RESEARCH PAPER

Prevalence and Complications of Subclinical Hypothyroidism during Pregnancy at a Tertiary Care Center: A Cross-Sectional Study

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ABSTRACT

Background: Thyroid hormones are essential for maintaining maternal metabolism and fetal neurodevelopment. Subclinical hypothyroidism (SCH) is defined as elevated thyroid-stimulating hormone (TSH) level with normal free thyroxine (fT4) level, based on trimester-specific reference ranges. SCH has been linked to adverse maternal and neonatal outcomes, but its prevalence and complication profile vary across populations, particularly in different iodine-variable regions.

Objective: To find out the prevalence of subclinical hypothyroidism among pregnant women in a tertiary care center and also to analyse the complications of pregnancy associated with subclinical hypothyroidism.

Methods: A cross-sectional study was conducted at KLE's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka from July 2024 to June 2025. A total of 600 pregnant women (200 in each trimester) were enrolled by simple random sampling. Serum Thyroid Stimulating Hormone and fT4 levels were measured using electro-chemiluminescence immunoassay (ECLIA) methods. Thyroid function was assessed using trimester-specific reference ranges based on the 2017 American Thyroid Association (ATA) guidelines. Participants were categorized as Euthyroid or Subclinical Hypothyroidism. Maternal and neonatal outcomes were assessed. Associations were assessed using Chi-square or Fisher's exact tests, and relative risks (RR) with 95% confidence intervals (CI) were calculated. A p-value of <0.05 was considered statistically significant.

Results: The overall prevalence of SCH was 11.5%. The trimester-wise prevalence was 5.5%, 6.0%, and 23.0% in the first, second, and third trimesters respectively. SCH showed significant associations with anemia (RR=2.02, 95%CI:1.43–2.84, p=<0.001), pregnancy-induced hypertension (RR=2.73, 95%CI: 1.44–5.18, p=0.002), preterm delivery (RR=2.13, 95%CI: 1.22–3.73, p=0.008), low Apgar score (RR=5.13, 95% CI: 1.88–13.98, p=0.001), low birth weight (RR=1.78, 95% CI: 1.16–2.75, p=0.009), and NICU admission (RR=3.01, 95% CI: 1.45–6.24, p=0.003). No significant association was observed with gestational diabetes mellitus or mode of delivery.

Conclusion: Subclinical hypothyroidism showed an increasing prevalence in later gestation and was significantly associated with adverse maternal and neonatal outcomes. These findings highlight the importance of early screening and management of subclinical hypothyroidism in pregnancy.

Keywords: Pregnancy outcome, Prevalence, Subclinical Hypothyroidism, Thyroid Disease

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INTRODUCTION

Thyroid hormones play a crucial role in the regulation of metabolism, growth, and neurodevelopment in both the mother and the developing fetus. During pregnancy, physiological adaptations in thyroid function occur to meet increased metabolic demands and to ensure adequate thyroxine transfer to the fetus [1]. Human chorionic gonadotropin (hCG) further stimulates the thyroid gland during the first trimester, suppressing TSH and increasing

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the metabolic demands placed on maternal thyroid tissue [2]. As a result, the maternal thyroid gland must increase hormone production by 30–50% to maintain euthyroidism throughout gestation [3].

Subclinical hypothyroidism (SCH), characterized by elevated serum TSH with normal free thyroxine (fT4), represents a condition that is often clinically silent but associated with multiple adverse maternal and fetal outcomes [4]. Although overt hypothyroidism is widely recognized as hazardous during pregnancy, evidence increasingly demonstrates that even SCH can contribute to complications such as anemia, gestational hypertension, placental insufficiency, miscarriage, and preterm delivery [5,6]. The global prevalence of hypothyroidism during pregnancy ranges between 1.5% and 4%, with SCH forming the majority of cases [7]. In India, the prevalence is highly variable, ranging from 1.2% to 67% due to differences in iodine sufficiency, dietary practices, BMI, and autoimmune thyroiditis [8]. Several studies across India have highlighted persistent iodine deficiency disorders despite the nationwide salt iodization program, contributing to thyroid dysfunction in pregnancy [9].

