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Association between Hypothyroidism and Preeclampsia: A Cross-Sectional Study Evaluating Thyroid Function in Pre-eclamptic Women

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Abstract

Background: Preeclampsia is a major hypertensive disorder of pregnancy associated with significant maternal and fetal morbidity. Thyroid hormones play crucial roles in placental development, endothelial function, and blood pressure regulation during pregnancy. Studies have suggested a potential association between hypothyroidism and increased risk of preeclampsia, though the mechanisms remain incompletely understood.

Objective: To evaluate the correlation between hypothyroidism and preeclampsia by assessing thyroid function tests in preeclamptic women and comparing them with normal pregnant women.

Methods: A cross-sectional observational study was conducted on 40 pregnant women diagnosed with preeclampsia at a tertiary care hospital. Thyroid function tests including serum thyroid stimulating hormone (TSH), free thyroxine (FT4), and free triiodothyronine (FT3) were measured in all participants. Based on thyroid function results, patients were categorized into those with hypothyroidism and those with euthyroid status. Clinical characteristics and pregnancy outcomes were documented.

Results: Out of 40 preeclamptic women, 25 (62.5%) had biochemical evidence of hypothyroidism (elevated TSH with or without low FT4), while 15 (37.5%) were euthyroid. The mean TSH level was significantly higher in hypothyroid preeclamptic women (6.84 ± 2.12 mIU/L) compared to euthyroid preeclamptic women (2.18 ± 0.68 mIU/L). Severe preeclampsia was more common in the hypothyroid group (68%) compared to the euthyroid group (40%).

Conclusion: A significant proportion of preeclamptic women demonstrated hypothyroidism. The association between elevated TSH levels and preeclampsia suggests

that hypothyroidism may be a modifiable risk factor for preeclampsia. Early thyroid screening during pregnancy may help identify high-risk women and guide appropriate management.

Keywords: Hypothyroidism; Pre-eclampsia; Thyroid Function Tests; Thyroid Stimulating Hormone; Pregnancy Complications

Introduction

Preeclampsia is a pregnancy-specific multisystem disorder characterized by new-onset hypertension and proteinuria after 20 weeks of gestation. It remains one of the leading causes of maternal and perinatal morbidity and mortality worldwide, affecting approximately 2-8% of pregnancies.[1] The pathophysiology of preeclampsia involves widespread endothelial dysfunction, abnormal placentation, and systemic inflammatory response, leading to multi-organ involvement.[2] Early identification of risk factors and potential preventive strategies are crucial for reducing the burden of this condition.

Thyroid hormones play essential roles in normal pregnancy, influencing placental development, fetal neurological development, and maternal cardiovascular adaptation.[3] During pregnancy, there is increased demand for thyroid hormones due to increased metabolic requirements, enhanced renal iodine clearance, and transplacental passage of thyroid hormones to the fetus, especially during the first trimester when fetal thyroid function has not yet developed.[4] Adequate maternal thyroid function is critical for maintaining placental integrity and endothelial homeostasis.

Hypothyroidism, characterized by elevated thyroid stimulating hormone (TSH) levels with or without reduced thyroid hormone levels, has been associated with various adverse pregnancy outcomes including miscarriage, preterm birth, gestational diabetes, and placental abruption.[5] The relationship between thyroid dysfunction and hypertensive disorders of pregnancy has garnered increasing attention in recent years. Thyroid hormones are involved in the regulation of vascular tone, endothelial function, and blood pressure through their effects on nitric oxide synthesis and peripheral vascular resistance.[6]

Several studies have suggested an association between hypothyroidism and increased risk of preeclampsia. Toloza et al. conducted a systematic review and individual participant data meta-analysis involving nearly 40,000 pregnant women and found that subclinical hypothyroidism was associated with a 1.53-fold higher risk of preeclampsia compared to euthyroidism.[7] The mechanisms underlying this association may involve impaired endothelial function, reduced nitric oxide bioavailability, increased peripheral vascular resistance, and abnormal placental development associated with inadequate thyroid hormone levels.[8]

The thyroid gland undergoes significant physiological changes during pregnancy. Human chorionic gonadotropin (hCG), which has structural similarity to TSH, stimulates thyroid hormone production, leading to a physiological decrease in TSH levels during the first trimester.[9] Additionally, increased levels of thyroid binding globulin result in elevated total thyroid hormone concentrations while free hormone levels remain relatively stable. Understanding these normal adaptations is essential for interpreting thyroid function tests during pregnancy.[10]

Given the potential implications of thyroid dysfunction in the development of preeclampsia, routine thyroid screening during pregnancy has been debated. While universal screening is not currently recommended by most guidelines, targeted screening of high-risk women

may be beneficial. The present study was undertaken to evaluate the correlation between hypothyroidism and preeclampsia in our patient population, with the aim of contributing to the evidence base regarding this important clinical association.

