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Original Research Article

Thyroid stimulating hormone and thyroid peroxidase antibodies assessment in screening of hypothyroidism in pregnancy

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ABSTRACT

Background: Thyroid dysfunction in pregnancy is often underdiagnosed due to overlapping physiological changes, yet it poses significant risks to maternal and foetal outcomes. This study aimed to evaluate role of anti-thyroid peroxidase (TPO) antibodies alongside thyroid stimulating hormone (TSH) in diagnosing and guiding treatment for hypothyroidism in pregnant women.
Methods: A prospective cohort study was conducted from September 2019 to June 2021 at a tertiary hospital in Mumbai among antenatal women at booking visit. TSH, free T4, and anti-TPO antibodies were measured using chemiluminescent immunoassay. Follow-up and treatment were based on trimester-specific reference ranges and standard guidelines. Statistical analysis was done with significance set at $p<0.05$.
Results: Hypothyroidism was found in 11.46% pregnant women (n=253) using TSH cutoff level of 4.0 µIU/ml. The prevalence of anti-TPO antibodies was 5.1%. Among anti-TPO antibody-positive cases, 23.08% had history of miscarriage. Out of 13 participants who tested positive for anti-TPO antibodies, 6 had TSH levels above upper reference limit (4 mIU/l). Participants with borderline TSH levels were followed up; at the second follow-up, 1 out of 72 participants had TSH >4 mIU/l and was started on treatment. At third follow-up, 5 out of 69 participants had TSH >4 mIU/l and were also treated accordingly.
Conclusions: A significant association was observed between elevated TSH levels and presence of anti-TPO antibodies. A notable decrease in mean TSH levels was observed in second and third visits compared to baseline values among pregnant women with borderline TSH, without thyroid hormone supplementation.

Keywords: Hypothyroidism, Pregnancy, TSH

INTRODUCTION

Disorders of the thyroid are relatively common in women of childbearing age and have a significant impact on reproductive function and development of the unborn child. Thyroid disorder is the most common endocrine disorder, after diabetes, in reproductive age.[1] Thyroid hormones are crucial for foetal growth and maturation, particularly of the brain and skeleton. During the critical period of first trimester of pregnancy, maternal thyroxine is essential for foetal development and growth, as it is the only source of thyroid hormone for the foetus, as the foetal thyroid axis matures only by the 12th to 14th gestational week.

Thyroid dysfunction is often overlooked in pregnant women due to nonspecific clinical symptoms and physiological changes in thyroid associated with normal pregnancy. Laboratory diagnosis of thyroid dysfunction during pregnancy is based on serum TSH concentration.

However, its concentration is physiologically lower in pregnant women compared to the non-pregnant population. The use of reference ranges derived from non-pregnant individuals can result in misclassification.[1] Results of multiple international studies point toward creation of trimester-specific reference intervals for TSH in pregnancy. The 2017 American thyroid association (ATA) guidelines recommend continued use of TSH as the primary parameter for evaluating maternal thyroid function and guiding treatment decisions. Given population-level variations in TSH levels, it is advisable for healthcare institutions to establish their own trimester-specific reference ranges using data from healthy, TPO antibody (TPOAb)-negative, and iodine-sufficient pregnant women.[2]

Several determinants contribute to the risk of thyroid dysfunction in pregnancy, including iodine intake, body mass index, ethnicity, and the influence of placental factors such as human chorionic gonadotropin (HCG).[3] Globally, the prevalence of hypothyroidism in pregnancy ranges from 1.5% to 4%, with overt hypothyroidism accounting for 0.3% to 0.5% of cases and the remainder being subclinical. In India, reported prevalence rates vary widely, from 1.2% to 67%.[2]

The presence of TPO antibodies in pregnant women with subclinical hypothyroidism increases the risk of progression to overt hypothyroidism. TPOAb positivity is also associated with several adverse outcomes, including miscarriage, subfertility, preterm delivery, perinatal mortality, large for gestational age infants, low birth weight, and postpartum thyroiditis.[2,4] Both overt and subclinical hypothyroidism can negatively affect maternal and foetal outcomes. Untreated overt hypothyroidism is linked with gestational hypertension, abruptio placentae, anaemia, gestational diabetes, and postpartum haemorrhage. It is also associated with spontaneous miscarriage, premature birth, low birth weight, foetal distress, perinatal death, and impaired neurocognitive development of the foetus. While the evidence is less definitive for subclinical hypothyroidism, some studies suggest it may also contribute to adverse outcomes.[5] A recent U. S. national database study highlighted considerable variation in clinical practices and guidelines regarding the treatment of subclinical hypothyroidism during pregnancy.[6]

