mellitus, gestational hypertension, abruptio placentae, postpartum haemorrhage, and mode of delivery. Secondary neonatal outcomes included gestational age at delivery, birth weight, APGAR score at birth, and neonatal intensive care unit (NICU) admission.

### *Statistical analysis*

Data were entered into Microsoft Excel and analysed using the Statistical Package for the Social Sciences (SPSS) software version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation (SD), while categorical variables were presented as frequencies and percentages.

Comparisons of continuous variables between overt and subclinical hypothyroidism groups were performed using the independent Student's t-test. Categorical variables were analysed using the Chi-square test or Fisher's exact test, wherever appropriate. A p value of $<0.05$ was considered statistically significant.

## RESULTS

A total of 50 pregnant women diagnosed with hypothyroidism during the first trimester were included in the study. Of these, 25 (50%) were previously known cases of hypothyroidism, while the remaining 25 (50%) were newly diagnosed during the current pregnancy. Based on clinical and biochemical criteria at baseline, 27 (54%) participants were diagnosed with subclinical hypothyroidism and 23 (46%) had overt hypothyroidism. The baseline demographic and clinical characteristics are detailed in Table 1.

Serial assessment of thyroid function demonstrated a progressive improvement following levothyroxine therapy across pregnancy. As shown in Table 2, women with overt hypothyroidism consistently had significantly higher serum TSH levels and significantly lower mean serum free T3 and free T4 concentrations compared to those with subclinical hypothyroidism at baseline, six weeks post-treatment, during the second trimester, and during the third trimester ($p=0.001$ for all parameters).

Levothyroxine dose adjustment was required in the majority of participants throughout pregnancy to achieve target TSH levels. Dose modification was required in 46 women (92.0%) at baseline, 45 (90.0%) at six weeks, 41 (82.0%) during the second trimester, and 34 (68.0%) during the third trimester, reflecting progressive stabilization of thyroid function over time (Table 3).

Out of 50 women, 9 (18.0%) experienced miscarriage (5 in the subclinical group and 4 in the overt group). Among the 41 women who completed pregnancy beyond the period of viability (22 subclinical, 19 overt), normal vaginal delivery occurred in 37 women (90.2%; 20 subclinical, 17 overt), while 4 women (9.8%; 2 subclinical, 2 overt) underwent caesarean section.

The distribution of maternal complications according to thyroid status is detailed in Table 4. Among the 41 live births, NICU admission was required in 8 neonates (19.5%, 4 in subclinical and 4 in overt hypothyroid groups). Overall neonatal parameters demonstrated a mean birth weight of 2.67±0.23 kg and a mean APGAR score at birth of 8.62±1.12 (Table 5).

**Table 1: Baseline demographic and clinical characteristics of study participants (n=50).**

| Variables | Frequency (N) | % |
|---|---|---|
| **Age group (in years)** | | |
| 18–25 | 35 | 70.0 |
| 26–35 | 15 | 30.0 |
| Mean age (in years) | 24.30±3.04 | - |
| **Hypothyroidism status** | | |
| Known hypothyroidism | 25 | 50.0 |
| Newly diagnosed hypothyroidism | 25 | 50.0 |
| **Parity** | | |
| Primigravida | 29 | 58.0 |
| Multigravida | 21 | 42.0 |
| **Body mass index (kg/m²)** | | |
| Normal (18.5–22.9) | 24 | 48.0 |
| Overweight (23–24.9) | 14 | 28.0 |
| Obese (≥25) | 12 | 24.0 |
| **Type of hypothyroidism** | | |
| Overt hypothyroidism | 23 | 46.0 |
| Subclinical hypothyroidism | 27 | 54.0 |

**Table 2: Serial comparison of thyroid function parameters (TSH, fT3, fT4) between overt and subclinical hypothyroidism groups.**