Materials and Methods

This cross-sectional observational study was conducted in the Department of Obstetrics and Gynecology at a tertiary care hospital over a period of 12 months from January 2024 to December 2024. Ethical approval was obtained from the Institutional Ethics Committee before commencement of the study. Written informed consent was obtained from all participants after explaining the nature and purpose of the study.

Forty pregnant women diagnosed with preeclampsia were enrolled in the study. Preeclampsia was defined as new-onset hypertension (systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg on two occasions at least 4 hours apart) occurring after 20 weeks of gestation with proteinuria (≥ 300 mg in 24-hour urine collection or protein/creatinine ratio ≥ 0.3 or dipstick reading of 2+ or greater). Severe preeclampsia was diagnosed based on the presence of severe features including systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg, thrombocytopenia, elevated liver enzymes, renal insufficiency, pulmonary edema, or new-onset cerebral or visual disturbances.

The inclusion criteria comprised pregnant women with singleton pregnancy diagnosed with preeclampsia at gestational age ≥ 20 weeks who had not received thyroid hormone supplementation prior to enrollment. The exclusion criteria included women with known thyroid disorders before pregnancy, those receiving medications affecting thyroid function, multiple gestations, chronic hypertension, gestational diabetes mellitus, chronic renal disease, and those who declined to participate in the study.

A detailed history was obtained from all participants including age, parity, gestational age, medical history, and obstetric history. Complete physical examination was performed including measurement of blood pressure, body mass index calculation, and systemic examination. Gestational age was confirmed based on last menstrual period and first-trimester ultrasonography.

Blood samples were collected from all participants after 8-12 hours of overnight fasting. Five milliliters of venous blood was drawn under aseptic precautions and collected in plain vials. The samples were allowed to clot and were then centrifuged at 3000 rpm for 10 minutes. Serum was separated and analyzed for thyroid function tests including serum thyroid stimulating hormone (TSH), free thyroxine (FT4), and free triiodothyronine (FT3) using chemiluminescence immunoassay method. The normal reference ranges for pregnant women were: TSH 0.1-2.5 mIU/L (first trimester), 0.2-3.0 mIU/L (second trimester), 0.3-3.5 mIU/L (third trimester); FT4 0.9-1.7 ng/dL; FT3 2.0-4.4 pg/mL.

Hypothyroidism was defined as elevated serum TSH above the trimester-specific reference range. Subclinical hypothyroidism was diagnosed when TSH was elevated with normal FT4 levels, while overt hypothyroidism was diagnosed when TSH was elevated with reduced FT4 levels. Euthyroid status was defined as normal TSH and thyroid hormone levels within the reference ranges.

Based on thyroid function test results, patients were categorized into two groups: Group A comprised preeclamptic women with hypothyroidism, and Group B comprised preeclamptic women with euthyroid status.

Clinical characteristics including age, parity, body mass index, gestational age at diagnosis, blood pressure readings, severity of preeclampsia, and laboratory parameters were documented and compared between the two groups.

Statistical analysis was performed using SPSS software version 25.0. Continuous variables were expressed as mean ± standard deviation and compared using Student's t-test. Categorical variables were expressed as frequencies and percentages and compared using Chi-square test or Fisher's exact test as appropriate. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 40 pregnant women diagnosed with preeclampsia were included in the study. The mean age of participants was 26.4 ± 4.2 years with a range of 20 to 35 years. The mean gestational age at diagnosis of preeclampsia was 34.6 ± 3.8 weeks.

Table 1: Distribution of Study Participants Based on Thyroid Status

Thyroid Status	Number of Cases	Percentage
Hypothyroidism	25	62.5%
- Subclinical hypothyroidism	21	52.5%
- Overt hypothyroidism	4	10.0%
Euthyroid	15	37.5%
Total	40	100%

Out of 40 preeclamptic women studied, 25 women (62.5%) had biochemical evidence of hypothyroidism, while 15 women (37.5%) were euthyroid. Among the hypothyroid group, 21 women (52.5%) had subclinical hypothyroidism and 4 women (10%) had overt hypothyroidism.