Measurement of free T4, alongside TSH, is useful to differentiate overt from subclinical thyroid dysfunction. During early gestation, free T4-rather than total T4-has been shown to correlate with adverse maternal and foetal outcomes.[7] Therefore, accurate assessment using both TSH and free T4 is essential in comprehensive evaluation. Early diagnosis and treatment of hypothyroidism in pregnancy is crucial. Initiation of levothyroxine therapy should not be delayed, as irreversible effects on foetal brain development may occur after the 14th week of gestation due to insufficient thyroid hormone exposure. [8]

The aim of this study is to determine the role of anti-TPO antibodies along with TSH in the diagnosis and management of hypothyroidism in pregnant women. It also seeks to evaluate fluctuations in TSH levels and assess the need for thyroid hormone supplementation during pregnancy.

METHODS

This prospective cohort study was conducted in the department of obstetrics and gynaecology at Dr. Babasaheb Ambedkar memorial hospital (tertiary care public hospital), central railway, Byculla, Mumbai, from September 2019 to June 2021, after obtaining ethical clearance from the institutional ethics committee.

All antenatal women presenting for their booking visit at the antenatal care clinic were considered for inclusion in the study. Women who gave written informed consent were enrolled consecutively. Those with pregestational hypothyroidism already on thyroid supplementation and those with TSH levels in the hyperthyroid range (<0.1 mIU/L) were excluded. A detailed history and clinical examination were conducted using a predesigned and pretested proforma. Gestational age was calculated based on the last menstrual period and confirmed by ultrasonography. Relevant investigations were carried out as per hospital protocol, and blood samples were collected in the outpatient department.

TSH, free T4, and anti-TPO antibody levels were measured using chemiluminescent magnetic microparticle immunoassay technology. Women with serum TSH levels >4.0 mIU/l (pregnancy-specific upper reference limit for hypothyroidism treatment used in this study) in any trimester were started on treatment and excluded from further follow-up. Participants with TSH levels in the borderline range-2.5 to 4.0 mIU/L in the first trimester and 3.0 to 4.0 mIU/l in the second and third trimesters-underwent further testing for free T4 and anti-TPO antibodies. The trimester-specific reference ranges for free T4 were: 0.8-1.2 ng/dl (first trimester), 0.6-1.0 ng/dl (second trimester), and 0.5-0.8 ng/dl (third trimester). Women in the borderline TSH group who had either elevated anti-TPO antibody levels (biological reference interval ≤5.61 IU/ml) or low serum free T4 levels were treated and excluded from further follow-up.

Those with borderline TSH levels but normal free T4 and negative anti-TPO antibodies were followed up after four weeks. If TSH remained within the borderline range, they were observed without treatment and reassessed with repeat TSH testing at 28 weeks of gestation. Participants with TSH levels >4.0 mIU/l at any point were started on treatment.[9]

Treatment was administered according to the 2017 ATA guidelines and recent ITS and FOGSI 2019 recommendations. [8]

Sample size

As per a previous study titled "High prevalence of subclinical hypothyroidism in pregnant women in South India", the prevalence of hypothyroidism was 19.41% (say p). Sample size was calculated using the formula, $n=Z^2\times p(1-p)/d^2$; where, n=sample size, proportion (p)=0.1941, margin of error (d)=0.05, α is the level of significance i.e. 5%, Z is the standard normal variate value at α is 1.96.[10,11] The sample size was calculated to be 243. After accounting for 5% loss due to non-response or incomplete data, final sample size was adjusted as 253.

Statistical analysis

The study included qualitative and quantitative variables. Qualitative variables were presented as frequencies and percentages, while quantitative variables were expressed as mean±SD. Normality of quantitative data was checked using the Shapiro-Wilk test; and p>0.05 indicated that data followed a normal distribution.

Descriptive statistics were used to summarize distribution of patients based on age, clinical features, baseline characteristics, previous and family history, TSH status or anti-TPO status. Inter-group comparison between mean values was done using unpaired t-test (for two groups) or one-way ANOVA (for more than two groups) at a time-point. Chi-square test was applied to measure the association between categorical variables such as TSH and anti-TPO status. P<0.05 considered statistically significant. Data entry done using MS excel, and statistical analysis was performed using GraphPad InStat v3.0.

