Research Article

Association Between Second-Trimester Dyslipidemia And The Risk Of Developing Preeclampsia Among Pregnant Women With Subclinical Hypothyroidism

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Abstract

Background: Subclinical hypothyroidism (SCH) is one of the most common endocrine disorders encountered during pregnancy and has been increasingly recognized as a contributor to adverse maternal and fetal outcomes.

Aim: This study aimed to evaluate the association between second-trimester dyslipidemia and the risk of developing preeclampsia among pregnant women with subclinical hypothyroidism and to identify clinically useful biochemical markers for early prediction of preeclampsia.

Patients and Methods: A hospital-based case–control study was conducted at the Department of Obstetrics and Gynecology, Tikrit Teaching Hospital, Salah Al-Din Governorate, Iraq, from 1 October 2025 to 30 June 2026. A total of 120 pregnant women diagnosed with SCH during the second trimester were enrolled. The participants were divided into two equal groups: 60 women who subsequently developed preeclampsia (cases) and 60 women who remained normotensive throughout pregnancy (controls). fasting serum samples were analyzed for thyroid-stimulating hormone (TSH), free thyroxine (FT4), total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C).

Results: Women who developed preeclampsia had significantly higher serum TSH concentrations than controls (5.92 ± 1.31 vs. 4.51 ± 0.88 mIU/L, $P < 0.001$), whereas FT4 levels did not differ significantly ($P = 0.148$). Maternal lipid profile demonstrated significantly higher concentrations of TC (228.64 ± 29.81 vs. 198.73 ± 24.16 mg/dL), TG (242.51 ± 47.36 vs. 182.45 ± 36.28 mg/dL), and LDL-C (146.82 ± 26.14 vs. 118.41 ± 22.33 mg/dL), together with significantly lower HDL-C levels (41.62 ± 6.83 vs. 50.48 ± 7.22 mg/dL) among women with preeclampsia (all $P < 0.001$). Composite dyslipidemia was present in 81.67% of cases compared with 45.00% of controls ($P < 0.001$). Serum TSH demonstrated significant positive correlations with TC ($r = 0.421$), TG ($r = 0.487$), and LDL-C ($r = 0.394$), while HDL-C showed a significant inverse correlation ($r = −0.336$).

Conclusions: Pregnant women with subclinical hypothyroidism who subsequently developed preeclampsia exhibited significantly more pronounced dyslipidemia and higher serum TSH concentrations than those who remained normotensive. Elevated triglycerides, total cholesterol, LDL-C, increased TSH, reduced HDL-C, and higher maternal BMI independently predicted the development of preeclampsia.

Introduction

Hypothyroidism is a common endocrine disorder arising from insufficient thyroid hormone action at target tissues. Depending on the level of the hypothalamic–pituitary–thyroid axis that is affected, it is classified as primary (thyroidal), secondary (pituitary), or tertiary (hypothalamic) hypothyroidism, and may be congenital, acquired, or pharmacologically induced [(1)]. Subclinical hypothyroidism (SCH) is defined biochemically by an elevated serum thyroid-stimulating hormone (TSH) with normal free thyroxine (FT4), whereas overt hypothyroidism shows elevated TSH with reduced FT4; both states are frequently encountered in adult populations and often present with nonspecific or absent symptoms, making case identification heavily reliant on laboratory testing [(2,3)]. Beyond its classical metabolic and neurocognitive effects, SCH has been linked to cardiometabolic risk, including hypertension, weight gain, insulin resistance, and atherosclerotic cardiovascular disease [(4,5)].