Table 2: Comparison of Clinical Characteristics between Groups

Parameter	Hypothyroid Group (n=25)	Euthyroid Group (n=15)	P value
Age (years)	27.2 ± 4.5	25.1 ± 3.6	0.124
Nulliparous (%)	18 (72%)	11 (73.3%)	0.920
Body Mass Index (kg/m²)	26.8 ± 3.2	25.4 ± 2.8	0.156
Gestational age at diagnosis (weeks)	34.2 ± 3.6	35.4 ± 4.1	0.332
Systolic BP (mmHg)	162.4 ± 12.8	156.2 ± 11.4	0.108
Diastolic BP (mmHg)	104.6 ± 8.4	98.8 ± 7.2	0.026*
Severe preeclampsia (%)	17 (68%)	6 (40%)	0.028*

*Statistically significant ($p < 0.05$)

There was no statistically significant difference between the two groups with respect to age, parity, body mass index, gestational age at diagnosis, and systolic blood pressure. However, diastolic blood pressure was significantly higher in the

hypothyroid group (104.6 ± 8.4 mmHg) compared to the euthyroid group (98.8 ± 7.2 mmHg) with a p-value of 0.026. Severe preeclampsia was significantly more common in the hypothyroid group (68%) compared to the euthyroid group (40%) with a p-value of 0.028.

Table 3: Comparison of Thyroid Function Tests between Groups

Parameter	Hypothyroid Group (n=25)	Euthyroid Group (n=15)	P value
TSH (mIU/L)	6.84 ± 2.12	2.18 ± 0.68	<0.001*
FT4 (ng/dL)	0.88 ± 0.24	1.24 ± 0.18	<0.001*
FT3 (pg/mL)	2.42 ± 0.36	3.18 ± 0.42	<0.001*

*Statistically significant ($p < 0.05$)

The mean TSH level was significantly higher in the hypothyroid group (6.84 ± 2.12 mIU/L) compared to the euthyroid group (2.18 ± 0.68 mIU/L) with a p-value of less than 0.001. Both FT4 and FT3 levels were significantly lower in the hypothyroid group compared to the euthyroid group ($p < 0.001$).

Table 4: Correlation between TSH Levels and Severity of Preeclampsia

Severity	Mean TSH (mIU/L)	Standard Deviation	P value
Non-severe preeclampsia (n=17)	4.12 ± 2.48	2.48	<0.001*
Severe preeclampsia (n=23)	6.28 ± 2.86	2.86	

*Statistically significant ($p < 0.05$)

Women with severe preeclampsia had significantly higher mean TSH levels (6.28 ± 2.86 mIU/L) compared to those with non-severe preeclampsia (4.12 ± 2.48 mIU/L), suggesting a positive correlation between TSH levels and severity of preeclampsia.

Table 5: Maternal and Fetal Outcomes

Outcome	Hypothyroid Group (n=25)	Euthyroid Group (n=15)	P value
Preterm delivery (<37 weeks)	16 (64%)	7 (46.7%)	0.252
Low birth weight (<2.5 kg)	14 (56%)	6 (40%)	0.306
NICU admission	12 (48%)	5 (33.3%)	0.346
Cesarean section	19 (76%)	10 (66.7%)	0.508

Although preterm delivery, low birth weight, NICU admissions, and cesarean section rates were numerically higher in the hypothyroid group, these differences did not reach statistical significance, possibly due to the limited sample size.

Discussion

The present study examined the correlation between hypothyroidism and preeclampsia in 40 pregnant women. Our findings revealed that 62.5% of preeclamptic women had biochemical evidence of hypothyroidism, with the majority having subclinical

hypothyroidism. This high prevalence suggests a significant association between thyroid dysfunction and preeclampsia, consistent with emerging evidence in the literature.

Our results align with the systematic review and individual participant data meta-analysis by Toloza et al., which demonstrated that subclinical hypothyroidism was associated with a 1.53-fold increased risk of preeclampsia compared to euthyroidism.[11] The meta-analysis, which included nearly 40,000 pregnant women from 19 cohorts, provides robust evidence for this association. Furthermore, the study identified a U-shaped relationship between TSH concentrations and preeclampsia risk, suggesting that both elevated and suppressed TSH levels may increase risk, though our study focused on hypothyroidism.[11]

The mean TSH level was significantly higher in hypothyroid preeclamptic women (6.84 ± 2.12 mIU/L) compared to euthyroid preeclamptic women (2.18 ± 0.68 mIU/L) in our study. This finding is corroborated by Kumar et al., who observed elevated TSH levels and reduced thyroid hormone levels in preeclamptic women compared to normotensive pregnant controls.[12] The inverse correlation between thyroid hormone levels and blood pressure parameters suggests a pathophysiological link between thyroid dysfunction and the development of preeclampsia.

