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Original Research Article

Assessment of the Iodine Deficiency Disorders and Household Salt Iodine Consumption Among Pregnant Mother Attending Tertiary Care Hospital in North 24 Parganas

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Conflict of interest: Nil

Abstract

Introduction: Iodine is a non-metallic halogen found in the human body in trace amounts. It is an essential micronutrient required for production of thyroid hormones which are important for maintaining basal metabolic rate, growth and development.

Aims & Objectives: The study aims to assess iodine deficiency disorders and household salt iodine consumption among pregnant mothers attending a tertiary care hospital in North 24 Parganas. It also evaluates thyroid hormonal status (TSH and free T4) in first-trimester pregnant women attending the antenatal OPD of the Department of Obstetrics and Gynaecology, along with measurement of urinary iodine levels and iodine content in household dietary salt.

Materials & Methods: This was an observational and analytical study conducted in the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynaecology, College of Medicine and Sagore Dutta Hospital. The study was carried out from July 2024 to December 2025 with a total sample size of 164 patients.

Result: Based on urinary iodine excretion levels, 86% (141 participants) had adequate iodine intake (15–24.9 µg/dL), while 14% (23 participants) were found to have insufficient iodine intake (<15 µg/dL). This distribution was statistically significant ($p < 0.001$).

Conclusion: We concluded that assessing iodine deficiency disorders and household salt iodine consumption among pregnant women attending a tertiary care hospital in North 24 Parganas, the majority of participants were primigravida, followed by various multigravida categories, with a statistically significant variation in parity distribution.

Keywords: Iodine Deficiency Disorders, Pregnant Women, Urinary Iodine Excretion, Household Salt Iodization, Iodized Salt Consumption, Thyroid Function.

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Introduction

Iodine is a non-metallic halogen found in the human body in trace amounts. It is an essential micronutrient required for production of thyroid hormones which are important for maintaining basal metabolic rate, growth and development [1]. 90% of iodine required for the human body comes from diet. The major source of iodine in industrialized countries is iodized salt [2].Pregnancy is a state of altered hemodynamic, metabolic and immunological status and hence there is significant alteration of iodine homeostasis also [2]. Adequate dietary iodine supply should be ensured throughout pregnancy to meet the increased demand of thyroid hormones required for growth, development, organogenesis, and neurodevelopment of the fetus in utero. Thyroid hormone synthesis is also very important for maintaining the reproductive hormonal status which is significantly altered in different stages of pregnancy [3].The thyroid hormonal status of the fetus is highly dependent on the thyroid status of the mother and hence, in turn, on the iodine status of the mother, particularly in the early trimester when organogenesis occurs [4].The beta subunit of

human chorionic gonadotropin (β-hCG), which is essential for implantation and maintaining placental function in the first trimester of pregnancy, possesses a similar structure to thyroid-stimulating hormone (TSH). Studies have shown that a slight drop in TSH levels in early pregnancy is common and symptoms of hypothyroidism may be less prominent if present [5]. Higher levels of circulating oestrogens result in a progressive increase in thyroxine-binding globulin, and the increased secretion of type 2 and type 3 deiodinases from the placenta affects the synthesis and homeostasis of thyroid hormones [6]. The clearance of iodine is also increased during pregnancy due to increased renal blood flow, leading to an overall increased turnover of iodine [7].

Iodine deficiency disorder is almost exclusively caused by dietary insufficiency of iodine, most specifically due to low intake of iodized salt. Worldwide, it is the most common cause of preventable hypothyroidism. A spectrum of disorders may occur in pregnant women with iodine deficiency, ranging from maternal hypothyroidism and goitre to fetal complications such as intrauterine growth restriction, early pregnancy loss, preterm labour, birth asphyxia, neonatal hypothyroidism, cretinism, and delayed or impaired mental and social development [8].

Materials and Methods

Type of Study: Observational and analytical study.

Place of Study: Biochemistry department with joint collaboration of Department of Obstetrics and Gynaecology, College of Medicine and Sagore Dutta Hospital

Study Duration: July 2024 to December 2025

Sample Size: 164 patients

Inclusion Criteria: Pregnant women aged between 25 and 45 years in their first trimester (0-12 weeks).

Exclusion Criteria

- Any concomitant acute or chronic illness.
- Known history of thyroid disorder: present or past (history of underwent any thyroid surgery)
- Ingestion of Iodine containing products like cough syrups, history of administration of radio opaque dye etc.
- With any bad obstetric history eg., Intrauterine fetal death, congenital anomaly etc.
- Any malignancy

Study Variables

- Age of the pregnant mother
- Parity (primigravida / multigravida)
- Trimester of pregnancy
- Educational status
- Socioeconomic status
- Urinary Iodine Concentration (UIC) level
- Presence of iodine deficiency (yes/no)
- Thyroid function status:

Statistical Analysis: Data were entered into Excel and subsequently analyzed using SPSS and GraphPad Prism. Continuous variables were summarized as means with standard deviations, while categorical variables were presented as counts and percentages.

