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Original Research Article

Thyroid dysfunction in pregnancy and its feto-maternal outcome: a prospective observational study

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ABSTRACT

Background: Thyroid disorders constitute the most prevalent endocrine abnormality encountered during pregnancy, with well-documented potential for significant adverse effects on both maternal and fetal health. Physiological adaptations unique to gestation alter thyroid hormone economy, making diagnosis and interpretation of thyroid function tests particularly challenging in this population. The burden of undetected thyroid dysfunction in pregnant Indian women remains incompletely characterised, with most available evidence derived from non-Indian cohorts.

Methods: This prospective observational study enrolled 150 antenatal women attending the Department of Obstetrics and Gynaecology, Dr. D.Y. Patil Hospital, Nerul, Navi Mumbai, over 16 months (March 2023–June 2024). Thyroid function tests (T3, T4, TSH) were performed each trimester using chemiluminescent and ELFA techniques, with trimester-specific TSH reference ranges as per the 2011 American Thyroid Association guidelines. Maternal and feto-neonatal outcomes were documented at delivery.

Results: The prevalence of thyroid dysfunction was 28%, with hypothyroidism accounting for 25.3% (overt 8.7%, subclinical 16.7%) and hyperthyroidism for 2.7%. Thyroid dysfunction was significantly associated with preterm delivery ($p=0.001$), low birth weight ($p=0.001$), and lower APGAR scores at 1 minute ($p=0.015$) and 5 minutes ($p=0.001$). Higher rates of NICU admission ($p=0.012$), stillbirth, IUGR, abortion ($p=0.004$), and miscarriage ($p=0.006$) were observed in women with thyroid disorders. No significant associations were found with maternal age ($p=0.969$) or mode of delivery ($p=0.686$).

Conclusions: Thyroid dysfunction in pregnancy carries a significant burden of adverse feto-maternal outcomes. The high observed prevalence underscores the need for universal thyroid function screening in early pregnancy and the pre-conceptional period to enable timely detection and management.

Keywords: Thyroid dysfunction, Pregnancy, Feto-maternal outcomes, Hypothyroidism, Hyperthyroidism, Preterm delivery, Low birth weight, NICU admission

INTRODUCTION

Thyroid disease ranks as the most prevalent endocrine disorder affecting women of reproductive age, and its impact during pregnancy is of particular clinical importance. Maternal thyroid hormones whether in excess or deficiency influence ovulation and fertility, and exert effects on both maternal physiology and fetal development at every stage of pregnancy.^{1,2}

Structural similarity between thyrotropin (TSH) and human chorionic gonadotropin (HCG) allows HCG to activate TSH receptors in thyroid tissue; the surge in HCG during the first trimester therefore induces a transient rise in thyroxine synthesis accompanied by a reciprocal suppression of TSH, such that a mildly suppressed TSH and slightly elevated free T4 are considered physiologically normal in early pregnancy.³

The vague symptomatology of thyroid dysfunction and the heightened metabolic state of pregnancy frequently result in thyroid disorders going unrecognised in pregnant women. Thyroid function testing is therefore essential not only to establish baseline thyroid status but also to detect subclinical disease that may otherwise escape clinical notice.⁴ Overt and subclinical thyroid dysfunction in pregnancy are associated with a spectrum of maternal complications including preeclampsia, placental abruption, postpartum haemorrhage, and threatened abortion, as well as fetal and neonatal complications such as low birth weight, spontaneous first-trimester abortion, preterm delivery, fetal or neonatal death, neonatal hypothyroidism, and increased perinatal mortality.⁵ The neurodevelopmental consequences are equally sobering: children of mothers with untreated thyroid deficiency (defined as elevated serum TSH) during pregnancy demonstrated IQ scores an average of seven points lower at seven to nine years of age compared to controls.⁶

Despite the well-established global burden of thyroid dysfunction in pregnancy, data specifically pertaining to pregnant Indian women remain limited, with the preponderance of evidence derived from non-Indian populations. Autoimmune-positive euthyroid pregnancies carry a doubled risk of miscarriage and preterm delivery, and worldwide, intrauterine iodine deficiency is estimated to cause neurological sequelae in over 20 million people annually.⁷ Given the paucity of Indian data and the potentially significant public health implications, this study was conducted to determine the prevalence of thyroid dysfunction in pregnant women attending a tertiary care centre and to characterise the associated fetomaternal outcomes.

