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Original Research Article

Maternal urinary iodine status during antepartum and postpartum periods and its association with neonatal TSH: a hospital-based study from eastern India

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ABSTRACT

Background: Iodine requirement increases during pregnancy and early postpartum, and inadequate iodine intake may adversely influence neonatal thyroid function. Region-specific data from eastern India remain limited. Objective was to assess maternal urinary iodine concentration (UIC) during antepartum and postpartum periods and evaluate its association with neonatal thyroid-stimulating hormone (TSH).

Methods: This hospital-based prospective observational study (December 2024 to April 2025) included 70 mother-neonate dyads. Maternal midstream urine samples were collected during antepartum and postpartum periods, and UIC was measured using the Sandell-Kolthoff method. Neonatal screening samples were obtained on day 3-5 of life, and TSH and free T4 (fT4) were measured by chemiluminescent immunoassay. Maternal iodine status was categorized using WHO pregnancy cut-offs. Associations between maternal UIC and neonatal thyroid parameters were evaluated using Spearman's correlation. Receiver operating characteristic (ROC) analysis was performed to assess the ability of maternal UIC to predict neonatal TSH >5 mIU/l.

Results: Mean antepartum and postpartum UIC were 255.6±78 µg/l and 249.6±82 µg/l, respectively. Iodine deficiency (<150 µg/l) was observed in 18.6% (antepartum) and 22.9% (postpartum) of mothers. Mean neonatal TSH was 4.44±3.1 mIU/l and mean neonatal fT4 was 3.04±1.1 ng/dl; 47.1% of neonates had TSH ≥5 mIU/l. Antepartum UIC showed a significant inverse correlation with neonatal TSH ($\rho=-0.625$, $p<0.001$), and postpartum UIC also correlated inversely with neonatal TSH ($\rho=-0.503$, $p<0.001$). No significant association was observed between maternal UIC and neonatal fT4. Antepartum UIC demonstrated good predictive performance for neonatal TSH>5 mIU/l (AUC=0.830) at a cut-off of 222.8 µg/l (sensitivity 69.7%, specificity 94.6%).

Conclusions: Maternal UIC during both antepartum and postpartum periods is inversely associated with neonatal TSH, highlighting the influence of maternal iodine status on neonatal thyroid function. Despite overall iodine sufficiency, a subset of mothers remained iodine-deficient, underscoring the need for continued monitoring of iodine nutrition during pregnancy and early postpartum.

Keywords: Iodine deficiency, Neonatal TSH, Newborn screening, Postpartum period, Pregnancy, Urinary iodine concentration

INTRODUCTION

Iodine is an essential micronutrient required throughout life, particularly during prenatal development and early infancy. It is crucial for the synthesis of thyroid hormones, which regulate metabolism and are vital for the proper functioning of the brain, liver, and kidneys. A healthy adult body contains approximately 15-20 mg of iodine, with nearly 70-80% localized in the thyroid gland.[1] Adequate iodine levels are indispensable for thyroid hormone production.[2]

Iodine deficiency results in a spectrum of conditions collectively known as iodine deficiency disorders (IDD), which includes goitre, hypothyroidism, cretinism, miscarriages, stillbirths, and various degrees of neurodevelopmental impairment such as hearing and speech disabilities, learning difficulties, and psychomotor delays. It is recognized as the leading cause of preventable brain damage globally.[3,4] During pregnancy, iodine requirements increase significantly by approximately 50% to meet the heightened demand for maternal and foetal thyroid hormone synthesis.[5] Maternal iodine is actively transferred to the foetus, and deficiency during this period and is associated with adverse outcomes, including impaired growth, neurocognitive dysfunction, and increased infant mortality.[6,7]

Thyroid hormone synthesis is tightly regulated by a hypothalamic-pituitary-thyroid axis through a negative feedback loop. Thyrotropin-releasing hormone (TRH) from the hypothalamus stimulates the anterior pituitary to secrete thyroid-stimulating hormone (TSH), which in turn prompts the thyroid gland to produce thyroxine (T4) and triiodothyronine (T3) using iodine as a key substrate. Elevated T4 and T3 levels inhibit further TSH release, while low levels stimulate its secretion.[8] In cases of iodine excess, the Wolff-Chaikoff effect transiently inhibits thyroid hormone biosynthesis as a protective mechanism.[9]