Dyslipidemia is a well-established metabolic signature of reduced thyroid function. Multiple population and clinical studies show progressive increases in total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) as thyroid function declines from euthyroidism to SCH and overt hypothyroidism [(6,7)]. Mechanistically, thyroid hormones regulate hepatic cholesterol homeostasis, in part by controlling LDL receptor (LDL-R) expression via sterol regulatory element–binding protein 2 (SREBP-2), thereby modulating LDL particle clearance [(8)]. Thyroid hormones also promote bile acid synthesis through induction of cholesterol 7α-hydroxylase (CYP7A1), depleting hepatic cholesterol and enhancing circulatory LDL uptake; recent human and animal data support this pathway as a contributor to thyroid hormone–mediated hypocholesterolemia [(9)]. In parallel, emerging evidence suggests TSH itself can directly suppress hepatic bile acid synthesis through the SREBP-2/HNF-4α/CYP7A1 axis, offering a thyroid hormone–independent route by which elevated TSH in primary hypothyroidism worsens lipid profiles [(10)]. High-density lipoprotein cholesterol (HDL-C) responses are variable, but alterations in cholesteryl ester transfer protein and hepatic lipase activity—both thyroid-responsive—can raise HDL-C in severe hypothyroidism while impairing HDL function; reduced hepatic lipase activity also favors remnant-like particle accumulation and atherogenic dyslipidemia [(11)].

Within SCH specifically, atherogenic changes—higher TC, LDL-C, apoB, remnant cholesterol, postprandial lipemia, and qualitative LDL alterations—are consistently observed, especially when TSH exceeds 10 mIU/L, and several studies report improvement after levothyroxine therapy, aligning with restoration of thyroid-driven lipid pathways [31–38]. Despite this, heterogeneity in HDL levels and function, variability in TSH thresholds across populations, and potential independent effects of TSH continue to complicate risk stratification and treatment decisions in SCH-associated dyslipidemia [12,13].

Pregnancy introduces additional complexity. Physiologic shifts in thyroid economy and lipid metabolism, coupled with trimester- and population-specific TSH reference ranges, can unmask SCH and amplify dyslipidemia. Because dyslipidemia and endothelial dysfunction are central to hypertensive disorders of pregnancy, there is a biologically plausible link between SCH-related lipid disturbances and preeclampsia risk. However, clinically actionable lipid and TSH thresholds for predicting adverse obstetric outcomes remain undefined, particularly in the second trimester when routine screening data are widely available [14,15]..

Aim

This study aims to evaluate the association between second-trimester dyslipidemia and the risk of developing preeclampsia among pregnant women with subclinical hypothyroidism, and to identify clinically useful biochemical thresholds for early prediction and prevention of adverse pregnancy outcomes.

Objectives

To assess the correlation between serum TSH and lipid profile markers to identify thyroid-related patterns influencing lipid metabolism in pregnancy.

Patients and Methods

Study Design

This investigation was designed as a hospital-based case–control study conducted at the Department of Obstetrics and Gynecology, Tikrit Teaching Hospital, Salah Al-Din Governorate, Iraq, over a period extending from 1 October 2025 to 30 June 2026. The biochemical characteristics of women who subsequently developed preeclampsia (case group) were compared with those of women with subclinical hypothyroidism who completed pregnancy without hypertensive complications (control group).

Study Population

A total of 120 pregnant women diagnosed with subclinical hypothyroidism during the second trimester of pregnancy were enrolled in the study.

The participants were allocated into two comparable groups:

- **Case group (n = 60):** Pregnant women with subclinical hypothyroidism who subsequently developed preeclampsia during the course of pregnancy.
- **Control group (n = 60):** Pregnant women with subclinical hypothyroidism who remained normotensive throughout pregnancy and delivered healthy term infants without maternal or fetal complications.

Controls were selected to closely match the case group with regard to maternal age, gestational age at recruitment, and parity whenever feasible in order to minimize potential confounding factors. Eligible women were recruited consecutively throughout the study period after fulfilling the predefined eligibility criteria.

Methods

Sampling Technique

A consecutive non-probability sampling technique was employed throughout the study period. All pregnant women attending the antenatal clinics and obstetric outpatient services at Tikrit Teaching Hospital who fulfilled the eligibility criteria were invited to participate. Eligible women were enrolled consecutively until the predetermined sample size of 120 participants was achieved. Women were prospectively followed from recruitment during the second trimester until delivery. Participants who developed preeclampsia constituted the case group, whereas those who remained normotensive throughout pregnancy served as controls.

Blood Sample Collection and Laboratory Investigations

Following an overnight fast of approximately 10–12 hours, venous blood samples were collected from all participants during the second trimester under strict aseptic conditions. Approximately 5–10 mL of venous blood was obtained from the antecubital vein using sterile disposable syringes.

