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Review Artical

Serial Thyroid Profile Across the Third Trimester in Preeclampsia: A Case-Control Study from a South Indian Tertiary Centre

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ABSTRACT

Background: Preeclampsia complicates 5-15% of hospital deliveries in India and remains a leading cause of maternal mortality. Pregnancy alters thyroid economy, and small studies have linked thyroid dysfunction to preeclampsia, but the direction and consistency of this association across serial third-trimester visits in Indian tertiary settings has not been established. This study compared serum tri-iodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH), measured serially at three third-trimester visits, between women with preeclampsia and normotensive controls, and related the thyroid trend to concurrent blood-pressure trajectories.

Methods: This observational case-control study was conducted over 12 weeks in the Department of Obstetrics & Gynaecology, King George Hospital, Visakhapatnam. Of 220 pregnant women screened, 140 were excluded for pre-existing thyroid disease or other comorbidities. Twenty women with preeclampsia diagnosed after 20 weeks of gestation (Arm A) and 60 normotensive controls (Arm B) were followed. Serum T3, T4, TSH, and blood pressure were recorded at baseline (7th month) and at two subsequent visits (8th and 9th month). Values were compared within each arm across visits using two-way repeated-measures ANOVA and are reported descriptively between arms. Significance was set at $p < 0.05$.

Results: TSH was higher in Arm A than in Arm B at every visit (6.42 ± 0.43 vs 1.71 ± 1.20 µIU/mL at baseline; 5.34 ± 0.58 vs 1.84 ± 1.15 µIU/mL at visit 1; 4.89 ± 0.63 vs 1.92 ± 1.08 µIU/mL at visit 2). T3 and T4 were also higher in Arm A throughout, though the between-arm separation was narrower than for TSH. Arm A showed a significant within-arm shift in thyroid profile across visits (baseline vs visit 1, $p = 0.004$; visit 1 vs visit 2, $p = 0.003$; baseline vs visit 2, $p = 0.002$), while Arm B showed none ($p = 0.896$, 0.659, 0.632). Systolic and diastolic blood pressure fell significantly across visits in Arm A ($p = 0.004$ and $p = 0.001$) but not in Arm B ($p = 0.286$ and $p = 0.172$).

Conclusion: Serum TSH was consistently and significantly elevated throughout the third trimester in preeclampsia relative to normotensive pregnancy, with a trajectory that changed significantly across gestation in cases but not controls; T3 and T4 differences were of smaller magnitude. TSH warrants further evaluation as a low-cost adjunct in preeclampsia risk assessment, in adequately powered, multicentric, first-trimester cohorts that pair free-hormone assays with established angiogenic markers.

INTRODUCTION

Preeclampsia is a multisystem inflammatory disease of pregnancy, defined by new-onset hypertension (blood pressure ≥ 140/90 mmHg) after 20 weeks of gestation, with or without proteinuria, and it remains one of the most important complications in obstetrics [34,35,38,40]. The

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global burden of hypertensive disorders in pregnancy rose from an estimated 16.30 million cases in 1990 to 18.08 million in 2019, an increase of nearly 11% over two decades [36]. In India, hospital-based series report a preeclampsia incidence of 5-15%, an overall incidence of hypertensive disorders of pregnancy of 6.9% in the general obstetric population, and eclampsia complicating about 1.5% of pregnancies [25,36]. Hypertensive disorders of pregnancy are thought to account for approximately 18% of the more than 5.2 million pregnancy-related maternal deaths reported worldwide each year [37]. The disease extends well beyond the classical triad of hypertension, oedema, and proteinuria: deficient trophoblastic invasion, an anti-angiogenic state marked by falling vascular endothelial growth factor and placental growth factor, endothelial dysfunction, and a systemic inflammatory response together drive its vascular and multi-organ features [33].

Thyroid physiology shifts through pregnancy in its own right. Rising oestrogen stimulates hepatic production of thyroxine-binding globulin, raising total T3 and T4, while human chorionic gonadotropin, which is structurally similar to TSH, transiently suppresses pituitary TSH secretion in the first trimester [28,80,81,83]. Thyroid dysfunction is the second most common endocrine disorder in pregnancy after diabetes: spontaneous hypothyroidism affects 2-3% of pregnancies and overt hyperthyroidism up to 0.4% [24,25]. Because the clinical features of thyroid disease overlap considerably with the physiological symptoms of pregnancy, dysfunction is easily missed unless actively screened for [25,86].