Foetal thyroid function begins at approximately the 10th week of gestation, with iodine uptake and T4 synthesis. However, maternal thyroid hormone remains the primary source until around the 20th gestational week. By the 34th week, the foetal hypothalamus becomes functionally mature, producing endogenous TRH and enabling the foetus to overcome the Wolff-Chaikoff effect. Post-delivery, neonatal thyroid function becomes independent of maternal regulation.[5,10] Significant reductions in maternal TSH have been observed during the first trimester, especially in twin pregnancies.[11] A neonatal TSH value >5 mIU/l in more than 3% of screened newborns indicates population-level iodine deficiency and has also been used as an indicator for monitoring iodine nutrition and evaluating the effectiveness of iodine deficiency control programmes.[12-14]

Urinary iodine concentration (UIC) is a reliable biomarker for assessing iodine status, as approximately 90% of dietary iodine is excreted in urine. Under normal physiological conditions, urinary iodine excretion closely reflects the dietary intake.[15]

According to WHO criteria, a median UIC of 150-249 µg/l indicates adequate iodine intake in pregnant women, whereas <150 µg/l indicates iodine deficiency.[16] Globally, more than 2 billion people are estimated to be iodine deficient, with a high burden in South Asia.[17] In India, iodine deficiency remains a concern due to the widespread consumption of food grown in iodine-depleted soil.[18] A joint UNICEF-GAIN (The Global Alliance for Improved Nutrition) report estimates that 19 million babies are born each year at risk of iodine deficiency-induced brain damage, with 4.3 million of these in South Asia.[19] At the national level, data from 2018-19 revealed that the median UICs in Indian pregnant, lactating, and non-pregnant non-lactating women were 173.4 µg/l, 172.8 µg/l, and 178.0 µg/l respectively indicative of adequate iodine nutrition. However, a study in eastern India reported a severe endemic goitre prevalence of 33.1%.[20,21]

Despite the susceptibility of the Indian population to IDD, particularly among pregnant and lactating women, there is a paucity of region-specific data from eastern India. Given the vulnerability of these groups and the critical role of iodine in neonatal thyroid function, there is an urgent need to evaluate iodine status in this region. This study, therefore, aimed to estimate maternal urinary iodine concentrations during the antepartum and postpartum periods and correlate these levels with neonatal TSH values, thereby addressing a critical public health gap in eastern India.

METHODS

Study design and setting

This hospital-based prospective observational study was conducted in the department of biochemistry, in collaboration with the department of obstetrics and gynecology, Kalinga Institute of Medical Sciences (KIMS), and the Disease Biology Laboratory, KIIT School of Biotechnology, Bhubaneswar, Odisha, India.

Study population

Pregnant women aged 18-45 years admitted to the labour ward and delivering a live-born neonate between 1st December 2024 and 30th April 2025 were eligible. To minimize confounding, women with high-risk pregnancies, chronic systemic illnesses, known thyroid disorders, malnutrition, use of thyroid-interfering drugs, or foetal structural anomalies detected on antenatal ultrasound were excluded.

Sample size

Sample size was calculated for correlation analysis using Fisher's z-transformation (two-sided α=0.05; power =80%), assuming an expected correlation of r=0.30

between maternal urinary iodine concentration and neonatal TSH, yielding a required sample of approximately 85. The final analysis included 70 mother-neonate dyads with complete paired UIC and neonatal thyroid data, providing adequate power for the moderate-to-strong associations observed.

Data collection

Maternal sociodemographic and clinical information was collected using a structured proforma, including medical and obstetric history and maternal thyroid status (serum TSH where available). Blood pressure, height, and weight were recorded using standard procedures. Delivery details (mode of delivery, gestational age) and neonatal outcomes were obtained from medical records. Neonatal clinical data were recorded from the postnatal ward/NICU, and neonatal thyroid screening values were captured between day 3 and day 5 of life.

Sample collection and biochemical analysis

Maternal blood

After an overnight fast, venous blood was collected into appropriate vacutainers. Serum was separated by centrifugation and stored at −80°C until analysis. Maternal TSH, free T4 (fT4), serum sodium (Na^+), potassium (K^+), creatinine, and glucose were measured using standard laboratory protocols.

Neonatal blood

Neonatal screening samples were obtained via heel-prick on the 3rd-5th day of life. Neonatal TSH and fT4 were measured using chemiluminescent immunoassay on the VITROS 5600 autoanalyzer, with intra- and inter-assay coefficients of variation <5%.

Maternal urine sample collection

Midstream urine samples were collected in sterile containers during the antepartum and postpartum periods. Samples were labelled and stored at −80°C until estimation. Urinary iodine concentration (UIC) was measured using the Sandell-Kolthoff reaction, a catalytic colorimetric method for iodide quantification after appropriate standardization.[22]

All biochemical analyses were performed in the central laboratory following internal quality control procedures.