| Parameter and follow-up period | Overt hypothyroidism (n=23) (Mean±SD) | Subclinical hypothyroidism (n=27) (Mean±SD) | P value |
|---|---|---|---|
| **TSH (mIU/l)** | | | |
| Baseline | 10.30±3.07 | 7.09±1.54 | 0.001 |
| 6 weeks post-treatment | 8.71±3.15 | 5.88±1.48 | 0.001 |
| Second trimester | 7.82±3.21 | 4.82±1.37 | 0.001 |
| Third trimester | 7.19±3.18 | 4.21±1.35 | 0.001 |
| **Free T3 (fT3, pg/ml)** | | | |
| Baseline | 2.33±0.48 | 2.95±0.53 | 0.001 |
| 6 weeks post-treatment | 2.61±0.49 | 3.26±0.54 | 0.001 |
| Second trimester | 2.83±0.50 | 3.45±0.55 | 0.001 |
| Third trimester | 2.98±0.50 | 3.60±0.57 | 0.001 |
| **Free T4 (fT4, ng/dl)** | | | |
| Baseline | 0.67±0.12 | 1.03±0.14 | 0.001 |
| 6 weeks post-treatment | 0.91±0.13 | 1.29±0.15 | 0.001 |
| Second trimester | 1.10±0.16 | 1.49±0.16 | 0.001 |
| Third trimester | 1.24±0.17 | 1.61±0.18 | 0.001 |

Independent Student's t-test; $p<0.05$ considered statistically significant.

**Table 3: Levothyroxine dose adjustment pattern during follow-up (n=50).**

| Follow-up period | Dose adjusted n (%) | No dose adjustment n (%) |
|---|---|---|
| **Baseline** | 46 (92.0) | 4 (8.0) |
| **6 weeks** | 45 (90.0) | 5 (10.0) |
| **Second trimester** | 41 (82.0) | 9 (18.0) |
| **Third trimester** | 34 (68.0) | 16 (32.0) |

**Table 4: Maternal outcomes, mode of delivery, and obstetric complications according to type of hypothyroidism.**

| Outcome/complication | Subclinical hypothyroidism (n=27) N (%) | Overt hypothyroidism (n=23) N (%) | Total (n=50) N (%) |
|---|---|---|---|
| **Miscarriage** | 5 (18.5) | 4 (17.4) | 9 (18.0) |

Continued.

| Outcome/complication | Subclinical hypothyroidism (n=27) N (%) | Overt hypothyroidism (n=23) N (%) | Total (n=50) N (%) |
|---|---|---|---|
| **Mode of delivery (n=41)*** | n=22 | n=19 | n=41 |
| **Normal vaginal delivery** | 20 (90.9) | 17 (89.5) | 37 (90.2) |
| **Caesarean section** | 2 (9.1) | 2 (10.5) | 4 (9.8) |
| **Maternal complications (n=50)**** | | | |
| **Gestational diabetes mellitus** | 4 (14.8) | 2 (8.7) | 6 (12.0) |
| **Gestational hypertension** | 3 (11.1) | 2 (8.7) | 5 (10.0) |
| **Postpartum haemorrhage** | 3 (11.1) | 2 (8.7) | 5 (10.0) |
| **Abruptio placentae** | 1 (3.7) | 1 (4.3) | 2 (4.0) |

*Percentages for mode of delivery calculated among women who delivered past viability (n=41). Complications are non-mutually exclusive.

**Table 5: Perinatal characteristics and NICU admissions among live births (n=41).**

| Parameter/outcome | Subclinical hypothyroidism (n=22) | Overt hypothyroidism (n=19) | Total cohort/Mean±SD |
|---|---|---|---|
| **NICU admission, N (%)** | | | |
| Yes | 4 (18.2) | 4 (21.1) | 8 (19.5) |
| No | 18 (81.8) | 15 (78.9) | 33 (80.5) |
| **Clinical parameters (Mean±SD)** | | | |
| Haemoglobin (g/dl) | - | - | 10.35±0.82 |
| Random blood glucose (mg/dl) | - | - | 129.18±26.77 |
| Birth weight (kg) | - | - | 2.67±0.23 |
| APGAR score at birth | - | - | 8.62±1.12 |

## DISCUSSION

Hypothyroidism during pregnancy is associated with adverse maternal and neonatal outcomes if not diagnosed and treated promptly.[1,6] Early initiation of levothyroxine therapy and regular monitoring of thyroid function are essential to maintain euthyroidism throughout pregnancy.[2,20] The present prospective observational study evaluated the effect of exogenous levothyroxine therapy among women with known and newly diagnosed hypothyroidism during the first trimester and assessed its impact on maternal and neonatal outcomes.

