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Original Research Article

SUBCLINICAL HYPOTHYROIDISM IN PREGNANCY AND ADVERSE OUTCOMES: A RETROSPECTIVE COHORT ANALYSIS

Juhi Gupta[1], Abhishek Sharma[2], Poonam Bhojwani[3]

[1]Associate Professor, Department of Obstetrics and Gynecology, Balvir Singh Tomar Institute of Medical Sciences and Research, Jaipur, India
[2]Professor, Department of Internal Medicine, National Institute of Medical Sciences and Research (NIMS University), Jaipur, Rajasthan, India
[3]Professor, Department of Obstetrics and Gynecology, National Institute of Medical Sciences and Research (NIMS University), Jaipur, Rajasthan, India

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Corresponding Author:
Dr. Juhi Gupta,
Associate Professor, Department of Obstetrics and Gynecology, Balvir Singh Tomar Institute of Medical Sciences and Research, Jaipur, India
Email: drjuhigoyal@gmail.com

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ABSTRACT

Background: Subclinical hypothyroidism (SCH) in pregnancy is a frequently encountered thyroid dysfunction and has been increasingly recognized for its potential impact on maternal and neonatal outcomes. Even mild elevations in thyroid-stimulating hormone (TSH) during early gestation may affect placental function, fetal development, and pregnancy progression. However, evidence regarding the extent of associated risks remains inconsistent across populations. The aim is to evaluate the association between subclinical hypothyroidism diagnosed in early pregnancy and adverse maternal and neonatal outcomes in comparison with euthyroid pregnant women.

Materials and Methods: This retrospective cohort study was conducted at a tertiary care hospital and included 115 pregnant women who underwent early antenatal thyroid screening. Based on first-trimester TSH and free thyroxine (FT4) levels, participants were categorized into a subclinical hypothyroidism group (n = 40) and a euthyroid control group (n = 75). Maternal demographic data, thyroid profiles, pregnancy complications, and neonatal outcomes were extracted from medical records. Maternal outcomes assessed included gestational hypertension, preeclampsia, preterm labor, gestational diabetes mellitus, cesarean delivery, and postpartum hemorrhage. Neonatal outcomes included low birth weight, preterm birth, small for gestational age (SGA), low Apgar scores, NICU admission, and intrauterine fetal demise.

Results: Women with SCH had significantly higher TSH levels compared to euthyroid women (5.42 ± 0.80 vs. 2.10 ± 0.50 mIU/L; $p < 0.001$), while FT4 levels were similar. Although not statistically significant, SCH was associated with higher rates of gestational hypertension (17.50% vs. 8.00%), preeclampsia (12.50% vs. 5.33%), preterm labor (20.00% vs. 12.00%), and cesarean delivery (45.00% vs. 33.33%). Neonatal complications—including low birth weight (22.50% vs. 13.33%), NICU admission (15.00% vs. 9.33%), and SGA (17.50% vs. 10.67%)—were also more frequent in the SCH group. Logistic regression revealed increased odds for both maternal (OR 1.58) and neonatal complications (OR 1.74), though not statistically significant.

Conclusion: Subclinical hypothyroidism in pregnancy demonstrated a consistent trend toward increased maternal and neonatal risks despite the lack of statistical significance, indicating the clinical importance of early thyroid screening. Larger prospective studies are warranted to validate these associations and refine management strategies.

Keywords: Subclinical hypothyroidism, Pregnancy outcomes, Thyroid function, Neonatal complications, Maternal health.

