International Journal of Contemporary Pediatrics
Shanmuga PS et al. Int J Contemp Pediatr. 2026 Feb;13(2):220-225
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20260092

Original Research Article

A study of maternal hypothyroidism on neonatal thyroid profile and outcome

Shanmuga Priya S.*, Ilangumaran L., Saravanan S., Manivannan D.

Department of Paediatrics, Government Medical College and Hospital, Cuddalore, Chidambaram, Tamil Nadu, India

Received: 14 November 2025
Revised: 12 December 2025
Accepted: 09 January 2026

***Correspondence:**
Dr. Shanmuga Priya S.,
E-mail: priyafrancis1993@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Maternal hypothyroidism is one of the most common endocrine disorders during pregnancy and is associated with adverse fetal and neonatal outcomes. Early detection and management are critical, as thyroid hormone deficiency during intrauterine life may lead to irreversible neurodevelopmental impairment.
Methods: This hospital-based observational study was conducted at Government Medical College, Cuddalore, from April 2023 to October 2024. A total of 100 neonates born to mothers diagnosed with hypothyroidism were enrolled. Maternal thyroid profiles were reviewed antenatally. Neonatal thyroid function tests, including serum TSH and free T4, were performed at 72 hours of life. Abnormal results were re-evaluated after two weeks. Data were analysed using STATA version 16 and Microsoft excel, employing descriptive statistics.
Results: Among the neonates studied, 7.07% exhibited elevated TSH levels (>10 mIU/l), and 2% required levothyroxine therapy. Preterm birth was observed in 35% of cases, and 29% required NICU admission. Low birth weight (LBW) and jaundice were the most common clinical findings. Congenital anomalies were rare, with only one case of bilateral congenital talipes equinovarus reported.
Conclusions: Maternal hypothyroidism has a measurable impact on neonatal thyroid function and early neonatal outcomes. Universal antenatal thyroid screening and targeted neonatal follow-up are essential to identify transient or delayed-onset hypothyroidism and prevent long-term neurodevelopmental sequelae.

Keywords: Maternal hypothyroidism, Neonatal thyroid profile, Congenital hypothyroidism, TSH, Free T4

INTRODUCTION

Thyroid dysfunction is the second most common endocrine disorder among women of reproductive age (15-45 years) and represents the most common congenital endocrine disorder worldwide.[1] In the Indian subcontinent, the prevalence of hypothyroidism during pregnancy has been reported to be approximately 14%.[2]

As this condition is largely preventable, early identification and appropriate management constitute the cornerstone of care, particularly because maternal hypothyroidism is an important and preventable cause of intellectual disability among infants.[3]

Several studies have demonstrated a significant correlation between maternal and fetal thyroid hormone levels, supporting the view that maternal hypothyroidism acts as a major risk factor for the development of congenital thyroid dysfunction in newborns.[4] During early gestation, fetal development is critically dependent on maternal thyroid hormones, which play a pivotal role in optimal neurological maturation.[5]

Any deficiency of thyroid hormones during this crucial period may result in irreversible fetal damage. Previous studies have shown a significant association between maternal hypothyroidism and adverse neurodevelopmental outcomes in children, including

lower intelligence quotient (IQ) scores, delayed psychosocial and motor development, and an increased risk of neurodevelopmental disorders such as attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder.[6] In addition, adverse pregnancy and neonatal outcomes such as miscarriage, congenital anomalies, congenital heart defects, LBW, hyperbilirubinemia, neonatal hypothyroidism, and neonatal hyperthyroidism have been reported at higher frequencies among offspring of hypothyroid mothers.[7]

Aim and objectives

Aim was to assess the effect of maternal hypothyroidism on neonatal thyroid function.

Objectives were to evaluate neonatal thyroid function in infants born to hypothyroid mothers and to correlate maternal thyroid status with neonatal outcomes.