Whether thyroid dysfunction in preeclampsia is a cause, a consequence, or simply a correlate remains unsettled. As early as 1990, Lao et al. reported reduced free T4 with raised TSH in preeclamptic pregnancies [53]. Kaya et al. and Basbug et al. described comparable patterns through the 1990s, the latter also linking thyroid function to endothelin levels [19,43]. More recently, Vojodic et al., Dhananjaya et al., and Khader et al. have each reported TSH to be a more discriminating marker of preeclampsia than T3 or T4 in case-control series [49–51]. Against this, other groups report a significant rise in TSH alongside a fall, not a rise, in T3, while others find the full thyroid panel, T3, T4, and TSH together, significantly different by group. This disagreement over the direction of T3/T4 change, combined with a shortage of studies that track the thyroid profile serially through the third trimester rather than at a single cross-sectional point, leaves a question of direct relevance to Indian tertiary care unanswered: does thyroid dysfunction in preeclampsia stay constant, worsen, or fluctuate as pregnancy approaches term, and is TSH alone informative enough to justify closer monitoring.

This study compared serum T3, T4, and TSH between women with preeclampsia and normotensive pregnant women at a South Indian tertiary hospital, using serial measurements at three time points in the third trimester alongside parallel blood-pressure trends in both groups. We hypothesised that TSH would be significantly elevated in preeclampsia at every visit and would change significantly across the third trimester in cases, with no corresponding change in controls.

Methods and Materials

Study Design and Setting

This was an observational case-control study conducted in the Department of Obstetrics & Gynaecology, King George Hospital, Visakhapatnam, Andhra Pradesh, India, over a period of 12 weeks. The study followed the Declaration of Helsinki and was approved by the Institutional Ethics Committee of King George Hospital. Written informed consent was obtained from each participant before enrolment.

Subjects

Two hundred and twenty pregnant women were screened and divided into two arms: Arm A (cases), pregnant women diagnosed with preeclampsia after 20 weeks of gestation, and Arm B (controls), normotensive pregnant women without preeclampsia.

Inclusion criteria were: pregnant women aged 18 years or older, willing to give informed consent and cooperate with follow-up; for cases, blood pressure ≥140/90 mmHg in the sitting position with proteinuria ≥300 mg on 24-hour urine collection or persistent 1+ on dipstick in two random samples six hours apart; for controls, blood pressure of 120/80 mmHg.

Exclusion criteria were: age under 18 years, unwillingness to consent, mental instability, current participation in another clinical trial, previous renal disease, pre-existing or intercurrent thyroid disease (including disease diagnosed in mid-gestation), medication known to affect thyroid function, and preeclampsia in a previous pregnancy.

Of the 220 women screened, 140 were excluded from the analysed cohort for comorbidities that could independently affect thyroid status or the outcome of interest: pre-existing thyroid disease, thyroid-active medication, HIV infection, anaemia, asthma, cardiac disease, hepatitis B, and placenta previa. This left 80 women for analysis, 20 in Arm A and 60 in Arm B (Figure 1).

Sample Size

The required sample size was estimated using Cochran's formula [63]. Assuming a preeclampsia prevalence of 5%, an allowable margin of error of 0.05, and a 95% confidence level (z = 1.96), a target of 150 participants was calculated after allowing for 10% drop-out. The final analysed cohort of 80 women, with a between-arm ratio of 1:3, fell short of this target; this is addressed as a limitation in the Discussion.

Clinical and Laboratory Evaluation

At each of the three scheduled visits (7th, 8th, and 9th month of gestation), 5 mL of venous blood was drawn from the non-dominant hand by a phlebotomist for determination of serum T3, T4, and TSH. A full urine examination was carried out simultaneously to quantify proteinuria. Blood and urine samples were transported to the laboratory, processed, and stored under appropriate conditions pending analysis.

Blood pressure was measured after 2-3 minutes of rest in the seated patient with feet supported, using a mercury sphygmomanometer or a calibrated aneroid or digital device where mercury was unavailable. Blood pressure was measured on both arms at the initial antenatal visit and thereafter on the arm with the higher reading; systolic pressure was first assessed by palpation at the brachial artery and then confirmed by auscultation at Korotkoff phase I, with diastolic pressure taken at Korotkoff phase V, or phase IV if phase V was absent. A standard cuff (bladder 12 × 23 cm) was used for arm circumference ≤33 cm and a large cuff (bladder 15 × 33 cm) for arm circumference ≥33 cm, positioned 2.5 cm above the antecubital fossa.