INTRODUCTION

Subclinical hypothyroidism (SCH) in pregnancy has emerged as a key area of interest in obstetric endocrinology because even mild disturbances in maternal thyroid function may have important implications for both mother and fetus. Thyroid disease is among the most common endocrine disorders encountered during pregnancy, and its recognition has increased with the wider use of early antenatal screening.[1,2] Maternal thyroid hormones play a central role in implantation, placental development, and fetal neurocognitive maturation, particularly in the first trimester when the fetus is largely dependent on transplacental transfer of maternal thyroxine.[3] As a result, even modest deviations from normal thyroid function during early gestation may contribute to adverse obstetric and neonatal outcomes, making SCH a clinically significant but often under-recognized condition. Pregnancy is associated with profound physiological changes in thyroid function, which complicate the interpretation of thyroid function tests. Human chorionic gonadotropin (hCG) exerts a weak thyrotropic effect, leading to a transient reduction in serum thyroid-stimulating hormone (TSH), particularly in the first trimester, while rising estrogen levels increase thyroxine-binding globulin and alter total thyroid hormone concentrations.[3,4] Increased renal iodine clearance and placental deiodinase activity further modify maternal thyroid homeostasis, increasing the demand for thyroid hormone synthesis.[3,4] These adaptations mean that nonpregnant reference ranges are inappropriate in pregnancy and that trimester-specific or population-specific reference intervals are recommended by major endocrine societies.[1,2] Inaccurate application of reference ranges may either miss clinically meaningful SCH or overdiagnose thyroid dysfunction, with consequences for both under- and overtreatment. Subclinical hypothyroidism is classically defined as an elevated serum TSH with normal free thyroxine (FT4) levels in the absence of overt hypothyroid symptoms.[2] SCH is considered the most frequent thyroid dysfunction in pregnancy, with reported prevalence ranging from about 3–4% to more than 10–14%, depending on the TSH cut-off used, iodine status, and population characteristics.[2,5] Indian and other Asian cohorts, especially those in iodine-insufficient or borderline areas, have reported relatively higher prevalence rates of SCH compared with Western populations, raising concerns about the burden of unrecognized thyroid dysfunction in these regions.[5] This epidemiological variability underlines the importance of local data and institution-based audits to guide policy on screening strategies and management thresholds. The potential clinical impact of SCH in pregnancy has been the subject of intense debate. Observational studies and meta-analyses have suggested associations between untreated SCH and a spectrum of adverse maternal outcomes, including gestational hypertension, preeclampsia, placental abruption, and increased cesarean delivery rates, as well as neonatal complications such as preterm birth, low birth weight, and perinatal loss.[2,5,6] In particular, a large meta-analysis reported nearly a twofold higher risk of miscarriage in women with untreated SCH compared with euthyroid pregnant women, highlighting early gestation as a critical window for thyroid status.[6] These findings have strengthened arguments in favor of more proactive detection and management of SCH; however, heterogeneity between studies in diagnostic cut-offs, timing of testing, and adjustment for confounders has limited definitive conclusions. Beyond short-term obstetric and neonatal outcomes, concern has also focused on the potential long-term neurodevelopmental consequences for offspring. Experimental and epidemiological data suggest that maternal thyroid hormone insufficiency during critical periods of fetal brain development may impair cognitive outcomes and schooling performance in later childhood.[2,3] Early reports of reduced IQ scores and neuropsychological deficits in children born to mothers with untreated hypothyroidism drew attention to even mild maternal thyroid dysfunction as a potentially modifiable risk factor.[3] However, subsequent randomized and large controlled studies have yielded more nuanced results, with some trials failing to demonstrate cognitive benefit when levothyroxine was initiated after the late first trimester, especially in SCH rather than overt hypothyroidism. These complexities are reflected in evolving international guidelines. Earlier recommendations tended to support more aggressive treatment thresholds and, in some settings, even universal screening for thyroid dysfunction in pregnancy. More recent guideline iterations emphasize the use of population- and trimester-specific TSH reference ranges, careful consideration of thyroid peroxidase antibody (TPOAb) status, and individualized decision-making regarding levothyroxine therapy, particularly for women with mild TSH elevation.[2] While most societies agree on the need to treat overt hypothyroidism, the optimal approach to SCH—especially in TPOAb-negative women with only borderline TSH elevation—remains contentious.[1] In addition, the feasibility and cost-effectiveness of universal screening versus targeted high-risk case-finding continue to be debated, particularly in low- and middle-income countries with constrained resources.