METHODS

Thyroid hormones are essential for fetal neurodevelopment, particularly during the first trimester, when the fetal thyroid gland is not yet functional. During this period, maternal thyroxine (T4) serves as the primary source of thyroid hormone for fetal brain development.[8] Pregnancy induces significant physiological changes in thyroid function due to increased estrogen and β-human chorionic gonadotropin (β-hCG) levels, resulting in elevated thyroxine-binding globulin concentrations and suppression of thyroid-stimulating hormone (TSH), respectively.[9]

In India, maternal hypothyroidism has a reported prevalence ranging from 11% to 14%, with subclinical hypothyroidism being more common than overt disease. Iodine deficiency and autoimmune thyroiditis are the predominant etiological factors.[2,10] Untreated maternal hypothyroidism has been consistently associated with poor cognitive outcomes in offspring, including reduced IQ scores and delayed psychomotor development.[6]

Congenital hypothyroidism affects approximately 1 in 1130 Indian neonates, as reported by a large multicentric study conducted by the Indian council of medical research (ICMR). A substantial proportion of affected infants remain undiagnosed in the absence of universal newborn screening.[11] The American academy of pediatrics (AAP) and Indian academy of pediatrics (IAP) recommend neonatal screening using serum TSH and/or free T4 levels within 48-72 hours after birth to facilitate early diagnosis and prompt intervention.[12]

Maternal autoimmune thyroid disease may lead to the transplacental passage of TSH receptor–blocking antibodies, contributing to transient or permanent neonatal hypothyroidism.[13] Early initiation of levothyroxine therapy in affected neonates has been shown to prevent irreversible neurological impairment. International clinical guidelines recommend initiating treatment when TSH exceeds 10 mIU/l or free T4 levels are low, even in asymptomatic neonates.[14]

These findings underscore the importance of routine thyroid monitoring during pregnancy and mandatory neonatal thyroid screening in infants born to mothers with hypothyroidism.

Study design and setting

This hospital-based observational study was conducted in the Neonatology Unit of the Government Medical College and Hospital, Cuddalore District, Tamil Nadu, India. The study focused on neonates born to mothers diagnosed with thyroid dysfunction, with particular emphasis on the maternal hypothyroidism during pregnancy.

Study period and population

The study was carried out over a period of 18 months, from April 2023 to October 2024. The study population comprised neonates born to mothers with a documented diagnosis of thyroid dysfunction who delivered at the study institution during the specified period.

Inclusion and exclusion criteria

Neonates born to mothers with a confirmed diagnosis of thyroid dysfunction during pregnancy and who were delivered within the hospital premises during the study period were included in the study.

Neonates with known or suspected chromosomal abnormalities or major structural congenital anomalies, such as Down syndrome, trisomy 18, neural tube defects, or other major congenital malformations, were excluded from the study.

Sample size calculation

The sample size was calculated based on the expected proportion of neonatal thyroid dysfunction among infants born to hypothyroid mothers. Previous studies have reported the prevalence of abnormal neonatal thyroid function in this group to be approximately 7%. Using the formula for estimation of a single proportion:

$n=Z^2\times p\times qd^2n=\frac{Z^2\times p\times q}{d^2}$

$n=d^2Z^2\times p\times q$

Where n is the sample size, *Z* is the standard normal variate at 95% confidence level (1.96), p is the expected prevalence (0.07), q=1-p, and d is the allowable error set at 5%, the minimum required sample size was calculated to be approximately 100 neonates. Accordingly, a sample size of 100 neonates was included in the study.[1]

Sampling technique

A consecutive sampling technique was employed. All eligible neonates meeting the inclusion criteria during the study period were enrolled consecutively, without randomization, until the desired sample size was achieved.

Study procedure

The study procedure was carried out in two phases: maternal data collection and neonatal evaluation.

Maternal data were obtained from antenatal and hospital records and included maternal age, obstetric history, thyroid function test results (serum TSH, free T4, and anti-thyroid peroxidase antibodies where available), timing of diagnosis of thyroid dysfunction, and treatment details such as levothyroxine dosage and treatment compliance.

Neonatal thyroid screening was performed at 72 hours of life, in accordance with universal neonatal screening protocols. Blood samples were collected and analysed for serum thyroid-stimulating hormone (TSH) and free thyroxine (free T4) levels using chemiluminescent immunoassay (CLIA) methods.

Neonates with serum TSH levels greater than 10 mIU/L or free T4 levels below 1.1 ng/mL were scheduled for repeat thyroid function testing after two weeks to confirm persistent thyroid dysfunction. Appropriate treatment was initiated for neonates with confirmed abnormalities, as per standard clinical guidelines. Follow-up was carried out until discharge from the hospital or in-hospital death.