Samples were allowed to clot at room temperature before centrifugation at 3000 rpm for 10 minutes. The separated serum was transferred into sterile Eppendorf tubes and analyzed immediately or stored at −20°C until laboratory analysis according to laboratory protocol.

Ethical Considerations

Ethical approval was obtained from the Scientific and Ethical Committee of the Iraqi Board for Medical Specializations together with the Research Ethics Committee of Tikrit Teaching Hospital before commencement of patient recruitment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and its subsequent amendments. Each participant received a detailed explanation regarding the objectives, methodology, potential benefits, and possible risks of participation before enrollment. Written informed consent was obtained from every participant.

Statistical Analysis

All collected data were entered, coded, verified, and analyzed using the Statistical Package for the Social Sciences (SPSS) software (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation (SD).

Results

Baseline Demographic Characteristics

The study showed that the baseline demographic characteristics were comparable between both study groups. The mean maternal age was 31.84 ± 4.82 years among women who developed preeclampsia compared with 30.97 ± 4.51 years among controls (P = 0.302). Likewise, the mean body mass index (BMI) was 29.18 ± 3.74 kg/m² in cases and 28.41 ± 3.58 kg/m² in controls (P = 0.247). Urban residence was reported by 68.33% of cases and 65.00% of controls (P = 0.703). Housewives constituted 78.33% and 75.00% of the two groups, respectively (P = 0.669). Educational level was similarly distributed between both groups (P = 0.721), indicating satisfactory baseline comparability.

Table 1: Baseline demographic characteristics of the study participants

Variable	Cases (n=60)	Controls (n=60)	P-value
Age (years), Mean ± SD	31.84 ± 4.82	30.97 ± 4.51	0.302
BMI (kg/m²), Mean ± SD	29.18 ± 3.74	28.41 ± 3.58	0.247
Urban residence	41 (68.33%)	39 (65.00%)	0.703
Rural residence	19 (31.67%)	21 (35.00%)	
Housewife	47 (78.33%)	45 (75.00%)	0.669
Employed	13 (21.67%)	15 (25.00%)	
Primary education	18 (30.00%)	20 (33.33%)	0.721
Secondary education	24 (40.00%)	22 (36.67%)	
University education	18 (30.00%)	18 (30.00%)	

Obstetric Characteristics

The study showed that obstetric characteristics were comparable between the two groups. Gravidity was 3.92 ± 1.41 among cases and 3.71 ± 1.36 among controls (P = 0.412). Likewise, parity (2.47 ± 1.18 vs. 2.31 ± 1.12; P = 0.445) and gestational age at recruitment (22.61 ± 2.31 vs. 22.39 ± 2.18 weeks; P = 0.587) showed no statistically significant differences. Previous miscarriage, previous preeclampsia, and family history were numerically higher among cases but did not reach statistical significance.

Table 2: Obstetric characteristics of the study participants

Variable	**SCH with Preeclampsia**	**SCH without Preeclampsia**	P-value
Gravidity, Mean ± SD	3.92 ± 1.41	3.71 ± 1.36	0.412
Parity, Mean ± SD	2.47 ± 1.18	2.31 ± 1.12	0.445

Gestational age (weeks)	22.61 ± 2.31	22.39 ± 2.18	0.587
Previous miscarriage	18 (30.00%)	14 (23.33%)	0.407
Previous preeclampsia	10 (16.67%)	5 (8.33%)	0.169
Family history of preeclampsia	15 (25.00%)	10 (16.67%)	0.257

Comparison of thyroid function tests

The study demonstrated that women who subsequently developed preeclampsia had significantly higher serum TSH concentrations than controls (5.92 ± 1.31 vs. 4.51 ± 0.88 mIU/L; P < 0.001). However, FT4 concentrations remained within the normal range in both groups and showed no statistically significant difference (P = 0.148).