MATERIALS AND METHODS

This retrospective cohort analysis was conducted at a tertiary care hospital and involved a detailed review of antenatal records from pregnant women who underwent routine thyroid function testing during early pregnancy. All clinical data were retrieved from the hospital's electronic medical records and antenatal follow-up system. The study aimed to

evaluate the association between subclinical hypothyroidism and adverse maternal as well as neonatal outcomes. A total of 115 pregnant women were included in the study. Participants were categorized into two groups based on thyroid status at the time of first-trimester screening: women with subclinical hypothyroidism (defined as elevated thyroid-stimulating hormone [TSH] with normal free thyroxine [FT4]) and euthyroid controls. Only singleton pregnancies were considered for analysis, and all participants had complete obstetric and delivery data retrievable from institutional records.

Inclusion and Exclusion Criteria

The inclusion criteria comprised pregnant women aged 18–40 years with documented first-trimester thyroid profiles, including TSH and FT4 levels. Women with known thyroid disease prior to conception, overt hypothyroidism or hyperthyroidism, thyroid autoimmunity requiring treatment, multiple gestations, pre-existing diabetes mellitus, chronic hypertension, renal or hepatic disorders, or incomplete clinical records were excluded to minimize confounding factors.

Data Collection Procedures

Maternal demographic characteristics such as age, body mass index (BMI), parity, and previous obstetric history were extracted from clinical databases. Thyroid parameters including TSH, FT4, and, where available, anti-thyroid peroxidase (anti-TPO) antibody levels were recorded. Pregnancy-related variables including blood pressure measurements, development of gestational hypertension or preeclampsia, gestational diabetes status (based on oral glucose tolerance testing), and mode of delivery were noted. Neonatal outcomes including birth weight, Apgar scores at 1 and 5 minutes, gestational age at delivery, and NICU admission were documented. All laboratory investigations had been performed in hospital-certified laboratories using standardized assays.

Exposure and Outcome Measures

The primary exposure variable was subclinical hypothyroidism diagnosed in early pregnancy. Maternal outcomes assessed included gestational hypertension, preeclampsia, preterm labor, antepartum hemorrhage, postpartum hemorrhage, and cesarean delivery. Neonatal outcomes evaluated included low birth weight, preterm birth, small for gestational age (SGA) infants, low Apgar scores, intrauterine fetal demise, and need for neonatal intensive care unit admission. Adverse outcomes were defined according to recognized clinical criteria based on institutional protocols.

Statistical Analysis: Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality and expressed as mean ± standard deviation or median with interquartile range, as appropriate. Categorical variables were summarized using frequencies and percentages. Comparisons between groups were performed using the independent sample t-test or Mann–Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables. Multivariable logistic regression analysis was conducted to evaluate the association between subclinical hypothyroidism and adverse pregnancy outcomes while adjusting for relevant confounders. A p-value of <0.05 was considered statistically significant.

RESULTS

[Table 1] Baseline Maternal Characteristics

The baseline characteristics of the study population are presented in [Table 1]. The mean maternal age was comparable between the two groups, with women in the subclinical hypothyroidism (SCH) group having a mean age of 28.60 ± 4.10 years compared to 27.80 ± 4.30 years in the euthyroid group, and the difference was not statistically significant ($p = 0.34$). Similarly, the mean BMI showed no considerable variation, with the SCH group having a BMI of 26.40 ± 3.50 kg/m² and the euthyroid group 25.80 ± 3.30 kg/m² ($p = 0.41$). Parity distribution was also comparable between the groups, with primigravida women comprising 42.50% in the SCH group and 40.00% in the euthyroid group ($p = 0.78$). Multiparous women accounted for 57.50% and 60.00% of the respective groups, again showing no significant difference ($p = 0.78$). Although the proportion of anti-TPO positivity was higher in the SCH group (15.00%) compared to the euthyroid group (6.67%), this difference did not reach statistical significance ($p = 0.16$). These findings indicate that both groups were well matched in their baseline demographic and clinical profiles, minimizing confounding effects from maternal characteristics.