Thyroid hormones are critical for fetal neurological development, particularly during the first half of gestation, when the fetal thyroid gland is immature and dependent on maternal hormone supply [10]. Mild maternal thyroid dysfunction may disrupt neuronal migration, cortical layering, synaptogenesis, and myelination, leading to short- and long-term neurodevelopmental deficits [11,12]. Although the American Thyroid Association (ATA) recommends targeted screening for thyroid dysfunction [13], Indian data suggest that such an approach may miss 40–60% of SCH cases, encouraging a shift toward universal screening programs [14]. However, trimester-wise data on maternal and neonatal outcomes remain limited in Indian populations. In this context, the present study was undertaken to evaluate the prevalence of SCH and associated maternal and neonatal complications among pregnant women in a tertiary care hospital in Belagavi, Karnataka.

METHODS

Study Design and setting

This cross-sectional study was conducted in the Department of Obstetrics and Gynaecology, KLE's Dr. Prabhakar Kore Hospital, Belagavi from July 2024 to June 2025. Ethical clearance was obtained from the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi, Karnataka, India (Ref No: MDC/JNMCIEC/266, dated 26/04/2024). This study involved two phases, including a cross-sectional evaluation to assess the prevalence of subclinical hypothyroidism across different trimesters, followed by assessment of maternal and neonatal outcomes at the time of delivery. All pregnant women attending the antenatal clinic during the study period, irrespective of trimester, were eligible for inclusion. Women were enrolled only after providing written informed consent in a language they understood. Pregnant women with known thyroid disease, pre-gestational diabetes mellitus, chronic hypertension, or other chronic medical conditions such as autoimmune disorders, renal disease, hepatic dysfunction, or malignancy were excluded. Pregnancies with multiple gestations (twins or triplets), those with a history of miscarriage or stillbirth, and women who did not consent to participate were also excluded from the study.

Sample Size and Sampling

Based on a previously reported SCH prevalence of 37.69% [25], sample size was calculated using the formula $n = \frac{Z^2 \times p \times q}{(1-2)/(d\times)^2}$, where Z = 1.96, p = 0.38, q= 0.635 and d denotes the relative precision set at 20%. The minimum required sample size was 167 participants per trimester. To accommodate possible attrition, 200 pregnant women were included per trimester, resulting in a total sample size of 600 participants. Simple random sampling ensured unbiased selection.

Data Collection

Sociodemographic data, obstetric history, and clinical findings were recorded. Maternal outcomes included anemia, gestational hypertension, gestational diabetes mellitus(GDM), preterm delivery and mode of delivery. Neonatal outcomes included birth weight, Apgar scores, and NICU admissions recorded.

Analysis of Samples

Venous blood (3 mL) was collected from pregnant women, centrifuged at 3000 rpm for 10 minutes to separate serum, and serum analyzed using electro-chemiluminescence immunoassay (ECLIA) for TSH and fT4. SCH was diagnosed as per ATA guidelines. Maternal and neonatal outcomes were recorded from antenatal and delivery records.

Statistical Analysis

Data were analyzed using R software v4.4.3. Categorical variables were expressed as frequency and percentages. The continuous variable, age was summarized using Median and quartiles(Q_1,Q_3) as the data violated the assumption of normality. Normality was assessed using the Kolmogorov–Smirnov test. The prevalence of subclinical hypothyroidism among pregnant women was estimated along with 95% confidence intervals. Associations between subclinical hypothyroidism and maternal and neonatal outcomes were assessed using the Chi-square test or Fisher's exact test, as appropriate. Relative risks (RR) with 95% confidence intervals were reported. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 600 pregnant women were enrolled in this cross-sectional study. They were distributed evenly across the three trimesters - 200 each in the first, second, and third trimesters. The median age of the participants was 25 years [Q_1,Q_3:23,28]. Of these, 69 women (11.5%) were diagnosed with Subclinical Hypothyroidism (SCH), and 531 (88.5%) were Euthyroid. Prevalence increased across trimesters 5.5%, 6%, and 23% respectively. This observation is depicted in **(Table 1).**

Table 1: Trimester-wise prevalence of subclinical hypothyroidism

Trimester	Total (n)	SCH (n)	Prevalence (%)	95% CI
First	200	11	5.5	2.8 – 9.5
Second	200	12	6.0	3.2 – 10.1
Third	200	46	23.0	17.2 – 29.6
Total	**600**	**69**	**11.5**	**9.1 – 14.4**

Trimester-Wise Maternal and Neonatal Outcomes:

In the first trimester, SCH was significantly associated with Anemia (RR=2.70;95% CI:1.35-5.39, p=0.001) and pregnancy-induced hypertension (PIH) (RR 6.29;95% CI:2.37,16.47, p<0.001). Other complications including GDM, preterm delivery, low birth weight (LBW), NICU admission were more frequent in SCH women but statistically insignificant due to small sample size. Cesarean delivery rate was also higher (72.7%) among SCH women. During second trimester, anemia was significantly higher among SCH women compared with euthyroid women (33.3% vs 11.2%, p=0.017). Preterm delivery (25% vs 6.9%, p=0.023) showed a significant association. Apgar score **<7** was highly significant (25% vs 2.1%, p=<0.001). SCH women showed increased risk for neonatal compromise and prematurity.