Ethical considerations

The study was conducted according to the Declaration of Helsinki. Institutional ethics committee approval was obtained before initiation. Written informed consent was taken from all participants after explaining the study objectives, procedures, confidentiality measures, and the right to withdraw at any time.

Statistical methodology

Data were analysed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables [e.g., urinary iodine concentration (UIC), neonatal TSH, maternal serum sodium, systolic blood pressure, and gestational age] were summarized as mean±standard deviation (SD) for approximately normally distributed data and as median (IQR) where distributions were skewed. Categorical variables were expressed as frequencies and percentages. Data normality was assessed using the Shapiro-Wilk test. For normally distributed data, t-tests were used, whereas Mann-Whitney U tests were applied for non-normally distributed data where appropriate. The relationship between maternal UIC (both antepartum and postpartum) and neonatal thyroid parameters (TSH and fT4) was assessed using Pearson's correlation for parametric data and Spearman's rank correlation for non-parametric distributions. Maternal UIC was categorized using WHO pregnancy cut-offs as insufficient (<150 µg/l), adequate (150-249 µg/l), and above requirement (≥250 µg/l) and presented as n (%).[16] For descriptive reporting of neonatal thyroid screening distribution, neonatal TSH values were categorized into <5 mIU/l, 5-10 mIU/l, and ≥10 mIU/l, and presented as N (%). Where applicable, chi-square tests were used to compare categorical distributions across groups. Additionally, ROC analysis was performed to evaluate the predictive performance of maternal UIC for neonatal TSH >5 mIU/l, with optimal cut-offs selected using the Youden index.[23] A two-sided $p<0.05$ was considered statistically significant.

RESULTS

A total of 70 participants were included in the analysis. Baseline maternal and neonatal characteristics are summarized in Table 1. The mean maternal age was 29.3±5.8 years, and the mean gestational age at delivery was 39.5±1.8 weeks. Maternal biochemical parameters and neonatal anthropometric indices were within expected ranges.

Maternal urinary iodine concentration (UIC) during antepartum and postpartum periods is presented in Table 2. The mean antepartum UIC was 255.6±78 µg/l and postpartum UIC was 249.6±82 µg/l, with comparable median values. Overall, UIC values remained relatively stable across the peripartum period. Based on WHO criteria, the majority of women had UIC≥250 µg/l, indicating iodine intake above requirement, although a subset remained iodine deficient in both periods. The distribution of UIC is illustrated in Figure 1.

Table 1: Baseline maternal and neonatal characteristics of the study population.

Variables	Mean±SD
Maternal age (years)	29.3±5.8
Gestational age at delivery (weeks)	39.5±1.8
Systolic BP (mmHg)	125.1±10.2
Diastolic BP (mmHg)	75.58±10.84
Maternal serum sodium (mmol/l)	135.1±3.2
Maternal potassium (mmol/l)	4.25±0.42
Creatinine (mg/dl)	0.52±0.11
Random blood sugar (mg/dl)	95.47±18.4
Neonatal birth weight (kg)	2.88±0.45
Birth length (cm)	50±2
Head circumference (cm)	33±1.5
APGAR score	7±1

Values are presented as mean±standard deviation (SD) unless otherwise stated. BP- blood pressure; APGAR- appearance-pulse-grimace-activity-respiration.

Table 2: Maternal urinary iodine concentration (UIC) during antepartum and postpartum periods and distribution according to WHO criteria.

Parameters	Antepartum UIC (µg/l)	Postpartum UIC (µg/l)
Mean±SD	255.6±78	249.6±82
Median	263	250
Range (minimum-maximum)	22.7-349	90-360
Category	**Antepartum N (%)**	**Postpartum N (%)**
Deficient (<150 µg/l)	13 (18.6)	16 (22.9)
Adequate (150–249 µg/l)	16 (22.9)	26 (37.1)
Above requirement (≥250 µg/l)	41 (58.6)	28 (40.0)

Values expressed as mean±SD and median (minimum-maximum). UIC categorized as per WHO criteria for pregnancy: <150 µg/l (deficient), 150-249 µg/l (adequate), ≥250 µg/l (above requirement).

Table 3: Neonatal thyroid profile and TSH distribution.