[Table 2] Thyroid Function Parameters

Thyroid function parameters showed a distinct biochemical difference between the two groups, as expected. The mean TSH level was significantly elevated in the SCH group at 5.42 ± 0.80 mIU/L compared to 2.10 ± 0.50 mIU/L in the euthyroid group ($p < 0.001$), confirming clear separation between the exposure and control groups. However, FT4 levels remained comparable across both groups, with 1.10 ± 0.18 ng/dL in the SCH group versus 1.12 ± 0.20 ng/dL in controls ($p = 0.62$), consistent with the diagnostic definition of subclinical hypothyroidism. Elevated anti-TPO antibody levels were observed more frequently in the SCH group (15.00%) than in euthyroid women (6.67%), but this trend did not reach statistical significance ($p = 0.16$). Overall, the thyroid parameter profile validates accurate classification of study participants based on early pregnancy thyroid function screening.

[Table 3] Maternal Pregnancy Outcomes

The maternal pregnancy outcomes showed higher rates of complications in the SCH group compared to the euthyroid group, although several of these differences did not achieve statistical significance. Gestational hypertension occurred in 17.50% of SCH women, compared to 8.00% of euthyroid women (p

= 0.12), suggesting a trend toward increased hypertensive disorders among SCH patients. Preeclampsia was also more frequent in the SCH group (12.50%) versus controls (5.33%), though not statistically significant (p = 0.17). Similarly, preterm labor was more common in SCH women (20.00%) than in euthyroid patients (12.00%), with no significant difference (p = 0.23). The incidence of gestational diabetes mellitus was comparable between groups (15.00% vs. 13.33%, p = 0.80). Cesarean delivery was more frequently performed in the SCH group (45.00%) compared to the euthyroid group (33.33%), but this difference also did not reach statistical significance (p = 0.21). Postpartum hemorrhage rates were low in both groups, with 7.50% in SCH and 5.33% in euthyroid women (p = 0.68). Although the results suggest a general trend toward increased maternal complications in the SCH group, statistical significance was not observed in most outcomes.

[Table 4] Neonatal Outcomes

Neonatal outcomes also varied between the two groups, with adverse events occurring more frequently among infants born to mothers with SCH. Low birth weight was recorded in 22.50% of SCH pregnancies compared to 13.33% among euthyroid women, though this difference was not statistically significant (p = 0.20). Preterm birth rates paralleled this trend, occurring in 20.00% of the SCH group versus 12.00% of the euthyroid group (p = 0.23). Small-for-gestational-age (SGA) infants were also more frequent in the SCH group (17.50%) than in controls (10.67%), but without statistical significance (p = 0.29). Apgar scores below 7 at one minute were recorded in 12.50% of SCH neonates compared to 8.00% in the euthyroid group (p = 0.44). NICU admission was required for 15.00% of SCH infants compared to 9.33% of euthyroid infants (p = 0.35). While intrauterine fetal demise (IUFD) occurred in one case (2.50%) in the SCH group and none in the euthyroid group, this difference was not statistically significant (p = 0.18). These findings collectively indicate a higher frequency of unfavorable neonatal outcomes in the SCH group, although statistical significance was not achieved.

[Table 5] Logistic Regression for Adverse Pregnancy Outcomes

Logistic regression analysis was conducted to evaluate the independent association between subclinical hypothyroidism and adverse maternal and neonatal outcomes. After adjusting for potential confounders, SCH was associated with a 1.58-fold higher odds of maternal complications; however, this association was not statistically significant (p = 0.25). Similarly, SCH increased the odds of any neonatal complication by 1.74 times, although this also did not reach statistical significance (p = 0.16). The odds of preterm birth were elevated in SCH women (OR = 1.63), but again the result was not statistically significant (p = 0.31). Low birth weight showed the strongest association, with SCH increasing the odds by 1.89 times, yet statistical significance was not achieved (p = 0.15). Overall, logistic regression indicates that while SCH trends toward increasing the risk of several adverse outcomes, these associations did not persist as statistically significant predictors in the adjusted model.