RESULTS

A total of 253 participants were enrolled in the present study. These participants were categorized into three groups based on their serum TSH levels, considering the ongoing debate regarding the threshold at which treatment should be initiated during pregnancy. Group A includes participants with TSH levels less than 2.5 mIU/l if measured in the first trimester and less than 3 mIU/l if measured in the second or third trimester, which is considered the normal range. Group B consists of those with TSH levels greater than or equal to 2.5 mIU/l in the first trimester or greater than or equal to 3 mIU/l in the second or third trimester but less than or equal to 4 mIU/l, representing the borderline range according to pregnancy-specific guidelines. Group C comprises participants with TSH levels above 4 mIU/l, exceeding the upper reference limit and thus requiring treatment.

The majority of participants (79.4%) were between 21 and 30 years of age. The highest proportion of participants, across all TSH subgroups, belonged to the lower-middle socioeconomic class. There was a statistically significant association between socioeconomic status and TSH levels (p<0.05). There was also a significant relationship between regular menstrual cycles and normal TSH levels (p<0.05). In addition, a significant association was observed between family history of thyroid disease and elevated TSH levels (p<0.05) (Table 1).

At 1st antenatal visit, of total participants, 147 had TSH levels within normal range, 77 fell in borderline category (Group B), and 29 had TSH levels greater than 4 mIU/L (Group C), who were initiated on treatment (Table 2).

With respect to the follow-up data of participants who had borderline TSH levels at the first visit, 72 out of 77 were available for assessment at second follow-up, with 5 participants excluded from the study. At this stage, one participant exhibited a rise in TSH above 4 mIU/l and was initiated on treatment. At 3rd follow-up, data available for 69 of these 72 participants. Of them, 5 experienced an increase in TSH levels beyond 4 mIU/l and were subsequently started on treatment (Table 3 and 4).

TSH trends among participants who initially presented with borderline TSH levels were observed. Of the 72 participants, 62 (86.11%) demonstrated a decrease in TSH levels by the second follow-up visit. Ten participants showed an increase, but their TSH levels remained below 4 mIU/l. At 3rd visit, when compared to 2nd, 40.57% of participants experienced a further decrease in TSH levels, 52.17% showed an increase that still remained below treatment threshold, and 5 participants had TSH levels that rose above 4 mIU/l (Figure 1 and 2).

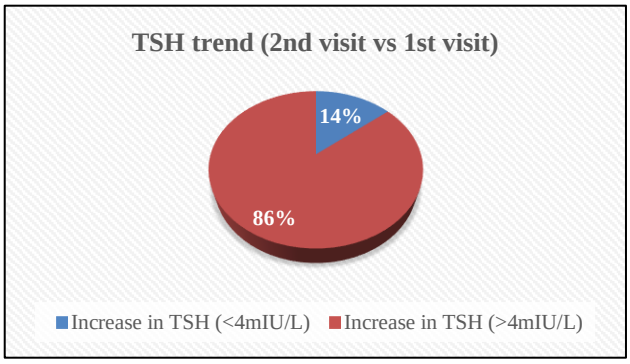

Figure 1: TSH increase in group B subjects from 1st to 2nd visit (<4 mIU/l vs >4 mIU/l).

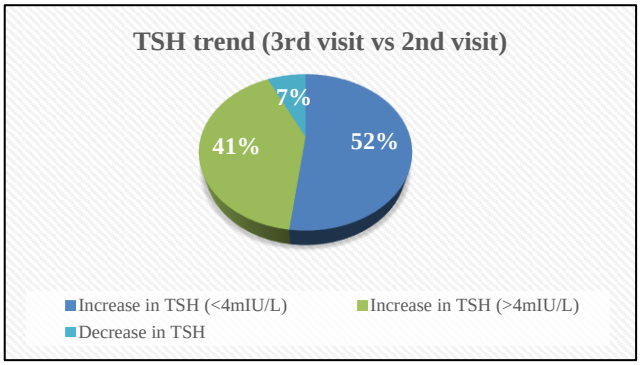

Figure 2: TSH trend in group B from 2nd to 3rd visit (<4 mIU/l vs >4 mIU/l vs decrease).