Parameters	Mean±SD	Median (IQR)	Range
Neonatal TSH (mIU/l)	4.44±3.1	3.70 (2.21-7.19)	0.97-13.17
Neonatal fT4 (ng/dl)	3.04±1.1	3.02 (2.26-4.03)	1.12-6.27
Neonatal TSH category		**N (%)**	
<5 mIU/l		37 (52.9)	
5-10 mIU/l		29 (41.4)	
≥10 mIU/l		4 (5.7)	

Values are expressed as mean±standard deviation (SD), median (interquartile range), and range (minimum-maximum). IQR = interquartile range (25^{th}-75^{th} percentile). Neonatal TSH categories are based on screening cut-offs: <5 mIU/l (normal), 5-10 mIU/l (borderline), and ≥10 mIU/l (elevated). Percentages are calculated based on the total sample size (n=70). TSH = thyroid-stimulating hormone; fT4 = free thyroxine.

Table 4: Correlation analysis of maternal factors, maternal iodine status, and neonatal thyroid parameters.

Variables	Antepartum UIC (µg/l)	Postpartum UIC (µg/l)	Neonatal TSH (mIU/l)	Neonatal fT4 (ng/dl)
Antepartum UIC (µg/l)	1.000	0.729 (p<0.001)	−0.625 (p<0.001)	0.195 (p=0.105)
Postpartum UIC (µg/l)	0.729 (p<0.001)	1.000	−0.503 (p<0.001)	0.168 (p=0.164)
Neonatal TSH (mIU/l)	−0.625 (p<0.001)	−0.503 (p<0.001)	1.000	−0.161 (p=0.182)
Neonatal fT4 (ng/dl)	0.195 (p=0.105)	0.168 (p=0.164)	−0.161 (p=0.182)	1.000

Spearman's rank correlation coefficients (ρ) with two-tailed p values are shown (n=70). UIC = urinary iodine concentration; TSH = thyroid-stimulating hormone; fT4 = free thyroxine. Statistical significance was set at p<0.05.

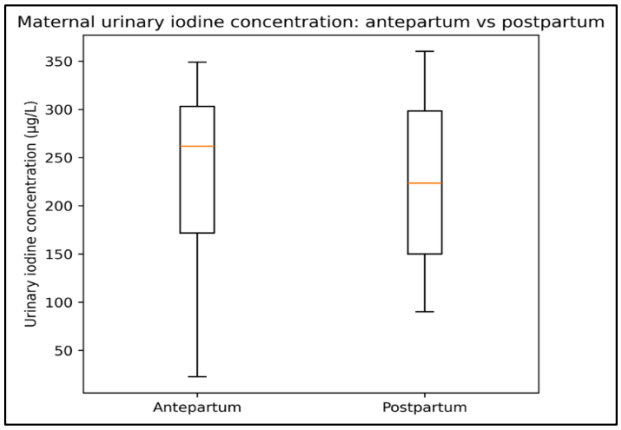

Figure 1: Box plot showing maternal urinary iodine concentration (UIC) in antepartum and postpartum periods.

Box plots showing the distribution of maternal urinary iodine concentration (UIC) in antepartum and postpartum periods (n = 70). The central line represents the median, the box indicates the interquartile range (IQR), and whiskers extend to 1.5×IQR. Data points beyond the whiskers, if present, represent outliers.

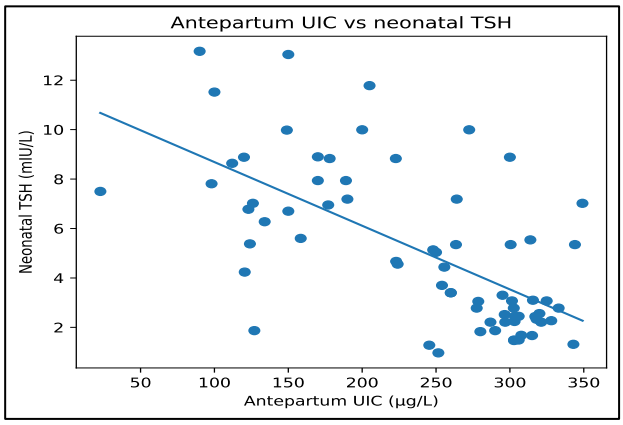

Figure 2: Scatter plot demonstrating the association between antepartum maternal urinary iodine concentration (UIC) and neonatal TSH.

Scatter plot showing the relationship between antepartum maternal urinary iodine concentration (UIC) and neonatal TSH. Each point represents an individual mother-neonate pair (n=70). The solid line indicates the linear regression trend. A significant inverse correlation was observed between antepartum UIC and neonatal TSH (Spearman's ρ=−0.625, p<0.001). UIC is expressed in µg/L and neonatal TSH in mIU/l.