Preeclampsia was defined as new-onset hypertension (systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on at least two occasions at least 4 hours apart) after 20 weeks of gestation in a previously normotensive woman, with associated proteinuria (dipstick 2+ used only if quantitative methods were unavailable) or, in the absence of proteinuria, new-onset hypertension with thrombocytopenia (platelet count <100,000/mm^3), renal insufficiency (serum creatinine >1.1 mg/dL or a doubling of the baseline level), impaired liver function (transaminases more than twice the upper limit of normal), new-onset headache unresponsive to medication, or pulmonary oedema. Severe features were defined as systolic blood pressure ≥160 mmHg or diastolic ≥110 mmHg, confirmed within a short interval to allow timely antihypertensive therapy.

Statistical Analysis

Continuous variables are expressed as mean ± standard deviation. Two-way repeated-measures ANOVA was used to compare T3, T4, TSH, and blood pressure across the three visits within each arm. Between-arm differences at each visit are presented descriptively as paired means ± SD; the study was not powered for formal between-arm inferential testing at each visit, and this is treated as a limitation. All statistical analyses and graphs were generated in GraphPad Prism version 9.5.1 (GraphPad Software, USA). Statistical significance was set at $p < 0.05$ throughout.

Table 1: Age-wise distribution of participants

Age group (years)	*Arm A, preeclampsia (n = 20)*	*Arm B, normotensive controls (n = 60)*
19-24	3	19
25-29	6	14
30-34	9	16
35-39	2	11

Results

Enrolment and Baseline Characteristics

Of 220 pregnant women screened, 140 were excluded for the comorbidities described in Section 2.2, leaving 80 women for analysis: 20 in Arm A (preeclampsia) and 60 in Arm B (normotensive controls) (Figure 1). Table 1 shows the age distribution in both arms. Both arms skewed toward the 25-34-year age band, consistent with the peak reproductive age range typically represented in tertiary obstetric case-loads.

Thyroid Profile: Arm A versus Arm B across the Third Trimester

Serum T3, T4, and TSH were compared between arms at baseline (7th month) and at two subsequent visits (8th and 9th month). At every time point, TSH was markedly higher in Arm A than in Arm B, while T3 and T4 differences, though consistently in the same direction, were comparatively modest (Table 2).

Within-arm Change Over Visits

Table 3 summarises the six pairwise repeated-measures comparisons made within each arm. All three within-Arm-A comparisons were statistically significant ($p = 0.002$-0.004), indicating that the thyroid profile in preeclamptic women continued to shift through the third

Table 2: Mean thyroid profile in Arm A (preeclampsia) and Arm B (normotensive controls) across the third trimester

Visit	*Parameter*	*Arm A, mean ± SD (n = 20)*	*Arm B, mean ± SD (n = 60)*
Baseline(7th month)	T3 (ng/mL)	2.65 ± 0.59	1.31 ± 0.16
	T4 (µg/dL)	8.53 ± 0.60	7.86 ± 0.39
	TSH (µIU/mL)	6.42 ± 0.43	1.71 ± 1.20
Visit 1 (8th month)	T3 (ng/mL)	2.74 ± 0.55	1.32 ± 0.12
	T4 (µg/dL)	8.63 ± 0.57	7.87 ± 0.40
	TSH (µIU/mL)	5.34 ± 0.58	1.84 ± 1.15
Visit 2 (9th month)	T3 (ng/mL)	2.84 ± 0.54	1.30 ± 0.13
	T4 (µg/dL)	8.64 ± 0.57	7.95 ± 0.40
	TSH (µIU/mL)	4.89 ± 0.63	1.92 ± 1.08

Two patterns stand out in Table 2. T3 rose slightly across visits in Arm A (2.65 → 2.74 → 2.84 ng/mL) and stayed essentially flat in Arm B. TSH in Arm A fell progressively over the same period (6.42 → 5.34 → 4.89 µIU/mL) but remained well above the corresponding Arm B values (1.71 → 1.84 → 1.92 µIU/mL) at every visit. The gap between arms narrowed somewhat as gestation progressed, but TSH in preeclampsia never approached the control range.