Table 1: Baseline Maternal Characteristics of the Study Population (N = 115)

Variable	Subclinical Hypothyroidism (n = 40)	Euthyroid (n = 75)	p-value
Maternal Age (years), Mean ± SD	28.60 ± 4.10	27.80 ± 4.30	0.34
BMI (kg/m²), Mean ± SD	26.40 ± 3.50	25.80 ± 3.30	0.41
Primigravida, n (%)	17 (42.50%)	30 (40.00%)	0.78
Multiparous, n (%)	23 (57.50%)	45 (60.00%)	0.78
Anti-TPO Positive, n (%)	6 (15.00%)	5 (6.67%)	0.16

Table 2: Thyroid Function Parameters

Variable	Subclinical Hypothyroidism (n = 40)	Euthyroid (n = 75)	p-value
TSH (mIU/L), Mean ± SD	5.42 ± 0.80	2.10 ± 0.50	<0.001*
FT4 (ng/dL), Mean ± SD	1.10 ± 0.18	1.12 ± 0.20	0.62
Elevated Anti-TPO (>35 IU/mL), n (%)	6 (15.00%)	5 (6.67%)	0.16

*Statistically significant.

Table 3: Maternal Pregnancy Outcomes

Maternal Outcome	Subclinical Hypothyroidism (n = 40)	Euthyroid (n = 75)	p-value
Gestational Hypertension, n (%)	7 (17.50%)	6 (8.00%)	0.12
Preeclampsia, n (%)	5 (12.50%)	4 (5.33%)	0.17
Preterm Labor (<37 weeks), n (%)	8 (20.00%)	9 (12.00%)	0.23
GDM, n (%)	6 (15.00%)	10 (13.33%)	0.80
Cesarean Delivery, n (%)	18 (45.00%)	25 (33.33%)	0.21
Postpartum Hemorrhage, n (%)	3 (7.50%)	4 (5.33%)	0.68

Table 4: Neonatal Outcomes

Neonatal Outcome	Subclinical Hypothyroidism (n = 40)	Euthyroid (n = 75)	p-value
Low Birth Weight (<2500 g), n (%)	9 (22.50%)	10 (13.33%)	0.20
Preterm Birth (<37 weeks), n (%)	8 (20.00%)	9 (12.00%)	0.23
SGA Infants, n (%)	7 (17.50%)	8 (10.67%)	0.29
Apgar Score <7 at 1 minute, n (%)	5 (12.50%)	6 (8.00%)	0.44
NICU Admission, n (%)	6 (15.00%)	7 (9.33%)	0.35
IUFD, n (%)	1 (2.50%)	0 (0.00%)	0.18

Table 5: Logistic Regression for Adverse Pregnancy Outcomes Associated with SCH

Adverse Outcome	Adjusted OR (95% CI)	p-value
Any Maternal Complication	1.58 (0.72–3.46)	0.25
Any Neonatal Complication	1.74 (0.80–3.77)	0.16
Preterm Birth	1.63 (0.63–4.16)	0.31
Low Birth Weight	1.89 (0.78–4.61)	0.15

DISCUSSION

In this retrospective cohort of 115 pregnancies, subclinical hypothyroidism (SCH) was identified in 34.78% (40/115) of women and was associated with a consistently higher—but mostly non-significant—frequency of adverse maternal and neonatal outcomes compared with euthyroid women. In contrast, large cohort data from a Chinese population by Chen et al (2014) reported a much lower prevalence of SCH of 4.63% (371/8012), though they similarly observed higher risks of gestational hypertension, premature rupture of membranes, intrauterine growth restriction and low birth weight among SCH pregnancies compared with euthyroid controls.[7] The higher SCH proportion in our study may reflect different TSH cut-offs and the tertiary-care referral setting, but the pattern of increased adverse outcomes is directionally similar to that of Chen et al (2014), who found gestational hypertension rates of 3.50% vs 1.82% and low birth weight 4.58% vs 1.89% in SCH versus euthyroid women.[7]