Neonatal thyroid parameters are summarized in Table 3. The mean neonatal TSH was 4.44±3.1 mIU/l and mean fT4 was 3.04±1.1 ng/dl. Nearly half of the neonates had TSH ≥5 mIU/l, indicating borderline elevation at the population level.

Correlation analysis (Spearman's rank) demonstrated a significant inverse association between maternal UIC and neonatal TSH (Table 4). Antepartum UIC showed a strong negative correlation with neonatal TSH (ρ=−0.625, p<0.001), and postpartum UIC also showed a moderate inverse correlation (ρ=−0.503, p<0.001). Antepartum and postpartum UIC were strongly positively correlated with each other (ρ=0.729, p<0.001). In contrast, maternal UIC did not show a statistically significant association with neonatal fT4. Neonatal TSH and fT4 were not significantly correlated.

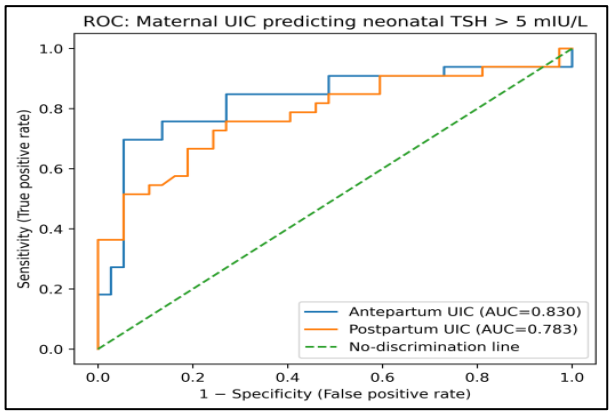

Figure 3: ROC curve of maternal urinary iodine concentration (UIC) for predicting neonatal TSH >5 mIU/l.

ROC analysis (Table 5, Figure 3) showed that antepartum UIC had good discriminatory ability for predicting neonatal TSH>5 mIU/l (AUC=0.830), while postpartum UIC demonstrated acceptable performance (AUC=0.783). Antepartum UIC showed higher specificity, whereas postpartum UIC demonstrated relatively higher sensitivity.

Table 5: ROC analysis of maternal urinary iodine concentration (UIC) for predicting neonatal TSH >5 mIU/l.

Predictor (maternal UIC)	AUC	Optimal cut-off (µg/l)*	Sensitivity	Specificity	PPV	NPV
Antepartum UIC	0.830	222.772	0.697	0.946	0.920	0.778
Postpartum UIC	0.783	223.000	0.758	0.730	0.714	0.771

*P<0.05.

DISCUSSION

Maternal iodine sufficiency is essential for optimal fetal and neonatal thyroid hormone homeostasis, particularly in late gestation and early postnatal life. In this study, maternal urinary iodine concentration (UIC) showed a clear relationship with neonatal thyroid stimulation, supporting the biological rationale that reduced iodine availability can increase neonatal TSH as a compensatory response.[5,24] Urinary iodine concentration remains a

practical population biomarker of iodine intake because most ingested iodine is excreted in urine; however, spot UIC reflects recent intake and can vary with hydration and timing of collection.[25] Thus, its interpretation is more robust at the population level rather than for individual diagnosis.

Maternal iodine status

The overall pattern of maternal UIC in this cohort suggests that a substantial proportion of women had iodine intake at or above recommended pregnancy levels. This is consistent with national improvements in iodized salt coverage and the broader trend toward iodine sufficiency reported in recent Indian survey data.[26] At the same time, regional heterogeneity remains important in India, and earlier studies from eastern settings have documented pockets of iodine deficiency disorders, indicating that local monitoring remains necessary even when national indicators appear favourable.[20,21,26] Findings from an Indian meta-analysis similarly suggest overall adequacy in pregnancy while highlighting heterogeneity across studies and regions.[25]

Postpartum UIC requires cautious interpretation because early postpartum sampling reflects a dynamic physiological period characterized by changes in renal function, fluid balance, and continuation or discontinuation of supplements.[27] In addition, iodine transfer into breast milk becomes relevant during lactation, and UIC measured during established lactation may differ from immediate post-delivery values.[24] Therefore, postpartum UIC obtained during the hospital stay provides useful immediate information, but longitudinal postpartum assessment would better characterize iodine status across lactation.