Outcome measures

The primary outcome measure was the proportion of neonates with abnormal thyroid function test results. Secondary outcomes included the correlation between neonatal thyroid status and maternal thyroid profile, timing of diagnosis, and adequacy of maternal treatment.

Statistical analysis

Data were entered and managed using Microsoft excel. Statistical analysis was performed using SPSS software version 26. Descriptive statistics were used to summarise the data. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as means with standard deviations.

Associations between maternal and neonatal thyroid parameters were analysed using the Chi-square test or Fisher's exact test for categorical variables, and the independent t test or Mann Whitney U test for continuous variables, depending on the distribution of data. A p value of less than 0.05 was considered statistically significant.

RESULTS

Among the 100 neonates studied, most mothers were aged 25-30 years (46%), and multigravida women constituted 58% of the cohort. Subclinical hypothyroidism was the predominant maternal condition (64%), with nearly half of the cases diagnosed during the first trimester. All mothers received levothyroxine therapy, and 78% achieved adequate thyroid control.

Preterm births accounted for 35% of deliveries, and 29% of neonates had LBW. There was a near-equal sex distribution. Vaginal delivery was the most common mode (61%). NICU admission was required for 29% of neonates, reflecting increased neonatal vulnerability in this high-risk group.

Neonatal treatment and clinical management

The majority of neonates in the study, 73 cases (73%), did not require any specific treatment and were managed with routine neonatal care. LBW specific care was provided to 14 neonates (14%), indicating that LBW was a relatively common clinical concern in this cohort.

Phototherapy was required in 5 neonates (5%) for the management of neonatal jaundice. A smaller proportion of neonates required more intensive interventions: 4 neonates (4%) received a combination of oxygen support and intravenous antibiotics, suggestive of respiratory distress or suspected sepsis, while 2 neonates (2%) were treated with intravenous antibiotics alone.

Eltroxin (levothyroxine) therapy was initiated in 2 neonates (2.02%), reflecting confirmed or persistent abnormalities in thyroid function consistent with neonatal hypothyroidism.

Congenital anomalies

Congenital anomalies were uncommon in the study population. Ninety-nine neonates (99.0%) did not exhibit any congenital malformations. Only one neonate (1.0%) was diagnosed with bilateral congenital talipes equinovarus (CTEV). Overall, the findings indicate a very low incidence of congenital anomalies among neonates born to mothers with hypothyroidism.

Neonatal free T3 profile

Abnormal free triiodothyronine (Free T3) levels were observed in 7 neonates. The recorded Free T3 values were 0.4, 0.45, 0.6, 0.8, 0.9, 1.1, and 1.32 pg/mL, with each value observed in a single neonate.

Given the small subgroup size, each individual value accounted for 14.29% of this subset. The observed Free T3 levels ranged from a minimum of 0.4 pg/mL to a maximum of 1.32 pg/mL.

Neonatal TSH levels and follow-up

In the present study, 7.07% of neonates were found to have serum TSH levels greater than 10 mIU/l.

Although some degree of TSH elevation in the early neonatal period may reflect a physiological postnatal surge, elevated TSH values in neonates born to hypothyroid mothers necessitate careful monitoring.

The distribution of TSH values in this cohort was right-skewed, indicating that a small number of neonates exhibited markedly elevated levels. Follow-up testing revealed a mean TSH value of 12.2 mIU/l, suggesting the possibility of a delayed rise in TSH in certain infants. This pattern is particularly relevant in neonates with risk factors such as maternal thyroid dysfunction, LBW, or prematurity, and underscores importance of longitudinal thyroid function monitoring in this high-risk group

Table 1: Sociodemographic characteristics of the study population.

Variables	Category	N	Percent (%)
Maternal age (in years)	<25	28	28.0
	25-30	46	46.0
	>30	26	26.0
Parity	Primigravida	42	42.0
	Multigravida	58	58.0
Type of maternal thyroid disorder	Subclinical hypothyroidism	64	64.0
	Overt hypothyroidism	36	36.0
Timing of diagnosis	Pre-pregnancy	21	21.0
	First trimester	49	49.0
	Second/third trimester	30	30.0
Maternal treatment status	On levothyroxine	100	100.0
	Adequately controlled	78	78.0
	Inadequately controlled	22	22.0
Gestational age at birth	Preterm (<37 weeks)	35	35.0
	Term (≥37 weeks)	65	65.0
Neonatal sex	Male	52	52.0
	Female	48	48.0
Birth weight	<2.5 kg (LBW)	29	29.0
	≥2.5 kg	71	71.0
Mode of delivery	Vaginal	61	61.0
	Caesarean section	39	39.0
NICU admission	Yes	29	29.0
	No	71	71.0

Table 2: Distribution of neonatal clinical features.