Comparisons between independent groups were performed using two-sample t-tests, and paired t-tests were applied for correlated (paired) data. Categorical data were compared using chi-square tests, with Fisher's exact test applied when expected cell counts were small. A p-value of ≤ 0.05 was considered statistically significant.

Result

Table 1: Distribution of study participants according to the parity

Parity	Total number of study participants	Percentage	P-value
PRIMI	82	50.00%	< 0.001
G2A1	12	7.32%	
G2P1L1	41	25.00%	
G3P1L1A1	13	7.93%	
G2D1	1	0.61%	
G3A2	1	0.61%	
G3P2L2	7	4.27%	
G4P1L1A2	4	2.44%	
G4P2L2A1	2	1.22%	
G5P1L1A3	1	0.61%	

Table 2: Urinary Iodine Excretion (µg/dL) and Iodine content (ppm) in household salt

UIE (µg/dL)	Iodine Content in salt	No. of study participants	Percentage	p-value
<15	<15 ppm	10	6%	< 0.001
<15	16–30 ppm	13	8%	
15–24.9	<15 ppm	62	38%	
15–24.9	16–30 ppm	79	48%	

Table 3: Correlation between Urinary Iodine Excretion (µg/dL) and Iodine content in salt(parts per million) used for household purpose

Spearman's correlation		
		UIE
Iodine Content in household salt	rho value	0.091
	p-value	0.255

Table 4: Comparison between Urinary Iodine Excretion and Serum Free T4 among study population

Pearson's correlation		
		UIE
Free T4	r value	-0.024
	p-value	0.763

Table 5: Urinary Iodine Excretion (UIE) of pregnant women

	UIE(µg/dL)
Mean ± SD	16.61 ± 1.65
Median	16.75

Urinary Iodine Excretion (µg/dL)	**Iodine Intake**	**Count**	**Percent**	**p-value**
<15 µg/dL	Insufficient	23	14%	< 0.001
15–24.9 µg/dL	Adequate	141	86%	

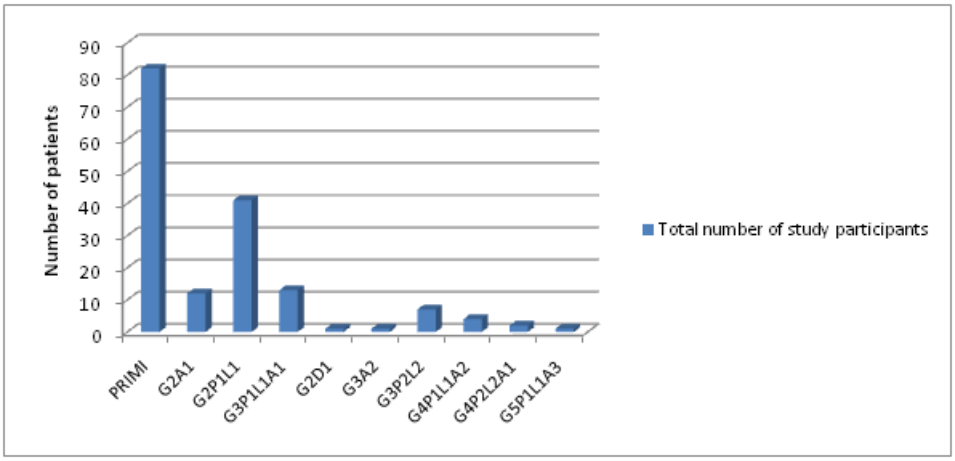

Figure 1: Distribution of study participants according to the parity

Among the study participants, primigravida women formed the largest group, comprising 50.0% (82 participants), followed by G2P1L1 (25.0%, 41 participants), while the remaining participants were distributed across G2A1 (7.32%), G3P1L1A1 (7.93%), G3P2L2 (4.27%), G4P1L1A2 (2.44%), and a few rare categories including G2D1 (0.61%), G3A2 (0.61%), and G5P1L1A3 (0.61%), showing a statistically significant variation in parity distribution ($p < 0.001$). The mean urinary iodine excretion (UIE) among pregnant women was 16.61 ± 1.65 µg/dL with a median value of 16.75 µg/dL, indicating a relatively narrow dispersion around the mean. Based on iodine status classification, the majority of participants, 86% (141 women), had adequate iodine intake (15–24.9 µg/dL), whereas 14% (23 women) were found to have insufficient iodine intake (<15 µg/dL), and this distribution was statistically significant ($p < 0.001$). In relation to household salt iodine content, among women with UIE <15 µg/dL, 6% used salt containing <15 ppm iodine and 8% used salt with 16–30 ppm iodine, whereas among those with adequate UIE (15–24.9 µg/dL), 38% used salt with <15 ppm iodine and 48% used adequately iodized salt (16–30 ppm), suggesting a higher proportion of adequate iodine status in those consuming iodized salt. However, Spearman's correlation analysis demonstrated a very weak positive correlation between UIE and household salt iodine content (rho = 0.091), which was not statistically significant ($p = 0.255$), indicating no meaningful association. Similarly, Pearson's correlation between UIE and serum free T4 levels showed a very weak negative correlation ($r = -0.024$) with no statistical significance ($p = 0.763$), suggesting that urinary iodine levels were not significantly associated with thyroid hormone levels in the studied population.