METHODS

This prospective observational study was conducted in the Department of Obstetrics and Gynaecology, Dr. D.Y. Patil Hospital, Nerul, Navi Mumbai, over a period of 16 months from March 2023 to June 2024, following Ethics Committee approval. The sample size of 150 was calculated using a single proportion formula based on a reference prevalence of 11% (Mahadik et al) at a 5% confidence limit.

All antenatal women irrespective of age, gestational age at presentation, parity, or socioeconomic status with deranged thyroid profiles were included. Women with multiple pregnancies, pre-existing known thyroid disorders, or any other significant comorbidity were excluded. Informed consent was obtained from all participants.

A detailed clinical history was recorded encompassing symptoms of thyroid dysfunction, past and family history of thyroid disease, menstrual and obstetric history, and relevant medical or surgical history. General physical examination including pulse rate and blood pressure, local thyroid examination for structural abnormalities, and signs

of hypo- or hyperthyroidism were elicited. Abdominal and obstetric examination was performed and correlated with ultrasonographic gestational dating. Per vaginal examination was carried out when indicated.

All participants underwent routine antenatal blood investigations in addition to thyroid function tests (T3, T4, TSH) in each trimester using chemiluminescent assay; discrepant values were confirmed with free T3 and free T4 estimation by the ELFA (Enzyme Linked Fluorescent Assay) technique. Trimester-specific TSH reference ranges as recommended by the American Thyroid Association (2011) were applied: 0.1–2.5 $\mu\text{IU/ml}$ (first trimester), 0.2–3.5 $\mu\text{IU/ml}$ (second trimester), and 0.3–3.5 $\mu\text{IU/ml}$ (third trimester); laboratory reference ranges for FT3 (4–8.3 pmol/l) and FT4 (9–20 pmol/l) were also utilised. Subjects were classified as euthyroid (normal TSH), subclinical hypothyroidism (elevated TSH with normal free T4), overt hypothyroidism (elevated TSH with low free T4), or hyperthyroidism (suppressed TSH). Enrolled participants were followed up at every antenatal visit, and pregnancy outcomes including mode of delivery, gestational age at delivery, birth weight, cord TSH, APGAR scores, and NICU admission were recorded. Maternal outcomes assessed included preeclampsia, abortion, miscarriage, anaemia, preterm delivery, and oligohydramnios.

Data were expressed as mean $\pm$ standard deviation and frequencies with percentages. Comparison between groups was performed using the Kruskal-Wallis test for quantitative variables and the Chi-square test for categorical data. A $p < 0.05$ was considered statistically significant. All analyses were carried out using SPSS version 21.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

The present hospital-based prospective observational study enrolled 150 antenatal women to study thyroid function and its fetomaternal outcomes. The following tables summarise the key findings.

The mean maternal age was 29.39 ± 5.49 years (range: 19–39 years), gestational age at first visit was 10.66 ± 3.74 weeks, and mean gestational age at delivery was 37.76 ± 3.01 weeks. The study population was evenly split between primigravida and multigravida women (Table 1).

Thyroid dysfunction was identified in 28% of participants (Table 1). Hypothyroidism was the dominant disorder, collectively accounting for 25.3% of all participants, with subclinical hypothyroidism being more prevalent (16.7%) than overt hypothyroidism (8.7%). Hyperthyroidism was uncommon, affecting 2.7% of the cohort. The majority of participants (72%) were euthyroid.

Among the 144 women who delivered, 22.9% had preterm deliveries (32–36+6 weeks) and 31.3% of neonates were of low birth weight (< 2.5 kg). At 1 minute, 13.2% of

neonates had an APGAR score <7, improving to 6.3% at 5 minutes.

TSH levels demonstrated a progressive increase across trimesters (mean: 2.28 → 2.92 → 3.53 mIU/l), while free T4 remained relatively stable and free T3 showed a slight decline (Table 2). The wide range between minimum and maximum values across all trimester parameters reflects the considerable inter-individual variability in this cohort.

The mean neonatal birth weight was 2703 g and mean cord TSH was 9.05 mIU/l (Table 3). Mean APGAR scores were 7.41 at 1 minute and 7.85 at 5 minutes, confirming overall satisfactory neonatal adaptation.