Neonatal thyroid indices

Neonatal TSH is influenced by multiple factors beyond iodine status, including gestational maturity, perinatal stress, and the timing of sample collection.[28,29] Even within recommended screening windows, values may vary between day 3 and day 5 depending on clinical status and sampling conditions, and this variability can influence the distribution of neonatal TSH.[30,31] A higher proportion of late-preterm or early-term births can further shift screening distributions because thyroid axis maturation is gestational-age dependent.[30,31] Delivery-related stress, including operative delivery (LSCS) and peripartum complications, may also transiently increase neonatal TSH independent of iodine intake.[28] In addition, transient neonatal thyroid adaptations such as the physiological postnatal TSH surge and, less commonly, autoregulatory mechanisms related to iodine excess (Wolff-Chaikoff effect) may contribute to elevated TSH levels without indicating persistent thyroid dysfunction.[32,33]

In iodine-replete settings, iodine excess can occasionally contribute to transient neonatal thyroid suppression via the Wolff-Chaikoff effect, particularly in vulnerable neonates; however, this mechanism is typically temporary and cannot be inferred without detailed exposure history (e.g., iodine-containing antiseptics, high-dose supplements) and follow-up testing.[9,29] Accordingly, interpretation of neonatal TSH as an iodine surveillance tool is strongest when applied at the population level with standardized sampling windows and consideration of prematurity and perinatal factors.[6,28,31-33]

Neonatal TSH is often more responsive to mild changes in thyroid hormone availability than fT4, which is maintained within a narrower physiological range.[28] Assay-related variation, transitional neonatal physiology, and differences in hormone binding and clearance can further blunt observable associations with fT4 in real-world cohorts.[28] While the observed correlations between maternal UIC and neonatal TSH were statistically significant, the clinical magnitude of these associations should be interpreted with caution, as neonatal thyroid function is influenced by multiple interacting physiological and environmental factors beyond iodine status alone.

Comparison with existing literature and public health implications

Our findings align with the broader Indian context of improving iodine nutrition. The India Iodine Survey and NFHS-linked estimates demonstrate substantial improvements in iodized salt coverage nationally, supporting population-level progress toward iodine sufficiency.[26] Postpartum iodine dynamics have also been reported elsewhere; Threapleton et al observed that postpartum iodine biomarkers can remain variable and are influenced by supplementation and physiological changes, reinforcing the importance of timing and lactation status when interpreting postpartum UIC.[24] Neonatal TSH has been used as a monitoring tool in several regions for assessing iodine nutrition, while consistently emphasizing the influence of sampling timing and perinatal factors on screening distributions.[12-14]

These results reinforce the need for continued emphasis on appropriate iodine nutrition during pregnancy. Antenatal programs should promote consistent use of iodized salt and evidence-based supplementation where indicated, while avoiding unnecessary high-dose iodine exposure.[16] Counselling should also address postpartum and breastfeeding periods, as iodine status may fluctuate across the immediate post-delivery phase and lactation.[24] At a systems level, neonatal TSH screening remains valuable for population surveillance, but interpretation should be standardized by sampling window and contextualized for prematurity and delivery-related stress where feasible.[28,31]

Future studies incorporating longitudinal follow-up and creatinine-adjusted urinary iodine measurements may provide a more precise understanding of maternal iodine dynamics and their impact on neonatal thyroid outcomes.

Strengths of this study include paired maternal UIC measurements across two time points linked to neonatal thyroid testing. Limitations include the single-centre design, modest sample size, use of spot UIC without creatinine correction, lack of direct dietary/supplement intake quantification, and postpartum sampling restricted to the early hospital period. These limitations should be addressed in future multicentre studies incorporating dietary assessment, repeated postpartum sampling into established lactation, and longer-term neonatal follow-up.

CONCLUSION

This study highlights the significant association between maternal iodine status and neonatal thyroid function. Maternal urinary iodine concentration (UIC) in both antepartum and postpartum periods demonstrated an inverse relationship with neonatal TSH, indicating the influence of maternal iodine availability on neonatal thyroid regulation. Despite overall iodine sufficiency in the study population, the presence of neonatal TSH elevations suggests that additional physiological and perinatal factors contribute to neonatal thyroid dynamics.

These findings support the role of neonatal TSH as a useful population-level indicator of iodine nutrition and emphasize the importance of continued monitoring of maternal iodine status during pregnancy and the early postpartum period. Further multicentric studies incorporating dietary assessment, creatinine-adjusted UIC, and longitudinal follow-up are needed to better define maternal-neonatal iodine interactions and optimize screening strategies.

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