The mean age of the study population was 24.30±3.04 years, with the majority (70%) belonging to the 18–25-year age group. This finding is comparable to previous Indian studies, where hypothyroidism was predominantly observed among women in the reproductive age group.[3,21] More than half of the study participants (58%) were primigravidae, which is consistent with reports suggesting that thyroid dysfunction is frequently identified during routine antenatal screening in first pregnancies.[4,21] In the present study, 54% of women had subclinical hypothyroidism, while 46% had overt hypothyroidism. Similar observations have been reported by Luo et al and Zhou et al who demonstrated that subclinical hypothyroidism is more common than overt hypothyroidism among pregnant women.[4,6] This emphasizes the importance of antenatal thyroid screening because women with subclinical disease are often asymptomatic but remain at risk of adverse pregnancy outcomes if untreated.[6,15] Serial assessment of thyroid function demonstrated a progressive reduction in serum TSH levels with corresponding improvement in free T3 and free T4 concentrations following levothyroxine therapy. Women with overt hypothyroidism consistently had significantly higher TSH levels and lower free thyroid hormone levels than those with subclinical hypothyroidism throughout pregnancy (p=0.001).

These findings indicate that although levothyroxine therapy improves thyroid function in both groups, women with overt hypothyroidism require closer monitoring and more frequent dose adjustments to achieve target thyroid hormone levels. Similar findings have been reported by Gao et al and Luo et al who observed improved thyroid function following individualized levothyroxine dose adjustment during pregnancy.[6,7] Levothyroxine dose adjustment was required in 92% of women at baseline, decreasing to 68% by the third trimester. This reflects the increased physiological requirement for thyroid hormone during early pregnancy and the importance of serial thyroid function monitoring.[2,21] Current recommendations advise reassessment of thyroid function every 4–6 weeks during pregnancy to ensure maintenance of trimester-specific TSH targets.[2,19] The findings support these recommendations and highlight that dose modification is frequently required, particularly during the first half of pregnancy. Normal vaginal delivery was achieved in the majority of women, while only a small proportion underwent caesarean section. Although hypothyroidism

has been associated with increased operative delivery in some studies the high rate of vaginal delivery observed in our study may be attributed to early diagnosis, timely initiation of levothyroxine therapy, and regular antenatal follow-up.[1,16]

Miscarriage was the most common maternal complication observed, followed by gestational diabetes mellitus, gestational hypertension, postpartum haemorrhage, and abruptio placentae. These findings are in agreement with previous studies demonstrating an increased risk of adverse obstetric outcomes among women with hypothyroidism.[9,15,16] However, the overall incidence of severe complications in the present study was relatively low, possibly reflecting effective thyroid hormone replacement and comprehensive antenatal care. Neonatal outcomes were generally favourable. NICU admission was required in 19.5% of live births, with a mean birth weight of 2.67±0.23 kg and a mean APGAR score of 8.62±1.12. Similar findings have been reported in studies evaluating adequately treated hypothyroid pregnancies suggesting that appropriate levothyroxine therapy can substantially reduce neonatal morbidity.[5,10,18]

The strengths of the present study include its prospective design, serial assessment of thyroid function throughout pregnancy, and comparison between known and newly diagnosed hypothyroid women. However, certain limitations should be acknowledged. The study was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalizability of the findings. In addition, long-term neurodevelopmental outcomes of the offspring were not evaluated. Larger multicentre studies with longer follow-up are recommended to further validate these findings.

Overall, the present study demonstrates that early diagnosis of hypothyroidism, individualized levothyroxine therapy, regular dose adjustment, and close antenatal surveillance contribute to improved thyroid function and favourable maternal and neonatal outcomes. These findings support the importance of timely screening and evidence-based management of hypothyroidism during pregnancy.

## CONCLUSION

The present study demonstrated that early diagnosis of hypothyroidism during the first trimester, prompt initiation or optimization of levothyroxine therapy, and regular monitoring of thyroid function resulted in significant improvement in thyroid hormone profile throughout pregnancy. Women with overt hypothyroidism had persistently higher TSH levels and lower free T3 and free T4 levels than those with subclinical hypothyroidism, highlighting the need for closer surveillance and more frequent dose adjustments in this group. Although miscarriage remained the most common maternal complication, the majority of women achieved favourable pregnancy outcomes, with normal vaginal delivery in most cases and satisfactory neonatal outcomes. The low incidence of severe maternal and neonatal complications observed in this study suggests that appropriate levothyroxine therapy, combined with regular antenatal follow-up, contributes to improved maternal and fetal health.

Early antenatal screening for thyroid dysfunction, individualized levothyroxine dose adjustment, and periodic assessment of thyroid function should be incorporated into routine antenatal care to optimize pregnancy outcomes in women with hypothyroidism. Further multicentric studies with larger sample sizes and long-term follow-up are recommended to validate these findings and evaluate the long-term neurodevelopmental outcomes of offspring.

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