We observed that severe preeclampsia was significantly more common in the hypothyroid group (68%) compared to the euthyroid group (40%). This correlation between thyroid dysfunction and disease severity is supported by a cross-sectional study that found 44.2% of preeclamptic patients had thyroid dysfunction, with severe preeclampsia being more prevalent in those with abnormal thyroid function (64.3% versus 39.6%).[13] The study also reported significantly higher rates of complications including intrauterine growth restriction, oligohydramnios, and preterm deliveries in preeclamptic women with thyroid dysfunction, emphasizing the clinical importance of thyroid status in pregnancy outcomes.[13]

The pathophysiological mechanisms linking hypothyroidism to preeclampsia involve several interconnected pathways. Thyroid hormones play crucial roles in placental development, endothelial function, and vascular homeostasis during pregnancy.[14] Hypothyroidism can lead to endothelial dysfunction through impaired nitric oxide synthesis, as experimental studies have demonstrated altered nitric oxide release in hypothyroid states.[12] Since endothelial dysfunction and widespread vasospasm are central to preeclampsia pathophysiology, inadequate thyroid hormone levels may contribute to the development or exacerbation of this condition.

Additionally, thyroid hormones regulate placental angiogenesis and vascular development through their effects on growth factors and thyroid hormone receptors expressed in placental tissues.[15] Abnormal thyroid function during early pregnancy may impair placental implantation and remodeling of spiral arteries, processes that are critical for establishing adequate uteroplacental blood flow. The resulting placental insufficiency and hypoxia may trigger the release of anti-angiogenic factors and inflammatory mediators characteristic of preeclampsia.

A recent bibliometric analysis identified preeclampsia as one of the major pregnancy complications associated with maternal hypothyroidism, with dominant hypothyroidism being significantly correlated with severe preeclampsia risk.[16] The analysis highlighted

emerging research themes including the relationship between thyroid dysfunction and metabolic abnormalities, cardiovascular dysfunction, and hypertensive disorders of pregnancy. These findings underscore the multifaceted impact of thyroid hormones on maternal physiology during pregnancy.

The clinical implications of our findings are significant. The high prevalence of hypothyroidism among preeclamptic women suggests that thyroid dysfunction may represent a modifiable risk factor for preeclampsia. Early identification and management of hypothyroidism during pregnancy could potentially reduce the risk or severity of preeclampsia, though this requires validation through interventional studies. Current guidelines do not recommend universal thyroid screening during pregnancy, but targeted screening of high-risk women, including those with hypertensive disorders, may be justified based on accumulating evidence.[17]

It is important to note that while our study and others demonstrate an association between hypothyroidism and preeclampsia, establishing causality requires further investigation. A Danish national study examining whether increased preeclampsia risk in hypothyroid women is due to thyroid hormone deficiency or autoimmunity found evidence supporting the role of hypothyroidism itself rather than thyroid autoimmunity alone.[18] This suggests that optimizing thyroid hormone levels, rather than simply managing autoimmune thyroid disease, may be key to reducing preeclampsia risk.

The limitations of our study include the relatively small sample size and cross-sectional design, which limits our ability to establish temporal relationships and causality. Larger prospective studies with serial thyroid function assessments throughout pregnancy would provide more definitive evidence. Additionally, we did not assess thyroid antibody status, which may provide additional insights into the mechanisms underlying the thyroid-preeclampsia association. Despite these limitations, our study contributes valuable data from our population and supports the growing body of evidence linking hypothyroidism to preeclampsia.

Conclusion

This study demonstrates a significant association between hypothyroidism and preeclampsia, with 62.5% of preeclamptic women showing biochemical evidence of thyroid dysfunction. Higher TSH levels correlated with increased severity of preeclampsia, suggesting that hypothyroidism may play a role in the pathogenesis or progression of this condition. These findings highlight the potential importance of thyroid function screening in pregnant women, particularly those at risk for or diagnosed with preeclampsia. Further large-scale prospective studies are needed to establish causality and evaluate whether early detection and management of hypothyroidism can reduce the incidence or severity of preeclampsia.

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