Table 3: Comparison of thyroid function tests

Variable	Cases	Controls	P-value
TSH (mIU/L)	5.92 ± 1.31	4.51 ± 0.88	<0.001
FT4 (ng/dL)	1.08 ± 0.14	1.12 ± 0.16	0.148

Comparison of lipid profile parameters

The study showed significantly altered lipid metabolism among women who developed preeclampsia. Total cholesterol, triglycerides, and LDL-C were significantly higher among cases, whereas HDL-C was significantly lower than in controls (all P < 0.001).

Table 4: Comparison of lipid profile parameters

Variable	Cases	Controls	P-value
Total cholesterol (mg/dL)	228.64 ± 29.81	198.73 ± 24.16	<0.001
Triglycerides (mg/dL)	242.51 ± 47.36	182.45 ± 36.28	<0.001
LDL-C (mg/dL)	146.82 ± 26.14	118.41 ± 22.33	<0.001
HDL-C (mg/dL)	41.62 ± 6.83	50.48 ± 7.22	<0.001

Dyslipidemia Categories

The study demonstrated that dyslipidemia was considerably more common among women who developed preeclampsia. Composite dyslipidemia affected 81.67% of cases compared with 45.00% of controls (P < 0.001). Hypertriglyceridemia represented the most prevalent lipid abnormality.

Table 5: Distribution of dyslipidemia categories among the study participants

Variable	Cases	Controls	P-value
Hypercholesterolemia	41 (68.33%)	24 (40.00%)	0.002
Hypertriglyceridemia	46 (76.67%)	28 (46.67%)	<0.001
Elevated LDL-C	38 (63.33%)	22 (36.67%)	0.003
Low HDL-C	33 (55.00%)	16 (26.67%)	0.001
Composite dyslipidemia	49 (81.67%)	27 (45.00%)	<0.001

Correlation Analysis

The study showed significant positive correlations between serum TSH and total cholesterol (r = 0.421; P < 0.001), triglycerides (r = 0.487; P < 0.001), and LDL-C (r = 0.394; P < 0.001). HDL-C demonstrated a significant inverse correlation with TSH (r = −0.336; P = 0.001).

Table 6: Correlation between serum TSH concentration and lipid profile parameters

Variable	Correlation coefficient (r)	P-value
Total cholesterol	0.421	<0.001
Triglycerides	0.487	<0.001
LDL-C	0.394	<0.001
HDL-C	-0.336	0.001

Logistic Regression Analysis

The study demonstrated that elevated serum TSH was the strongest independent predictor of preeclampsia (adjusted OR = 1.52; 95% CI: 1.20–1.92; P < 0.001). Maternal BMI, total cholesterol, triglycerides, LDL-C, and HDL-C also remained independently associated with the development of preeclampsia, whereas levothyroxine therapy showed no statistically significant association (P = 0.576).

Table 7: Multivariable logistic regression analysis for predictors of preeclampsia

Variable	Adjusted OR	95% CI	P-value
BMI	1.11	1.02–1.22	0.019
TSH	1.52	1.20–1.92	<0.001
Total cholesterol	1.02	1.01–1.03	0.003
Triglycerides	1.03	1.01–1.04	<0.001
LDL-C	1.02	1.01–1.04	0.005
HDL-C	0.94	0.90–0.98	0.007
Levothyroxine therapy	0.82	0.41–1.64	0.576

Discussion

The findings of the current study agree with Sullivan [15], who reported that women with subclinical hypothyroidism experience significantly higher rates of hypertensive disorders of pregnancy than euthyroid women. Similarly, Hamblin et al. [16] concluded that elevated maternal TSH concentrations are independently associated with adverse obstetric outcomes and recommended careful biochemical monitoring throughout pregnancy. Taylor and Lazarus [17] likewise emphasized that even mild thyroid dysfunction during pregnancy should not be regarded as physiologically insignificant because elevated TSH may reflect clinically important endocrine disturbances affecting placental development.

Several systematic reviews and meta-analyses further support the present findings. Nazarpour et al. [18] demonstrated that untreated subclinical hypothyroidism significantly increased the risk of preeclampsia, pregnancy loss, preterm birth, and other adverse maternal outcomes. Likewise, Bein et al. [19] concluded that appropriate levothyroxine therapy reduced several adverse pregnancy outcomes among women with subclinical hypothyroidism, particularly when treatment was initiated early in gestation. Ding et al. [20] reported similar findings and emphasized that early biochemical screening may improve pregnancy outcomes through timely therapeutic intervention.