With respect to association between anti-TPO antibodies and TSH levels, a statistically significant relationship is noted between elevated TSH levels (>4 mIU/l) at the first visit and raised anti-TPO antibody levels ($p<0.05$).

Among the 13 participants who tested positive for anti-TPO antibodies, 7 had TSH levels below 4 mIU/l at baseline. During follow-up, only one of these participants developed TSH levels above 4 mIU/l, while TSH levels remained below 4 mIU/l in four cases; the remaining two participants did not follow up (Table 5).

No statistically significant association was found between anti-TPO antibody status and history of miscarriage ($p>0.05$).

However, 23.08% of anti-TPO antibody-positive participants reported a history of miscarriage, compared to 10.84% of those who were anti-TPO antibody negative (Table 6).

Distribution of free T4 levels among participants in the borderline TSH group at the first visit reveals that 3.8% of these participants had low free T4 levels and were therefore started on treatment (Figure 3).

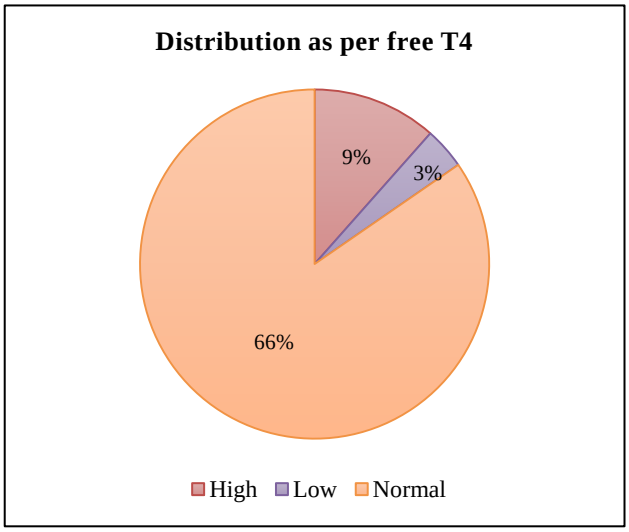

Figure 3: TSH trend in group B from 2nd to 3rd visit (<4 mIU/l vs >4 mIU/l vs decrease).

Table 1: Socio-demographic and baseline characteristic of study participants and their correlation with TSH.

Study variables	First visit TSH			Chi-square test (p value)
	Group A (<2.5 mIU/l/ <3 mIU/l	Group B (<2.5-4 mIU/l/ 3-4 mIU/l)	Group C (>4 mIU/l)	
Age (in years)				
≤20	9 (6.12%)	4 (5.19%)	3 (10.34%)	0.12
21-30	113 (76.87%)	66 (85.71%)	22 (75.86%)	
31-40	25 (17%)	7 (9.09%)	3 (10.34%)	
>40	0 (0%)	0 (0%)	1 (3.44%)	
BMI (kg/m^2)				
Normal	96 (67.13%)	54 (70.12%)	22 (75.86%)	0.49
Obese	11 (7.69%)	7 (9.09%)	4 (13.79%)	
Overweight	34 (23.77%)	14 (18.18%)	3 (10.34%)	
Underweight	6 (4.19%)	2 (2.59%)	0 (0%)	
Socio-economic status				
Lower	1 (0.68%)	0 (0.0%)	2 (6.895)	0.01
Upper lower	65 (44.21%)	43 (55.84%)	16 (55.17%)	
Lower middle	3 (2.04%)	4 (5.19%)	0 (0%)	
Upper middle	41 (27.89%)	13 (16.88%)	6 (20.68%)	
Upper	37 (25.17%)	17 (22.07%)	5 (17.24%)	
Menstrual cycle				
Irregular	8 (5.54%)	9 (11.69%)	7 (24.13%)	0.002
Regular	139 (94.55%)	68 (88.31%)	22 (75.87%)	
Infertility status				
Infertile	3 (2.05%)	3 (3.9%)	1 (3.45%)	0.51
Fertile	144 (97.95%)	74 (96.1%)	28 (96.55%)	
Miscarriage history				
Present	21 (14.28%)	6 (7.80%)	2 (6.90%)	0.25
Absent	126 (85.72%)	71 (92.20%)	27 (93.10%)	
Family history of thyroid disease				
Yes	3 (2.05%)	7 (9.1%)	1 (3.45%)	0.04
No	144 (97.95%)	70 (90.9%)	28 (96.55%)	

Table 2: Distribution of study participants according to TSH level in first visit.