Neonatal clinical features	N	Percent (%)
Birth asphyxia	3	3.0
ELBW	1	1.0
IUGR	2	2.0
IUGR/LBW	1	1.0
Jaundice	2	2.0
LBW	11	11
LBW/IUGR	5	5
Lethargy, refusal of feeds	1	1.0
Nil	66	66
Perinatal depression	1	1.0
Prolonged jaundice	3	3.0
Refusal of feeds	3	3.0
Respiratory distress	1	1.0
Total	100	100.0

Table 3: Distribution of the neonates as per congenital anomalies.

Variables	N	Percent (%)
B/L CTEV	1	1.0
No congential anomalies	99	99
Total	100	100.0

Table 4: Distribution of neonates as per free T3 levels.

Free T3 levels	N	Percent (%)
0.4	1	14.29
0.45	1	14.29
0.6	1	14.29
0.8	1	14.29
0.9	1	14.29
1.1	1	14.29
1.32	1	14.29
Total	7	100.0

Table 5: Distribution of the neonates as per the TSH levels.

TSH	N	Percent (%)
22.79	1	14.29
28.5	1	14.29
5	1	14.29
6.7	1	14.29
7	1	14.29
7.7	1	14.29
7.8	1	14.29
Total	7	100.0

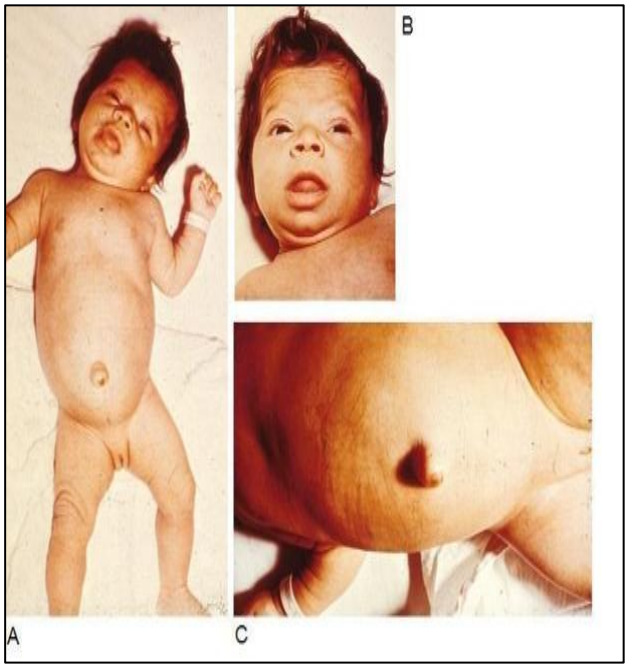

Figure 1 (A-C): Infant with untreated congenital hypothyroidism.

DISCUSSION

Most neonates (66.7%) did not display any overt clinical features suggestive of hypothyroidism or other complications. However, a considerable proportion presented with LBW or intrauterine growth restriction (IUGR), reinforcing the established association between maternal hypothyroidism and impaired fetal growth.[10] A smaller subset exhibited symptoms such as jaundice (5%), feeding difficulties, lethargy, or respiratory distress—clinical features that are largely non-specific but may be indirectly influenced by thyroid hormone deficiency during intrauterine life.[11]

Neonatal intensive care unit (NICU) admission was required in 29% of neonates, which is higher than the general neonatal admission rate and may be attributed to the combined effects of prematurity, LBW, or other perinatal complications associated with maternal hypothyroid status. Only one neonate presented with a congenital anomaly (bilateral congenital talipes equinovarus), and although no strong evidence directly links this anomaly with maternal thyroid dysfunction, thyroid hormones are known to play an essential role in fetal skeletal and neural development.[12]

Approximately 6% of neonates required regular follow-up, which may indicate a risk of delayed-onset infantile hypothyroidism among offspring of hypothyroid mothers. Follow-up evaluation revealed a mean serum TSH level of 13.2 mIU/l, higher than baseline values, with a mean T3 level of 0.8 pg/ml, suggesting the possibility of progressive or persistent thyroid dysfunction in a subset of infants.