More recent investigations have strengthened the relationship between maternal thyroid dysfunction and hypertensive disorders of pregnancy. Hu et al. [21] demonstrated that elevated maternal TSH concentrations during early pregnancy, particularly when accompanied by thyroid autoimmunity, significantly increased the subsequent risk of gestational hypertension and preeclampsia. Likewise, Li et al. [22] employed Mendelian randomization techniques and suggested a potential causal relationship between

thyroid dysfunction and preeclampsia through pathways involving endothelial dysfunction, oxidative stress, and abnormal lipid metabolism.

The absence of a statistically significant difference in FT4 concentrations between the study groups also agrees with previous investigations. Because both groups consisted exclusively of women with subclinical hypothyroidism, maintenance of normal FT4 concentrations was anticipated. Similar findings were reported by Gietka-Czernel and Glinicki [23], who noted that FT4 often remains within trimester-specific reference intervals despite clinically meaningful elevations in TSH. Liu et al. [24] similarly demonstrated that TSH provides greater predictive value than FT4 for adverse pregnancy outcomes among women with thyroid dysfunction.

Yang et al. [25] likewise observed that improvement in maternal lipid metabolism following levothyroxine therapy occurred primarily in association with reductions in serum TSH rather than alterations in FT4 concentrations. These observations support the concept that serum TSH represents the most clinically informative biochemical marker for monitoring maternal thyroid status during pregnancy.

One of the principal findings of the present study was the demonstration of significantly altered maternal lipid metabolism among women with subclinical hypothyroidism who subsequently developed preeclampsia. Women in the case group exhibited significantly higher serum total cholesterol, triglycerides, and LDL-C concentrations together with significantly lower HDL-C concentrations compared with normotensive controls.

Pregnancy is normally associated with progressive physiological increases in circulating lipids to satisfy the metabolic requirements of the growing fetus and placenta. Maternal estrogen stimulates hepatic lipoprotein synthesis, while increasing insulin resistance during late pregnancy promotes enhanced lipolysis and hepatic triglyceride production. These physiological adaptations generally remain well regulated in uncomplicated pregnancies. However, excessive maternal hyperlipidemia may exceed physiological requirements and become pathogenic through promotion of oxidative stress, endothelial dysfunction, placental vascular injury, and systemic inflammation [11].

Among the lipid abnormalities observed in the present investigation, hypertriglyceridemia appeared particularly prominent. Elevated triglyceride-rich lipoproteins undergo oxidative modification within the maternal circulation, generating toxic lipid peroxides capable of damaging vascular endothelial cells and reducing nitric oxide bioavailability [21].

The present findings agree closely with Sharami et al. [2], who demonstrated that maternal hyperlipidemia was associated with significantly poorer maternal and neonatal outcomes. Similarly, Bashir et al. [10] concluded that hypertriglyceridemia represents one of the most clinically important metabolic abnormalities encountered during pregnancy because severe elevations substantially increase the risks of preeclampsia, pancreatitis, and adverse fetal outcomes. Formisano et al. [11] likewise emphasized that dyslipidemia contributes directly to placental dysfunction through endothelial injury, oxidative stress, and inflammatory activation.

The observed elevation in LDL-C concentrations is also biologically plausible. LDL particles undergo oxidative modification during pregnancy, generating oxidized LDL that promotes endothelial activation, macrophage recruitment, foam-cell formation, and vascular inflammation. Vekic et al. [7] highlighted the importance of small dense LDL particles in accelerating endothelial dysfunction and atherosclerotic vascular injury, mechanisms that closely resemble those observed in preeclampsia. Liu and Peng [24] similarly described thyroid dysfunction as an important contributor to impaired LDL receptor activity, resulting in reduced LDL clearance and progressive hypercholesterolemia.