Baseline maternal characteristics in our cohort, including age, BMI and parity, were closely matched between SCH and euthyroid women, and anti-TPO positivity was numerically higher in the SCH group (15.00% vs 6.67%). This is in keeping with classic work by Glinoer et al (1994), who demonstrated that pregnant women with asymptomatic autoimmune thyroid disease had a substantial risk of progression to SCH during gestation and were more likely to experience mild thyroid impairment and obstetric complications compared with antibody-negative women.8 In our series, the modest excess of anti-TPO positivity among SCH pregnancies supports the concept that autoimmunity contributes to thyroid dysfunction in pregnancy, although the small sample size limits statistical demonstration of this association.

When overall maternal complications were examined using logistic regression, SCH in our study showed a non-significant trend towards higher odds of any maternal complication (adjusted OR 1.58, 95% CI 0.72–3.46), pregnancy-induced hypertension and preeclampsia, as well as slightly higher cesarean rates (45.00% vs 33.33%). These findings parallel the large FASTER trial analysis by Cleary-Goldman et al (2008), who studied 10,990 women and found that subclinical hypothyroidism defined by TSH above the 97.5th percentile was not consistently associated with pregnancy loss, gestational hypertension, preeclampsia, gestational diabetes, preterm delivery or altered birth weight when compared with euthyroid pregnancies.[9] The lack of statistical significance in our adjusted models is therefore consistent with these neutral findings, although our point estimates for several outcomes are slightly higher than those reported in FASTER, again suggesting limited power rather than absence of effect.

Hypertensive disorders of pregnancy were more frequent in our SCH group, with gestational hypertension in 17.50% vs 8.00% and preeclampsia in 12.50% vs 5.33% of euthyroid women. Wilson et al (2012) specifically evaluated subclinical thyroid disease and hypertensive disorders in pregnancy and found that SCH was associated with a higher incidence of severe preeclampsia and pregnancy-induced hypertension compared with euthyroid women, with subclinical thyroid dysfunction conferring an increased risk of hypertension and related cardiovascular complications during gestation.10 Although our p-values for gestational hypertension (p = 0.12) and preeclampsia (p = 0.17) did not reach significance, the roughly two-fold higher rates are directionally similar to the elevated odds observed by Wilson et al (2012), reinforcing a plausible link between SCH and hypertensive disorders.

Our findings also align with more recent data on diagnostic thresholds for SCH. Using ATA-based criteria, Li et al (2020) showed in 1,556 TPOAb-negative Chinese women that SCH diagnosed using the 2017 ATA upper TSH limit (4.0 mIU/L) was associated with significantly higher rates of pregnancy-induced hypertension (6.6% vs 1.9%), preeclampsia (5.3% vs 0.6%), cesarean delivery (42.1% vs 36.0%), preterm delivery (53.9% vs 47.2%) and composite maternal and neonatal complications compared with euthyroid pregnancies.[11] In our cohort, SCH women likewise exhibited higher, though non-significant, frequencies of gestational hypertension (17.50% vs 8.00%),

preeclampsia (12.50% vs 5.33%), cesarean delivery (45.00% vs 33.33%) and preterm labor (20.00% vs 12.00%), suggesting that when SCH is defined using relatively stringent early-pregnancy thresholds, there is a tendency towards multiple, modestly elevated maternal risks similar to those quantified by Li et al (2020).

Preterm birth is one of the most widely studied endpoints in SCH. In our study, preterm labor (<37 weeks) occurred in 20.00% of SCH pregnancies compared with 12.00% among euthyroid women (p = 0.23), and logistic regression showed a non-significant OR of 1.63 for preterm birth. Casey et al (2005) evaluated 404 women with SCH (TSH ≥97.5th percentile, normal FT4) and 15,689 euthyroid controls, reporting that early preterm birth ≤34 weeks occurred in 4.0% of SCH versus 2.5% of controls, with an adjusted relative risk of 1.8 (95% CI 1.1–2.9).[12] They also noted that NICU admission and respiratory distress were almost doubled in infants of SCH mothers. Our higher absolute preterm rates (20.00% and 12.00%) likely reflect inclusion of all births <37 weeks and the high-risk tertiary setting, but the relative increase in preterm delivery approximates that seen by Casey et al (2005), again suggesting that SCH may modestly increase the risk of preterm birth even if statistical significance is not consistently demonstrated in smaller samples.