There was no statistically significant association between thyroid status and maternal age ($p=0.969$) or parity ($p=0.077$), though a trend toward higher hypothyroidism prevalence in multigravida women was noted (65.8% vs. 34.2%) (Table 4). Significant associations were observed with gestational age at delivery ($p=0.001$): preterm delivery occurred in 75% of hyperthyroid and 39.5% of

hypothyroid women, compared to 13.9% of euthyroid women. Birth weight was significantly lower in women with thyroid dysfunction ($p=0.001$), with low birth weight (<2.5 kg) occurring in 75% of hyperthyroid and 66.7% of hypothyroid cases versus 19.6% in euthyroid women. APGAR scores were significantly poorer in neonates of thyroid-dysfunctional mothers at both 1 minute ($p=0.015$) and 5 minutes ($p=0.001$). Mode of delivery did not differ significantly across thyroid status groups ($p=0.686$). Cord TSH was not significantly different between groups ($p=0.939$).

Adverse fetal outcomes including NICU admission ($p=0.012$), low birth weight ($p<0.001$), stillbirth ($p=0.02$), preterm delivery ($p=0.002$), and IUGR ($p=0.01$) were significantly more common in the thyroid dysfunction groups (Table 5). Among maternal outcomes, abortion ($p=0.004$) and miscarriage ($p=0.006$) were significantly higher in women with thyroid dysfunction, particularly those with hypothyroidism. Pre-eclampsia trended toward significance ($p=0.06$), and placental abruption did not occur in any participant.

Table 1: Demographic, thyroid status and obstetric characteristics of study participants (n=150).

Parameter	Category	N	(%)
Age group (years)	<25	47	31.3
	26–30	38	25.3
	31–35	39	26.0
	>36	26	17.3
Parity	Primigravida	75	50.0
	Multigravida	75	50.0
Thyroid status	Normal (euthyroid)	108	72.0
	Subclinical hypothyroidism	25	16.7
	Overt hypothyroidism	13	8.7
	Subclinical hyperthyroidism	2	1.3
	Overt hyperthyroidism	2	1.3
Thyroid diagnosis	Normal	108	72.0
	Hypothyroidism (total)	38	25.3
	Hyperthyroidism (total)	4	2.7
	Preterm (32–36+6 weeks)	33	22.9
Gestational age at delivery	Term (37–40 weeks)	102	70.8
	Post-term (>40 weeks)	9	6.3
	Very low (<1.5 kg)	1	0.7
Birth weight	Low (<2.5 kg)	45	31.3
	Normal (2.5–4 kg)	98	68.1
Mode of delivery	Vaginal	96	64.0
	LSCS	48	32.0

Table 2: Mean values of thyroid parameters across trimesters.

Thyroid parameter	N	Minimum	Maximum	Mean	SD
T1-TSH (mIU/l)	149	0.00	11.00	2.28	1.38
T2-TSH (mIU/l)	144	0.01	36.00	2.92	3.21
T3-TSH (mIU/l)	144	0.00	14.00	3.53	2.26
T1-T4 (pmol/l)	149	0.20	11.30	1.41	1.17
T2-T4 (pmol/l)	144	−0.13	11.13	1.41	1.45
T3-T4 (pmol/l)	144	−0.16	11.04	1.43	1.92

Continued.

Thyroid parameter	N	Minimum	Maximum	Mean	SD
T1-T3 (pmol/l)	149	1.10	23.00	3.96	2.31
T2-T3 (pmol/l)	144	-0.20	21.00	3.81	2.35
T3-T3 (pmol/l)	144	-0.20	16.00	3.43	2.27

Table 3: Mean neonatal and maternal characteristics.

Characteristic	N	Minimum	Maximum	Mean	SD
Birth weight (g)	144	1430	3829	2703.0	488.2
APGAR score at 1 min	144	0	9	7.41	1.63
APGAR score at 5 min	144	0	9	7.85	1.76
Cord TSH (mIU/l)	144	6.0	19.0	9.05	2.14
Maternal age (years)	150	19	39	29.39	5.49
Gestational age at first visit (weeks)	150	7.4	32.0	10.66	3.74
Gestational age at delivery (weeks)	145	10.2	42.0	37.76	3.01

Table 4: Comparison of obstetric and neonatal parameters with thyroid status.