Total participants at first visit	Participant distribution as per TSH range		
	Group A (<2.5 mIU/l/<3 mIU/l	Group B (<2.5-4 mIU/l/3-4 mIU/l)	Group C (>4 mIU/l)
253 (100%)	147 (58.1%)	77 (30.43%)	29 (11.46%)

Table 3: Distribution of study participants on follow-up according to TSH level in second visit.

Total participants at second visit	Participant distribution as per TSH range (number of participants with TSH in the range of group B at 1st visit=72)		
	Group A (<2.5 mIU/l/<3 mIU/l	Group B (<2.5-4 mIU/l/3-4 mIU/l)	Group C (>4 mIU/l)
72 (100%)	37 (51.38%)	34 (47.22%)	1 (1.38%)

Table 4: Distribution of study participants on follow-up according to TSH level in third visit

Total participants at third visit	Participant distribution as per TSH range (number of participants with TSH in the range of group B at 1st visit=69)		
	Group A (<2.5 mIU/l/<3 mIU/l	Group B (<2.5-4 mIU/l/3-4 mIU/l)	Group C (>4 mIU/l)
69 (100%)	44 (51.38%)	20 (28.98%)	5 (7.24%)

Table 5: Association of anti-TPO antibody with serum TSH.

Anti-TPO antibody level	First visit TSH			Total	P value
	Group A	Group B	Group C		
Normal	141 (95.91%)	76 (98.71%)	23 (79.31%)	240 (94.9%)	0.01
Raised	6 (4.09%)	1 (1.29%)	6 (20.69%)	13 (5.1%)	

Table 6: Association of TPO with miscarriage history and autoimmune disease.

Parameters		First visit TPO		P value
		Anti-TPO antibody positive	Anti-TPO antibody negative	
Miscarriage history	Present	3 (23.08%)	26 (10.84%)	0.36
	Absent	10 (76.92%)	214 (89.16%)	
Autoimmune disease	Present	0 (0%)	5 (2.09%)	0.59
	Absent	13 (100%)	23 (97.91%)	

DISCUSSION

Hypothyroidism is the most common thyroid disorder in pregnancy, affecting 3-5% of pregnant women. Subclinical hypothyroidism is more prevalent than overt hypothyroidism and is typically defined by a serum TSH level above the pregnancy-specific reference range->2.5 mIU/l in the first trimester and >3 mIU/l in the second and third trimesters. Some sources define subclinical hypothyroidism as a TSH between 5-10 mIU/l and overt hypothyroidism as TSH >10 mIU/l. Once overt hypothyroidism is diagnosed, levothyroxine therapy should be promptly initiated to normalize TSH levels.

However, treatment re-commendations for the sub-clinical hypo-thyroidism vary among professional bodies due to inconsistent evidence on maternal as well as the foetal benefits.[9]

There is considerable geographic variation in the prevalence of hypothyroidism during pregnancy, with higher rates reported in Asian countries compared to the West.[12] In India, studies report a prevalence ranging from 4.8% to 11% among pregnant women.[13] Dhanwal et al found a prevalence of 13.13% (n=388) using a TSH cutoff of 4.5 µIU/ml, which increased further when applying the American thyroid association (ATA) 2011 guidelines (>2.5 µIU/ml in the first trimester and >3.0 µIU/ml later in pregnancy).[13] In our study, using the ATA 2017 guideline cutoff of 4 mIU/L, the prevalence was 11.46%, but rose to 41.89% when the >2.5 and >3.0 µIU/ml thresholds were applied-similar to findings from other Indian studies. Prasad et al reported a 14.3% prevalence among pregnant women at a tertiary public hospital in Delhi.[12] Factors contributing to the higher prevalence in India include low dietary iodine intake, goitrogens in the diet, and deficiencies of micronutrients such as selenium and iron.[10] The possible interrelation of hypothyroidism with other

major endocrinopathies also warrants further investigation.[10]

In India, iodine deficiency is the most common cause of hypothyroidism, leading to a lower prevalence of anti-TPO antibodies compared to Western countries, and thus autoimmune thyroid disorders are less frequently observed.[14] In our study, raised anti-TPO antibodies were detected in 13 cases (5.1%) at the first visit, which is lower than in other studies; for instance, Bhattacharya et al. reported 14.28% positivity. A significant association was observed between raised TSH (above the upper reference limit) and elevated anti-TPO antibody levels ($p<0.05$), similar to findings by Bajaj et al who found TPO positivity in 57.1% of subclinical hypothyroid cases and only 7% in euthyroid women.[13] Glinoer et al showed that 20% of initially euthyroid TPOAb-positive women developed TSH >4 mU/l during pregnancy, suggesting the need for increased surveillance.[4,15] However, in our study, of 13 anti-TPO-positive participants, 7 had TSH <4 mIU/l initially, and only one developed TSH >4 mIU/l on follow-up, while 4 remained below 4 and 2 were lost to follow-up.