SCH was associated with a significantly higher rate of NICU admissions (RR=4.01;95% CI: 1.29- 12.56, p=0.017) in the third trimester. Other complications like anemia, PIH, GDM, preterm delivery, mode of delivery, Apgar score and LBW were more frequent but not statistically significant. The third trimester demonstrated the highest SCH prevalence (23%) and the worst neonatal outcomes, indicating progressive thyroid insufficiency affecting fetal health.

SCH prevalence and associated complications-including anemia, PIH, preterm delivery, low Apgar scores, low birth weight, and NICU admissions- remain consistently higher among SCH pregnancies across all the trimesters, with several parameters showing peaks in mid to late pregnancy. All these observations are provided in the **(Table 2).**

Table 2: Trimester-wise and overall maternal and neonatal outcomes in Subclinical Hypothyroid and Euthyroid pregnancies

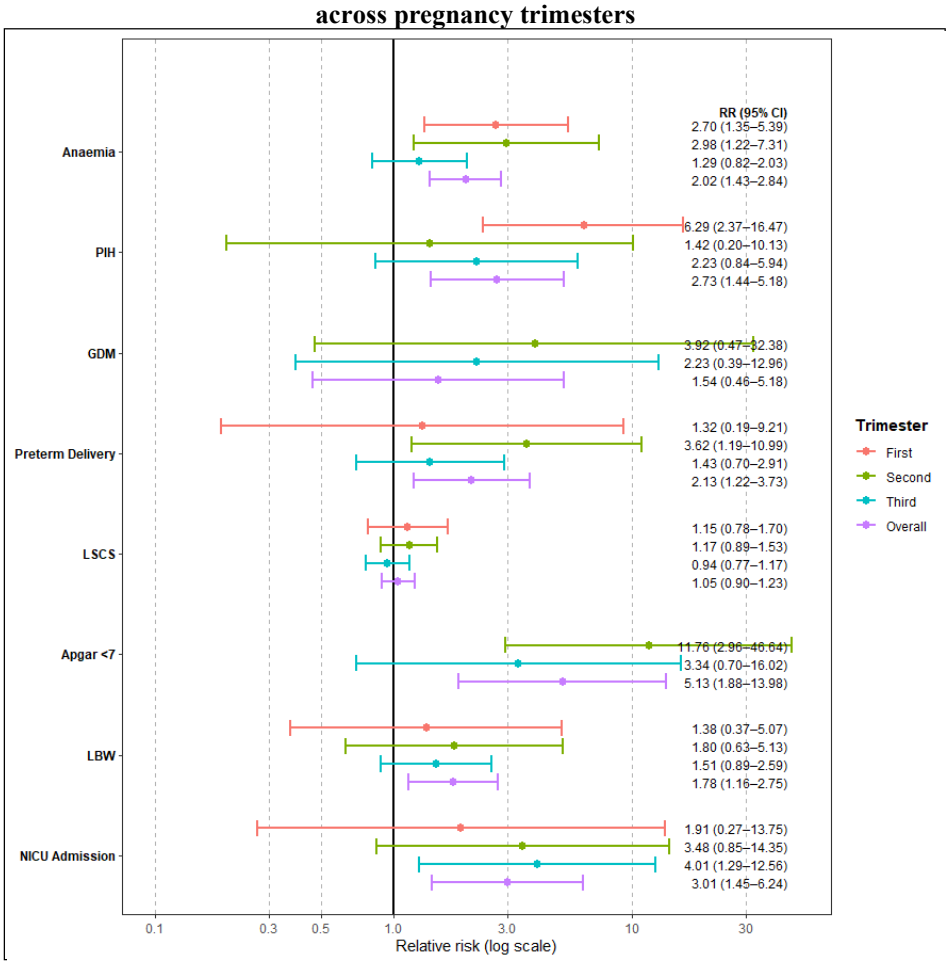
Outcome	Trimester	SCH events/total (%)	Euthyroid events/total (%)	p-value
Anaemia	First	6/11 (54.5)	38/189 (20.2)	0.001*
	Second	4/12 (33.3)	21/188 (11.2)	0.017*
	Third[$]	17/46 (37.0)	44/154 (28.6)	0.265
	Overall[$]	27/69 (39.1)	103/531 (19.4)	<0.001*
Pregnancy Induced Hypertension (PIH)	First	4/11 (36.4)	11/189 (5.8)	<0.001*
	Second	1/12 (8.3)	11/188 (5.9)	0.724
	Third	6/46 (13.0)	9/154 (5.8)	0.108
	Overall	11/69 (15.9)	31/531 (5.8)	0.002*
Gestational Diabetes Mellitus (GDM)	First	0/11 (0.0)	8/189 (4.2)	1.000
	Second	1/12 (8.3)	4/188 (2.1)	0.205
	Third	2/46 (4.3)	3/154 (1.9)	0.371
	Overall	3/69 (4.3)	15/531 (2.8)	0.486
Preterm Delivery	First	1/11 (9.1)	13/189 (6.9)	0.78
	Second	3/12 (25.0)	13/188 (6.9)	0.023
	Third[$]	9/46 (19.6)	21/154 (13.6)	0.318
	Overall[$]	13/69 (18.8)	47/531 (8.9)	0.008*
Mode of Delivery (LSCS)	First	8/11 (72.7)	119/189 (63.0)	0.46
	Second	10/12 (83.3)	134/188 (71.3)	0.255
	Third[$]	32/46 (69.6)	113/154 (73.4)	0.624
	Overall[$]	50/69 (72.5)	366/531 (68.9)	0.530
Apgar Score <7	First	0/11 (0.0)	2/189 (1.1)	1.000
	Second	3/12 (25.0)	4/188 (2.1)	<0.001*
	Third	3/46 (6.5)	3/154 (1.9)	0.130
	Overall	6/69 (8.7)	9/531 (1.7)	0.001*
Low Birth Weight (LBW)	First	2/11 (18.2)	25/189 (13.2)	0.63
	Second	3/12 (25.0)	26/188 (13.8)	0.266