Parameter	Normal (n=108)	Hyperthyroid (n=4)	Hypothyroid (n=38)	p value
Age (years)				
<25	36 (33.3%)	1 (25%)	10 (26.3%)	0.969
26–30	28 (25.9%)	1 (25%)	9 (23.7%)	
31–35	26 (24.2%)	1 (25%)	12 (31.6%)	
>36	18 (16.7%)	1 (25%)	7 (18.4%)	
Parity				
Multigravida	48 (44.4%)	2 (50%)	25 (65.8%)	0.077
Primigravida	60 (55.6%)	2 (50%)	13 (34.2%)	
Gestational age at delivery				
Preterm (32–36+6 weeks)	15 (13.9%)	3 (75%)	15 (39.5%)	0.001
Term (37–40 weeks)	85 (78.7%)	1 (25%)	16 (42.1%)	
Post-term (>40 weeks)	7 (6.5%)	0 (0%)	2 (5.3%)	
Birth weight (kg)				
<1.5 (very low)	0 (0%)	1 (25%)	0 (0%)	0.001
<2.5 (low)	21 (19.6%)	2 (50%)	22 (66.7%)	
2.5–4 (normal)	86 (80.4%)	1 (25%)	11 (33.3%)	
APGAR at 1 min				
<7	9 (8.4%)	1 (25%)	9 (27.3%)	0.015
≥7	98 (91.6%)	3 (75%)	24 (72.7%)	
APGAR at 5 min				
<7	2 (1.9%)	2 (50%)	5 (15.2%)	0.001
≥7	105 (98.1%)	2 (50%)	28 (84.8%)	
Mode of delivery				
LSCS	34 (31.8%)	2 (50%)	12 (36.4%)	0.686
Vaginal	73 (68.2%)	2 (50%)	21 (63.6%)	
Mean gestational age at delivery (weeks)	38.41 (±1.61)	35.55 (±2.53)	36.81 (±2.22)	0.001
Mean birth weight (g)	2825.8 (±427.5)	2022.5 (±507.2)	2387.2 (±473.6)	0.001
Mean APGAR 1 min	7.67 (±8.15)	5.25 (±5.25)	6.82 (±7.18)	0.001
Mean APGAR 5 min	8.15 (±1.29)	5.25 (±3.59)	7.18 (±2.36)	0.001
Mean Cord TSH (mIU/l)	9.01 (±1.8)	9.25 (±3.36)	9.14 (±2.93)	0.939

Table 5: Comparison of fetal-maternal outcomes with thyroid status.

Outcome	Normal (n=108)	Hyperthyroid (n=4)	Hypothyroid (n=38)	p value
Fetal outcomes				
NICU admission	20 (18.5%)	2 (50%)	17 (44.7%)	0.012
Low birth weight	11 (10.2%)	3 (75%)	18 (47.4%)	<0.001
Stillbirth	2 (1.9%)	1 (25%)	3 (7.9%)	0.02
Preterm delivery	6 (5.6%)	2 (50%)	7 (18.4%)	0.002
IUGR	7 (6.5%)	0 (0%)	9 (23.7%)	0.01
Maternal outcomes				
Pre-eclampsia	5 (4.6%)	0 (0%)	6 (15.8%)	0.06
Abortion	1 (0.9%)	0 (0%)	5 (13.2%)	0.004
Miscarriage/MSF	9 (8.3%)	1 (25%)	11 (28.9%)	0.006
Abruption	0 (0%)	0 (0%)	0 (0%)	0.054

DISCUSSION

Thyroid disorders are among the most common endocrine abnormalities in pregnancy, and their management requires an understanding of the complex physiological changes that alter thyroid hormone economy during gestation. This study, conducted in a tertiary care setting in Navi Mumbai, documented a thyroid dysfunction prevalence of 28%, with hypothyroidism constituting the dominant diagnosis (25.3%; overt 8.7%, subclinical 16.7%) and hyperthyroidism accounting for 2.7%. These figures are higher than the 14.3% hypothyroidism prevalence reported by Dhanwal et al from North India and the 12.5% reported by Thanuja et al from South India, though comparable to studies documenting rates of 25.6% abnormal TSH in first-trimester screening.⁸⁻¹⁰ The variation across studies likely reflects differences in iodine adequacy, environmental factors, and diagnostic criteria. Our study did not find a significant association between maternal age and thyroid dysfunction ($p=0.969$), contrasting with the findings of Männistö et al. who reported increased thyroid dysfunction risk with advancing maternal age; the relatively young mean age of our cohort (29.39 years) may explain this discordance.¹¹ A non-significant trend toward higher hypothyroidism prevalence in multigravida women (65.8% vs. 34.2%; $p=0.077$) was observed, aligning directionally with observations by Rajput et al suggesting multiparous women may carry a greater burden of thyroid disorders, an association warranting evaluation in larger cohorts.¹²