Neonatal outcomes in our cohort revealed higher frequencies of low Apgar score at 1 minute (12.50% vs 8.00%), NICU admission (15.00% vs 9.33%) and low birth weight (22.50% vs 13.33%) among neonates born to SCH mothers, though these differences were not statistically significant. Lee et al (2020) examined 8,413 mother–infant pairs and found that maternal TSH >4 mIU/L during pregnancy was associated with approximately a two-fold increased risk of prematurity and respiratory distress syndrome, while showing more modest and sometimes non-significant associations with NICU admission and low Apgar scores compared with women with TSH in the reference range.[13] The pattern in our data—higher but imprecise rates of NICU admission and suboptimal Apgar scores in the SCH group—is compatible with Lee et al (2020), suggesting that even mild thyroid dysfunction may subtly affect early neonatal adaptation, particularly in settings with additional obstetric risk factors.

With respect to fetal growth, our study showed a higher frequency of low birth weight (22.50% vs 13.33%) and SGA infants (17.50% vs 10.67%) in the SCH group. Derakhshan et al (2020) conducted an individual-participant data meta-analysis across multiple cohorts and reported that higher maternal TSH and lower FT4 within the population reference range were associated with small reductions in birth weight and a modestly increased risk of SGA, although effect sizes were generally small and heterogeneous across studies.[14] Our findings of a numerically higher burden of fetal growth restriction among SCH pregnancies are consistent with this meta-analytic signal, but the lack of statistical significance in our study likely reflects limited power rather than a true absence of effect.

Thyroid autoimmunity is another relevant determinant of adverse outcomes. In our cohort, anti-TPO positivity was more frequent among SCH women (15.00% vs 6.67%), although not statistically significant. A landmark meta-analysis by Thangaratinam et al (2011) showed that thyroid autoantibodies in otherwise euthyroid women were associated with a near doubling of miscarriage risk and a significantly increased risk of preterm birth compared with antibody-negative women.[15] While our study excluded women with overt disease and had relatively small numbers of antibody-positive pregnancies, the trend towards more anti-TPO positivity in the SCH group, together with numerically higher rates of low birth weight, preterm birth and one case of IUFD (2.50% vs 0.00%), is directionally compatible with the literature suggesting that even mild thyroid dysfunction and autoimmunity may jointly contribute to adverse obstetric and perinatal outcomes.

Finally, the non-significant odds ratios for composite maternal (OR 1.58) and neonatal (OR 1.74) complications in our logistic regression need to be interpreted in the context of emerging intervention data. Jiao et al (2022), in an updated systematic review and trial sequential analysis of levothyroxine therapy for SCH in pregnancy, reported that treatment may reduce the risk of certain adverse outcomes—particularly preterm birth and pregnancy loss—in selected high-risk subgroups, although overall evidence remains heterogeneous and did not uniformly demonstrate benefit across all endpoints.[16]

CONCLUSION

Based on the findings of this retrospective cohort analysis, subclinical hypothyroidism in pregnancy was associated with higher—but mostly non-significant—rates of maternal and neonatal complications compared with euthyroid women. Although trends toward increased gestational hypertension, preeclampsia, preterm birth, low birth weight, and NICU admissions were observed, the limited sample size may have reduced the ability to detect statistical significance. Nonetheless, the consistent directional increase in adverse outcomes underscores the clinical relevance of early thyroid screening. These results highlight the need for larger, well-designed prospective studies to clarify the true impact of subclinical hypothyroidism and to guide evidence-based management strategies during pregnancy.

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