	Third[$]	14/46 (30.4)	31/154 (20.1)	0.132
	Overall[$]	19/69 (27.5)	82/531 (15.4)	0.009*
NICU Admission	First	1/11 (9.1)	9/189 (4.8)	0.44
	Second	2/12 (16.7)	9/188 (4.8)	0.134
	Third	6/46 (13.0)	5/154 (3.2)	0.017*
	Overall	9/69 (13.0)	23/531 (4.3)	0.003*

*statistically significant, [$]Chi-square test otherwise Fisher's exact test was done

Maternal and Neonatal outcomes across pregnancy:
Subclinical hypothyroidism was significantly associated with several maternal and neonatal complications, including anemia (RR=2.02;95% CI: 1.43-2.84, p=<0.001), which emerged as the most common significant finding, effectively doubling the risk. The risk of pregnancy-induced hypertension was also markedly elevated (RR=2.73;95% CI:1.44-5.18, p=0.002), indicating a more than 2.7-fold increase. SCH mothers were over twice as likely to deliver preterm (RR= 2.13; 95% CI:1.22-3.73, p=0.008), and the likelihood of delivering a low-birth-weight infant was significantly higher (RR= 1.78;95% CI:1.16-2.75, p=0.009). Neonatal risks were substantial, with low Apgar scores showing the highest relative risk (RR= 5.13;95% CI:1.88-13.98, p=0.001), indicating a more than fivefold increase, and NICU admissions significantly elevated (RR= 3.01;95% CI:1.45-6.24, p=0.003), meaning infants of SCH mothers were three times more likely to require intensive care. Although rates of Caesarean section and gestational diabetes mellitus were higher among SCH women, these differences did not reach statistical significance. These results are depicted as forest plot showing the association between subclinical hypothyroidism with pregnancy outcomes in **(Figure 1).**

Figure 1: Forest plot showing the association of subclinical hypothyroidism with maternal and neonatal outcomes across pregnancy trimesters



DISCUSSION

The present study demonstrates that subclinical hypothyroidism is a relatively common thyroid disorder during pregnancy, with an overall prevalence of 11.5%. This finding is comparable to previously published national and international studies that have consistently reported elevated levels of thyroid dysfunction among pregnant women [8,14]. The sharp increase in SCH prevalence during the third trimester (23%) suggests that advancing

pregnancy places progressively greater demands on the maternal thyroid gland. This is consistent with physiological data indicating that increased estrogen levels, elevated thyroid-binding globulin, and rising fetal iodine requirements in late gestation significantly challenge maternal thyroid capacity [1,16].