Significant associations were identified between thyroid dysfunction and several key perinatal parameters. Preterm delivery (32–36+6 weeks) occurred in 75% of hyperthyroid and 39.5% of hypothyroid women, compared to 13.9% of euthyroid women ($p=0.001$), a finding consistent with the systematic review and meta-analysis by Sheehan et al demonstrating increased preterm birth risk with overt hyperthyroidism.¹³ The likely mechanism involves excess thyroid hormones augmenting uterine contractility and adversely affecting placental function. Low birth weight (<2.5 kg) was observed in 75% and 66.7% of hyperthyroid and hypothyroid neonates

respectively, versus 19.6% in the euthyroid group ($p=0.001$), corroborating the findings of Chen et al who reported increased low birth weight risk in infants of mothers with untreated thyroid disorders.¹⁴ Neonatal APGAR scores were significantly impaired in the thyroid dysfunction groups at both 1 minute ($p=0.015$) and 5 minutes ($p=0.001$), consistent with the findings of Männistö et al and likely reflecting the adverse effects of thyroid hormones on fetal neurodevelopment and cardiovascular function during the antepartum period.¹⁵ Notably, mode of delivery did not differ significantly across thyroid status groups ($p=0.686$), in contrast to Saki et al who reported higher cesarean section rates in thyroid-dysfunctional pregnancies; other obstetric and institutional factors may have modulated operative delivery decisions in our cohort.¹⁶

Adverse fetal-maternal outcomes encompassing NICU admission ($p=0.012$), stillbirth ($p=0.02$), preterm delivery ($p=0.002$), IUGR ($p=0.01$), abortion ($p=0.004$) and miscarriage ($p=0.006$) were all significantly more prevalent in pregnancies complicated by thyroid dysfunction, aligning with the systematic review by Vissenberg et al and the meta-analysis by van den Boogaard et al which collectively highlighted increased pregnancy loss and adverse outcomes in women with thyroid dysfunction and thyroid autoimmunity.^{17,18} Pre-eclampsia showed a trend toward significance ($p=0.06$), with 15.8% of hypothyroid women affected versus 4.6% of euthyroid women, consistent with previously reported associations between subclinical and overt hypothyroidism and hypertensive disorders of pregnancy.

The absence of placental abruption in any participant may reflect the relatively limited sample size, particularly in the hyperthyroid subgroup. Taken together, these findings underscore the imperative for universal thyroid function screening in early pregnancy and at the pre-conceptual visit, as well as for close interdisciplinary monitoring of affected pregnancies throughout gestation and in the postpartum period.

CONCLUSION

This prospective observational study documents a high prevalence of thyroid dysfunction (28%) among pregnant women at a tertiary care centre in Navi Mumbai, with hypothyroidism particularly subclinical hypothyroidism accounting for the majority of cases. Thyroid dysfunction was significantly associated with adverse fetomaternal outcomes including preterm delivery, low birth weight, reduced APGAR scores, NICU admission, stillbirth, IUGR, abortion, and miscarriage. No significant association was identified with maternal age or mode of delivery, though a trend toward higher thyroid dysfunction prevalence in multigravida women was noted. Maternal age, parity, and mode of delivery did not independently predict thyroid dysfunction status in this cohort. The findings reinforce the critical importance of routine thyroid function screening in the pre-conceptional period and early pregnancy to enable timely identification and management of thyroid disorders. Close surveillance, appropriate hormonal replacement or suppression, and a multidisciplinary care approach involving obstetricians, endocrinologists, and neonatologists are essential to mitigate the significant adverse outcomes associated with thyroid dysfunction in pregnancy. Future research should focus on the long-term neurodevelopmental outcomes of offspring exposed to maternal thyroid dysfunction, and on defining the optimal threshold for therapeutic intervention in subclinical hypothyroidism.

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