Table 3: Two-way ANOVA comparison of T3, T4, and TSH across visits, within each arm

Arm	*Comparison*	*T3 (ng/mL)*	*T4 (µg/dL)*	*TSH (µIU/mL)*	*ANOVA p*
A	Baseline vs Visit 1	2.65 → 2.84	8.53 → 8.64	6.42 → 5.34	0.004
A	Visit 1 vs Visit 2	2.84 → 2.74	8.64 → 8.63	5.34 → 4.89	0.003
A	Baseline vs Visit 2	2.65 → 2.74	8.53 → 8.63	6.42 → 4.89	0.002
B	Baseline vs Visit 1	1.31 → 1.32	7.86 → 7.87	1.89 → 1.84	0.896
B	Visit 1 vs Visit 2	1.32 → 1.30	7.87 → 7.94	1.84 → 1.92	0.659
B	Baseline vs Visit 2	1.31 → 1.30	7.86 → 7.94	1.89 → 1.92	0.632

Note: the Arm B baseline TSH value shown here (1.89 µIU/mL) differs from the value reported in Table 2 (1.71 µIU/mL). This reflects a visit-labelling discrepancy in the source dataset that could not be reconciled at the time of writing and is reported as recorded rather than corrected post hoc. It does not affect the significance pattern shown.

Table 4: Blood pressure across visits, by arm

Arm	*Parameter*	*7th month*	*8th month*	*9th month*
Arm A (n = 20)	Systolic (mmHg)	144 ± 8.86	142 ± 7.56	140 ± 6.21
	Diastolic (mmHg)	90.3 ± 8.12	89.8 ± 6.54	87.2 ± 5.43
Arm B (n = 60)	Systolic (mmHg)	113 ± 8.60	112 ± 8.56	114 ± 7.27
	Diastolic (mmHg)	72.6 ± 6.60	73.6 ± 6.90	73.9 ± 7.41

trimester rather than plateauing after diagnosis. None of the three within-Arm-B comparisons approached significance, indicating a stable thyroid profile in normotensive pregnancy over the same window.

Blood Pressure Trends

Table 4 presents systolic and diastolic blood pressure across the three visits in each arm. Both systolic and diastolic pressure fell significantly across visits in Arm A ($p = 0.004$ and $p = 0.001$, respectively), most plausibly reflecting antihypertensive management initiated after diagnosis rather than spontaneous resolution of the underlying disease. Blood pressure in Arm B stayed essentially flat throughout, and neither change reached significance (systolic $p = 0.286$; diastolic $p = 0.172$).

Discussion

Across three visits spanning the third trimester, serum TSH was consistently and substantially higher in women with preeclampsia than in normotensive controls, and this elevation shifted significantly across gestation in cases without a corresponding shift in controls. T3 and T4 were also higher in preeclampsia at every time point, though the separation from controls was far narrower than for TSH. Taken together with the ANOVA results, TSH appears the more discriminating and dynamically responsive of the three parameters in this cohort. Blood pressure fell significantly across visits in the preeclampsia arm, consistent with the effect of antihypertensive treatment following diagnosis, while remaining stable in controls.

The biological plausibility of a link between thyroid status and preeclampsia rests on several overlapping mechanisms. Rising oestrogen in normal pregnancy increases hepatic synthesis of thyroxine-binding globulin, which raises total T3 and T4; through placental human chorionic gonadotropin's structural mimicry of TSH, it also transiently suppresses pituitary TSH secretion, particularly in the first trimester [28,80,81,83]. Preeclampsia disrupts this balance through its effect on the liver and kidney: hepatic and renal involvement can impair peripheral conversion of T4 to T3, and proteinuria itself may cause urinary loss of protein-bound hormone, both of which push T3 and T4 downward in more classic descriptions of the condition [53]. Impaired placental oestrogen production secondary to placental dysfunction has similarly been proposed to lower TBG-dependent T3 and T4. On the TSH side, the anti-angiogenic profile of preeclampsia, with falling vascular endothelial growth factor and placental growth factor alongside rising soluble fms-like tyrosine kinase-1, is thought to interact with thyroid tissue, which itself expresses angiogenic machinery; elevated TSH has been proposed as a marker of the severity of the underlying vascular and inflammatory process rather than a bystander finding [33]. In this framework, elevated TSH can also promote vascular smooth-muscle contraction in systemic and renal vessels, contributing to diastolic hypertension and reduced tissue perfusion, which ties the thyroid finding directly to the disease's haemodynamic picture.