These findings underscore the importance of longitudinal monitoring in this high-risk group, as early detection and timely initiation of treatment for congenital hypothyroidism have been shown to significantly improve long-term neurodevelopmental outcomes.[13]

Limitations

This was a single-centre, hospital-based observational study, which may limit the generalisability of the findings. The relatively small sample size restricted the ability to detect rare neonatal outcomes. Absence of a control group of neonates born to euthyroid mothers limited direct risk comparison. Variations in timing and adequacy of maternal treatment could not be fully standardised. Neonatal follow-up was confined to the early postnatal period, precluding assessment of long-term neurodevelopmental outcomes and delayed-onset hypothyroidism.

CONCLUSION

This study highlights the significant clinical and biochemical impact of maternal hypothyroidism on neonatal thyroid function. While the majority of neonates appeared clinically stable, a noteworthy proportion exhibited altered thyroid hormone profiles, especially elevated TSH levels suggestive of subclinical or transient hypothyroidism. A small but important subset required levothyroxine therapy, and many others showed associated conditions such as LBW, feeding difficulties, or required NICU care.

The findings underscore the importance of vigilant antenatal screening for thyroid dysfunction in pregnant women and suggest that even well-managed maternal hypothyroidism may pose risks to the foetus and neonate. Neonatal thyroid screening, particularly in high-risk groups, should be comprehensive and, where necessary, include follow-up assessments to detect delayed TSH elevation or evolving hypothyroidism.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Glinoer D. The regulation of thyroid function during normal pregnancy. Endocr Rev. 1997;18(3):404-33.
2. 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. 2017;27(3):315-89.
3. Dhanwal DK, Bajaj S, Rajput R, Subramaniam KA, Chowdhury S, Bhadada SK. Prevalence of hypothyroidism in pregnancy: An Indian perspective. Indian J Endocrinol Metab. 2013;17(2):281-4.
4. Haddow JE, Palomaki GE, Allan WC, Williams JR, Knight GJ, Gagnon J, et al. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. N Engl J Med. 1999;341(8):549-55.
5. Indian Council of Medical Research. Final report on congenital hypothyroidism screening in India. New Delhi: ICMR. 2012.
6. Rose SR, Brown RS, Foley T, Kaplowitz PB, Kaye CI, Sundararajan S, et al. Update of newborn screening and therapy for congenital hypothyroidism. Pediatrics. 2006;117(6):2290-303.
7. Dhanwal DK, Prasad S, Agarwal AK, Dixit V, Banerjee AK. High prevalence of hypothyroidism during pregnancy in North India. Indian J Endocrinol Metab. 2013;17(2):281-4.
8. Olivieri A, Stazi MA, Mastroiacovo P, Fazzini C, Medda E, Spagnolo A, et al. A population-based study on the frequency of congenital hypothyroidism in Italy. Horm Res. 2002;57(2):18-21.
9. Léger J, Olivieri A, Donaldson M, Torresani T, Krude H, van Vliet G, et al. European Society for Paediatric Endocrinology consensus guidelines on screening, diagnosis, and management of congenital hypothyroidism. Horm Res Paediatr. 2014;81(2):80-103.
10. Hering NA, Fromm M, Schulzke JD. Determinants of intestinal barrier function in health and disease. J Infect Dis. 2016;214(1):S2-6.
11. American Thyroid Association. Guidelines for the diagnosis and management of thyroid disease during pregnancy and postpartum. Thyroid. 2017;27(3):315-89.
12. Indian Council of Medical Research. Study report on congenital hypothyroidism screening. New Delhi: ICMR. 2012.
13. American Academy of Pediatrics. Recommendations for screening and management of congenital hypothyroidism. Pediatrics. 2006;117(6):2290-303.
14. European Society for Paediatric Endocrinology. ESPE clinical practice guidelines for congenital hypothyroidism. Horm Res Paediatr. 2014;81(2):80-103.

Cite this article as: Shanmuga PS, Ilangumaran L, Saravanan S, Manivannan D. A study of maternal hypothyroidism on neonatal thyroid profile and outcome. Int J Contemp Pediatr 2026;13:220-5.