Discussion

In the present study, primigravida women constituted the largest proportion of the study population (50.0%), followed by multigravida categories in varying distributions, showing a statistically significant variation in parity pattern ($p < 0.001$). This predominance of primigravida mothers is consistent with findings reported by Andersson et al. [9], who observed that antenatal clinic attendees in iodine-deficient settings often include a higher proportion of first-time pregnant women, likely due to increased health-seeking behavior during first pregnancy. The mean urinary iodine excretion (UIE) in the present study was 16.61 ± 1.65 µg/dL with a median of 16.75 µg/dL, indicating overall marginal adequacy of iodine intake among the study participants. Similar findings were reported by Zimmermann et al. [10], who demonstrated that median urinary iodine concentrations in pregnant women frequently fall in the lower adequate range even in populations considered iodine sufficient, reflecting the increased physiological iodine requirement during pregnancy. In the current study, 86% of participants had adequate iodine status (15–24.9 µg/dL), while 14% showed insufficient iodine intake (<15 µg/dL), and this distribution was statistically significant ($p < 0.001$). Comparable results were observed in a study by Gowachirapant et al. [11], where a substantial proportion of pregnant women exhibited borderline or insufficient iodine levels despite national iodization programs, highlighting persistent subclinical iodine deficiency in vulnerable groups. Regarding household salt iodine content, the present study demonstrated that a higher proportion of women with adequate UIE consumed iodized salt (16–30 ppm), whereas those with low UIE had greater use of inadequately iodized salt (<15 ppm). However, the correlation between UIE and salt iodine content was very weak and not statistically significant (rho = 0.091, $p = 0.255$). Similar weak associations were reported by Ma et al. [12], who found that household salt iodine concentration alone may not strongly predict urinary iodine levels due to factors such as dietary diversity, cooking practices, and individual variability in iodine absorption. Furthermore, the present study showed no significant correlation between UIE and serum free T4 levels ($r = -0.024$, $p = 0.763$), indicating that urinary iodine status did not directly reflect thyroid hormone levels in this cohort. This finding aligns with the study by Glinoer [13], which demonstrated that thyroid hormone levels in pregnancy are influenced by multiple physiological adaptations, and mild iodine deficiency may not immediately alter free T4 concentrations due to compensatory thyroidal mechanisms. the findings of the present study emphasize that while a majority of pregnant women had adequate iodine status, a significant minority remained insufficient, and household salt iodization alone may not fully ensure optimal iodine nutrition. This underscores the importance of continuous monitoring of iodine status during pregnancy.

Conclusion

We concluded that assessing iodine deficiency disorders and household salt iodine consumption among pregnant women attending a tertiary care hospital in North 24 Parganas, the majority of participants were primigravida, followed by various multigravida categories, with a statistically significant variation in parity distribution. The urinary iodine excretion (UIE) findings showed that most women had adequate iodine status, while a smaller proportion demonstrated insufficient iodine intake. Although a considerable number of participants were using adequately iodized household salt, a subset still consumed salt with suboptimal iodine content, indicating persistent gaps in iodine utilization at the household level. However, correlation analysis revealed a very weak and statistically insignificant association between UIE and household salt iodine content, suggesting that urinary iodine levels are influenced by multiple dietary and physiological factors beyond salt iodine concentration alone. Similarly, no significant correlation was observed between UIE and serum free T4 levels, indicating that thyroid hormone levels remained relatively stable despite variations in iodine intake, likely due to physiological adaptations during pregnancy. Overall, the study indicates that while iodine status is generally adequate among most pregnant women, a vulnerable minority still faces risk of insufficient iodine intake. These findings emphasize the need for continued public health strategies, including strengthening antenatal nutritional education, ensuring consistent use of adequately iodized salt at the household level, and improving awareness regarding iodine requirements during pregnancy to safeguard maternal and fetal thyroid health.

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