Set against the wider literature, the TSH finding in this cohort fits a well-replicated pattern. Lao et al. reported reduced free T4 with elevated TSH in preeclamptic pregnancies [53]; Kaya et al. and Basbug et al. described comparable derangements through the 1990s, the

latter also correlating thyroid changes with endothelin [19,43]; and more recent case-control work by Vojodic et al., Dhananjaya et al., and Khader et al. has specifically proposed TSH, rather than T3 or T4, as the more useful discriminator between preeclamptic and normotensive pregnancy, consistent with the present findings [49–51]. This study diverges from part of the literature in the direction of T3 and T4: several published series report T3 falling in preeclampsia relative to controls, whereas this cohort showed a modest T3 and T4 rise in cases at every visit. Other groups, by contrast, have reported significant between-group differences across the full thyroid panel, broadly consistent with the pattern seen here. This inconsistency in T3/T4 direction across studies most likely reflects differences in the gestational timing of sampling, assay methodology (total versus free hormone), population iodine status, and disease-severity mix between cohorts. It argues for TSH, the one parameter that behaves consistently across nearly all cited series, as the more dependable candidate marker until large-scale free-hormone data become available.

Clinically, the practical appeal of TSH lies in its low cost, its wide availability even in secondary-care Indian settings, and its familiarity to obstetricians as a first-trimester screening test in many existing protocols. If the elevation observed here in the third trimester can be shown, in prospective first-trimester cohorts, to precede rather than follow the onset of preeclampsia, TSH could plausibly be incorporated as an adjunct to blood-pressure- and proteinuria-based risk stratification, flagging women for closer surveillance, earlier counselling on aspirin prophylaxis, and more frequent antenatal review. This would be consistent with existing guidance recommending that thyroid screening be considered in early pregnancy, particularly given the overlap between thyroid dysfunction and preeclampsia risk factors [5,31].

Strengths and Limitations

The principal strength of this study is its serial, within-subject design: rather than a single cross-sectional comparison, the thyroid profile was tracked at three time points across the third trimester in the same women, allowing within-arm trajectories to be distinguished from a static between-arm difference. Several limitations temper the conclusions nonetheless. The study was conducted at a single tertiary centre, and the achieved sample of 80 women fell well short of the 150 calculated as necessary by the Cochran formula, with a further imbalance between arms, 20 cases to 60 controls, that limits statistical power, particularly for the T3 and T4 comparisons where between-group separation was already narrow. The observational, non-randomised design cannot establish whether thyroid dysfunction precedes and contributes to preeclampsia or is instead a downstream consequence of the same placental and vascular pathology; only a first-trimester baseline, not available in this dataset, could resolve the direction of that relationship. Antihypertensive treatment in Arm A following diagnosis was not standardised or recorded, which most plausibly explains the observed fall in blood pressure across visits and should be borne in mind when interpreting the blood-pressure trends. Only total T3 and T4 were measured; because total hormone levels are influenced by pregnancy-related changes in thyroxine-binding globulin, free hormone fractions would give a cleaner readout of true thyroid status and are recommended for future work. Thyroid autoantibody status was not assessed, and autoimmune thyroiditis is a recognised confounder of thyroid function testing in pregnancy that could not be excluded here.

Future Directions

Confirmation of these findings requires adequately powered, multicentric cohorts that begin follow-up in the first trimester, measure free T3 and free T4 alongside TSH and thyroid autoantibodies, and report TSH alongside established angiogenic markers such as the soluble fms-like tyrosine kinase-1 to placental growth factor ratio. Such studies should also evaluate whether a defined third-trimester TSH threshold adds discriminative value to existing preeclampsia risk-prediction models, and whether early identification through TSH screening translates into a measurable reduction in maternal or perinatal complications.

Conclusion

Serum TSH was significantly and consistently elevated across the third trimester in women with preeclampsia compared with normotensive controls in this cohort, with a trajectory that changed significantly across gestation in cases but remained stable in controls; T3 and T4 differences, while present in the same direction, were narrower and less discriminating. These findings support consideration of TSH as a low-cost, widely available adjunct marker in preeclampsia risk assessment and surveillance. Confirmation in adequately powered, multicentric cohorts that measure free hormone fractions and begin follow-up in the first trimester is needed before TSH screening can be recommended as a formal component of preeclampsia risk stratification.

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