Conversely, HDL-C concentrations were significantly lower among women who developed preeclampsia. HDL normally exerts antioxidant, anti-inflammatory, and endothelial-protective functions by promoting reverse cholesterol transport and reducing oxidative modification of LDL particles. Reduced HDL concentrations therefore diminish these protective mechanisms and facilitate progression of endothelial dysfunction. These findings agree with Poornima et al. [8], who identified reduced HDL-C as an important component of the dyslipidemic profile associated with preeclampsia.

The present findings are also supported by Cai et al. [26], who reported that pregnant women with hypothyroidism exhibit profound alterations in plasma lipid composition that correlate with placental dysfunction and impaired fetal development. More recently, Businge and Longo-Mbenza [27] demonstrated that iodine deficiency and subclinical hypothyroidism were strongly associated with dyslipidemia in both normotensive and preeclamptic pregnancies, suggesting that thyroid dysfunction amplifies pregnancy-related lipid abnormalities through impaired thyroid hormone synthesis, altered hepatic lipid metabolism, and endothelial dysfunction.

Furthermore, Dong et al. [28] demonstrated that combining second-trimester biochemical markers significantly improved prediction of preeclampsia, supporting the integrated prediction model proposed in the present study.

More recent studies have also emphasized that maternal cardiovascular risk in pregnancy reflects the combined influence of endocrine, metabolic, and vascular abnormalities rather than thyroid dysfunction alone. Comprehensive cardiovascular risk assessment incorporating thyroid biomarkers, lipid abnormalities, obesity, and inflammatory mediators may therefore provide greater predictive accuracy than individual biomarkers alone [29].

Conclusions

Pregnant women with subclinical hypothyroidism who subsequently developed preeclampsia had significantly higher serum TSH concentrations than women who remained normotensive, while FT4 concentrations remained within the normal range and did not differ significantly between the study groups.Maternal dyslipidemia was strongly associated with the subsequent development of preeclampsia. Women who developed preeclampsia exhibited significantly higher serum total cholesterol, triglycerides, and LDL-C concentrations, together with significantly lower HDL-C levels. Serum TSH concentrations showed significant positive correlations with total cholesterol, triglycerides, and LDL-C, whereas HDL-C demonstrated a significant inverse correlation.