Maternal complications were significantly more frequent among women with SCH. The higher incidence of anemia is consistent with the role of thyroid hormones in stimulating erythropoiesis and regulating erythropoietin production; thus, SCH leads to functional impairment of red blood cell synthesis [17]. Similarly, pregnancy-induced hypertension was significantly associated with SCH in the present study, which may result from endothelial dysfunction, reduced nitric oxide production and increased systemic vascular resistance caused by thyroid hormone deficiency [18]. These findings are consistent with previous reports by Negro et al. and Toulis et al., who also demonstrated a strong connection between SCH and hypertensive disorders [19,20].

The incidence of preterm delivery was also significantly higher among pregnancies complicated by SCH. Thyroid hormone insufficiency affects placental development and vascular remodeling, reducing uteroplacental blood flow and promoting inflammatory pathways that precipitate premature onset of labor [19]. The findings of this study indicate that untreated SCH is associated with an increased risk of preterm birth. Although Gestational Diabetes Mellitus (GDM) and Caesarean section rates were higher among women with SCH, the associations were not statistically significant. The lack of significance for GDM (RR = 1.54) may be related to the overall low prevalence of GDM in the study population, reducing the statistical power to detect differences. Additionally, SCH may have a more pronounced effect on placental angiogenesis and vascular function rather than on glucose metabolism, which could explain the weaker association with GDM.

Similarly, the Caesarean section rate was higher among SCH mothers (72.5% vs 68.9%); however, this difference did not reach statistical significance. This trend may reflect a higher frequency of obstetric interventions due to concurrent complications (such as PIH or suspected neonatal compromise), rather than a direct causal effect of SCH on mode of delivery. Neonatal outcomes were similarly affected. Low birth weight, low Apgar scores, and increased NICU admissions were more frequent among infants born to mothers with SCH. These observations are consistent with previous research demonstrating that maternal thyroid insufficiency impairs placental nutrient transport, induces oxidative stress, and compromises fetal oxygenation, leading to intrauterine growth restriction and neonatal complications [21,22]. The significantly lower Apgar scores highlight the possibility of neonatal respiratory distress associated with reduced surfactant production, poor cardiac adaptation, or fetal hypoxia [23].

The trimester-wise analysis further highlights that both the prevalence and impact of SCH tend to increase as pregnancy advances. This observation underscores the dynamic nature of thyroid physiology during gestation and the potential for subclinical dysfunction to emerge or worsen in later trimesters. Overall, the findings of the present study highlight the clinical relevance of subclinical hypothyroidism during pregnancy and its association with adverse maternal and neonatal outcomes.

This study had several strengths. It included a large sample size of 600 participants with equal representation across all three trimesters, enhancing the reliability of prevalence estimates. The trimester-wise analysis enabled the identification of changing patterns in SCH prevalence and associated risks throughout pregnancy. The use of ATA 2017 trimester-specific reference ranges ensured accurate diagnosis, while the comprehensive evaluation of both maternal and neonatal outcomes strengthened the clinical relevance of the findings. Additionally, the use of a standardized ECLIA method improved reliability of TSH and fT4 measurements. Being a single-center study, the findings may not be generalizable to a broader population. Anti-TPO antibody testing was not performed, preventing assessment of autoimmune thyroiditis as a contributing factor. Furthermore, long-term neurodevelopmental follow-up of infants was not undertaken, limiting insight into the extended consequences of maternal SCH.

CONCLUSION

Subclinical hypothyroidism is a common thyroid disorder in pregnancy, with an overall prevalence of 11.5% and a marked increase in the third trimester. The condition was significantly associated with adverse maternal outcomes such as anemia, pregnancy-induced hypertension, and preterm delivery, as well as neonatal complications such as low Apgar scores, low birth weight, and increased NICU admissions. Trimester-wise analysis showed that both prevalence and severity of outcomes tended to increase as pregnancy progressed. These findings indicate that even mild thyroid dysfunction has important clinical implications. Early detection and timely management, with consideration for repeat screening during pregnancy, may help improve maternal and neonatal outcomes.

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DECLARATIONS

Competing interests

The authors declare that they have no competing interests.

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Clinical trial number

Not applicable

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