We also found that 23.08% of anti-TPO-positive women had a history of miscarriage, compared to 10.84% in TPO-negative women, suggesting a potential link between TPO positivity and poor maternal-foetal outcomes. This aligns with Rajput et al study, where 12% of TPO-positive and 3.3% of TPO-negative women had miscarriages ($p<0.004$), and TPO-Ab-positive women had significantly higher preterm deliveries (26.8% vs. 8.0%, $p=0.01$).[16] TSH levels fluctuate during pregnancy, especially in the first trimester, and using non-pregnant reference ranges can lead to misdiagnosis.[17] TSH levels also show diurnal variation, further complicating interpretation.[3]

According to the 2011 ATA guidelines, TSH upper limits were set at 2.5 mU/l (first trimester) and 3.0 mU/l (later trimesters). However, studies indicate substantial population variations in TSH norms, influenced by iodine status, assay methods, BMI, geography, and ethnicity.[8] The 2017 ATA guidelines recommend institution-specific, trimester-specific TSH reference ranges, ideally in healthy, TPOAb-negative, iodine-sufficient pregnant women. If not feasible, an upper limit of ~4.0 mU/l is acceptable.[18] This variation in reference ranges has sparked debate on whether to treat women with borderline TSH levels (≥2.5 in the first trimester or ≥3.0 in later trimesters but ≤4.0 mIU/l).[19]

In our study, 72 women with borderline TSH at first visit were re-evaluated. At second visit, 51.38% had normal TSH, 47.22% remained borderline, and only one exceeded 4 mIU/l. By third visit, 63.76% had normalized TSH, 20 remained borderline, and 5 (7.24%) exceeded 4 mIU/l. Overall, 86.11% of the borderline cases had a decrease in TSH by the second visit, and by the third visit, 40.57% showed further decrease, 52.17% showed an increase below 4 mIU/L, and 5 exceeded 4 mIU/l. This indicates that most borderline cases normalize without treatment, highlighting the risk of overtreatment based on a single mildly elevated TSH value.[8] Treatment should be considered in borderline TSH cases only when accompanied by raised anti-TPO antibodies or reduced free T4, due to their association with adverse pregnancy outcomes.[4,7] Due to resource constraints, free T4 testing was limited to selected cases.

We also analysed associations between TSH levels and participant characteristics. A significant association was observed between abnormal TSH and lower socioeconomic status ($p<0.05$). Irregular menstrual history was also significantly associated with raised TSH ($p<0.05$), whereas infertility showed no significant correlation ($p>0.05$). A strong association was found between borderline TSH and a family history of thyroid disease ($p<0.05$): 9.1% of borderline cases, 3.45% of cases with TSH >4 mIU/l, and only 2% of normal participants had such a history. These findings are supported by Mahadik et al who found 22.7% of hypothyroid women had irregular menstrual cycles, and Wang et al who reported a 12.7% prevalence of family history in hypothyroid pregnant women.[1,20]

This study underscores the critical role of both TSH and anti-TPO antibodies in the early detection and clinical management of hypothyroidism during pregnancy. By employing TSH reference ranges according to trimester and integrating anti-TPO antibody testing, the research highlights the potential to refine diagnostic accuracy and prevent overtreatment in borderline cases. The observation that the majority of pregnant women with mildly elevated TSH levels normalized without intervention suggests a more conservative approach may be appropriate in the absence of anti-TPO positivity or low free T4. Furthermore, the significant association between elevated TSH and anti-TPO antibodies reinforces the relevance of autoimmune screening in antenatal care. Overall, this study contributes to evolving evidence supporting individualized, context-specific thyroid screening protocols in pregnancy, particularly in iodine-deficient populations, to improve maternal and foetal outcomes while avoiding unnecessary therapy.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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