References

1. Graves M, Howse K, Pudwell J, Smith GN. Pregnancy-related cardiovascular risk indicators: Primary care approach to postpartum management and prevention of future disease. Can Fam Physician. 2019;65:883–889.
2. Sharami HS, Ranjbar AZ, Alizadeh F, Kazemnejad E. The relationship of hyperlipidemia with maternal and neonatal outcomes in pregnancy: A cross-sectional study. Int J Reprod Biomed. 2019;17:739–748.
3. Wang X, Xian T, Zhang L, Jia X, Man F, Liu L. Associations between urinary iodine concentration, lipid profile and other cardiometabolic risk factors in adolescents: A cross-sectional, population-based analysis. Br J Nutr. 2019;121:1039–1048.
4. Duttaroy AK, Basak S. Maternal fatty acid metabolism in pregnancy and its consequences in the feto-placental development. Front Physiol. 2021;12:787848.
5. Tabacu C, Manolea MM, Novac L, Dijmarescu AL, Boldeanu MV. Maternal lipid profile as a risk factor for gestational diabetes mellitus in obese women. Curr Health Sci J. 2021;47:209–214.
6. Retnakaran R, Shah BR. The adverse cardiovascular risk factor profile of women with pre-eclampsia develops over time in the years before pregnancy. BJOG. 2022;129:1512–1520.
7. Vekic J, Zeljkovic A, Cicero AFG, Janez A, Stoian AP, Sonmez A. Atherosclerosis development and progression: the role of atherogenic small, dense LDL. Medicina (Kaunas). 2022;58:299.
8. Poornima IG, Indaram M, Ross JD, Agarwala A, Wild RA. Hyperlipidemia and risk for preeclampsia. J Clin Lipidol. 2022;16:253–260.
9. Bohn MK, Adeli K. Physiological and metabolic adaptations in pregnancy: importance of trimester-specific reference intervals to investigate maternal health and complications. Crit Rev Clin Lab Sci. 2022;59:76–92.
10. Bashir M, Navti OB, Ahmed B, Konje JC. Hyperlipidaemia and severe hypertriglyceridaemia in pregnancy. Obstet Gynaecol. 2023;25:196–209.
11. Formisano E, Proietti E, Perrone G, Demarco V, Galoppi P, Stefanutti C. Characteristics, physiopathology and management of dyslipidemias in pregnancy: a narrative review. Nutrients. 2024;16:2927.
12. Lewek J, Bielecka-Dąbrowa A, Toth PP, Banach M. Dyslipidaemia management in pregnant patients: a 2024 update. Eur Heart J Open. 2024;4:oeae032.
13. Li LJ, Lu R, Rawal S, et al. Maternal plasma phospholipid polyunsaturated fatty acids in early pregnancy and thyroid function throughout pregnancy: a longitudinal study. Am J Clin Nutr. 2024; (online).
14. Wang Y, Sun F, Wu P, et al. A prospective study of early-pregnancy thyroid markers, lipid species, and risk of gestational diabetes mellitus. J Clin Endocrinol Metab. 2022;107(10):e4014-e4025.
15. Sullivan SA. Hypothyroidism in pregnancy. Clin Obstet Gynecol. 2019;62(2),
16. Hamblin PS, Sheehan PM, Allan C, et al. Subclinical hypothyroidism during pregnancy: the Melbourne public hospitals consensus. Intern Med J. 2019;49(8),
17. Taylor PN, Lazarus JH. Hypothyroidism in pregnancy. Endocrinol Metab Clin North Am. 2019;48(3),
18. Nazarpour S, Ramezani Tehrani F, Amiri M, et al. Levothyroxine treatment and pregnancy outcomes in women with subclinical hypothyroidism: systematic review and meta-analysis. Arch Gynecol Obstet. 2019;299,
19. Bein M, Yu OHY, Grandi SM, et al. Levothyroxine and the risk of adverse pregnancy outcomes in SCH: systematic review and meta-analysis. BMC Endocr Disord. 2021;21:
20. Ding Z, Liu Y, Maraka S, et al. Pregnancy and neonatal outcomes with LT4 in SCH using new diagnostic criteria: systematic review and meta-analysis. Front Endocrinol (Lausanne). 2021;12
21. Hu M, Gao S, Huang K, Wang X, Li J, et al. Elevated serum TSH levels and TPOAb positivity in early pregnancy are associated with increased risk of hypertensive disorders of pregnancy: a prospective cohort study. International Journal of Medical Sciences. 2025.
22. Li J, Xu Y, Cai Y, et al. Association of differential metabolites with small intestinal microflora and maternal outcomes in subclinical hypothyroidism during pregnancy. Front Cell Infect Microbiol. 2022;12

23. Gietka-Czernel M, Glinicki P. Subclinical hypothyroidism in pregnancy: controversies on diagnosis and treatment. Pol Arch Intern Med. 2021;131(9)
24. Liu H, Peng D. Update on dyslipidemia in hypothyroidism: mechanisms. Endocr Connect. 2022;11(4):e210683.
25. Yang Y, Yuan H, Wang X, et al. Significance of levothyroxine treatment on serum lipid in pregnant women with subclinical hypothyroidism. BMC Pregnancy Childbirth. 2022;22.
26. Cai Y, Xu Y, Ban Y, et al. Plasma lipid profile and intestinal microflora in pregnancy women with hypothyroidism and their correlation with pregnancy outcomes. Front Endocrinol (Lausanne). 2022;12:792536.
27. Businge CB, Longo-Mbenza B. Dyslipidemia associated with iodine deficiency and subclinical hypothyroidism in pregnant normotensive and preeclamptic women. Pathophysiology. 2025;32.
28. Dong L, Hong F, Ma J, Hong H. Role of second-trimester hematological and biochemical indicators in predicting preeclampsia: a retrospective study. BMC Pregnancy and Childbirth. 2026.
29. Xie Y, Chen B, Gao S, Li J, Han J, et al. Endocrine–Cardiac Axis: A narrative review of long-term cardiovascular risks in women with gestational diabetes, hypertensive disorders, and thyroid disease. Biomedicines. 2026.