

Iodine deficiency in pregnant women of Lahore

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Abstract

Objective: To know extent of iodine deficiency (ID), role of thyroid enlargement (goiter) as marker of ID and current status of iodized salt intake in pregnant women of Lahore.

Methods: A cross sectional study was carried out at Institute of Chemistry, University of the Punjab, during March 2002 to September 2005. Pregnant women (n = 254) during first trimester attending antenatal clinic participated voluntarily. Iodine intake status was determined by urinary iodine (UI) excretion.

Results: UI excretion ranged from 34 to 142 µg/L and median value was 67µg/L. According to international criteria, 202 (79.5%) pregnant women were iodine deficient (UI <100 µg/L) mostly (68.8%) of mild (UI: 50-99 µg/L) degree. Moderate iodine deficiency (MID; UI <50 µg/L) was found in 63 (24.8%) pregnant women. Among all pregnant women 80 (31.5%) had slightly visible goiter and only 87(34.2%) were currently taking iodized salt. The difference in UI excretion between goitrous and non-goitrous pregnant women was not significant. Among iodized salt users percentage of women with MID was less, though not significant, as compared to non-users (20.7% Vs 26.9%).

Conclusion: About one-fourth of pregnant women screened in this study are moderately iodine deficient in Lahore. These women and their neonates are at increased risk of iodine deficiency disorders. Goiter is not a good indicator of low iodine intake while iodized salt consumption is beneficial in this regard (JPMA 59:741; 2009).

Introduction

Iodine is essential for the synthesis of thyroid hormone. In pregnant women thyroid hormones are required for development and maturation of brain and neurological network of the foetus.¹ Trans-placental passage of iodine and thyroxine from mother to foetus takes place throughout pregnancy.² That is why in pregnant women thyroid hormones synthesis is physiologically increased that requires more iodine. Moreover, renal clearance of iodine from the kidney is increased and a part of the available iodine from the maternal circulation is diverted to the foetus for thyroid hormones synthesis by the foetal thyroid gland.³ For healthy pregnant women with iodine sufficiency, maternal gland readily adjusts the hormonal output due to ample supply of iodine. This state is difficult to achieve when iodine intake of pregnant women is limited and leads to a relative iodine deficiency state in mother as well as foetus.^{3,4} Iodine deficiency is detrimental to both mother and foetus leading to goitrogenesis and hypothyroxinemia in mother and consequently suboptimal brain development in foetus.⁵⁻⁷ The recommended dietary intake of iodine is 150 µg/day for adult and 200 µg/day for pregnant women.⁸ Iodine intake is assessed by determination of urinary iodine (UI) excretion. A UI excretion of 100 µg/L indicates sufficient iodine intake.^{8,9}

Pakistan is considered one of the severely iodine deficient country in the region.¹⁰ The results of surveys conducted by WHO and UNICEF during the last couple of years presented an alarming picture of the iodine deficiency disorders in the country.^{8,9} However, the quantification of

iodine deficiency in pregnant women is yet to be elucidated. This study was conducted to know the iodine nutrition status of healthy pregnant women residing in Lahore. Moreover, utility of thyroid enlargement (goiter) as an indicator of iodine deficiency and percentage of pregnant women taking iodized salt was assessed.

Subjects and Methods

This cross sectional study was carried out during the period of March 2002 through to September 2005. All participant women resided in the municipal jurisdiction of Lahore and belonged to middle socio-economic strata. These women attended antenatal clinics at outpatient department of obstetrics and gynecology, Government Mian Munshi Hospital, twice weekly. The objective of study was explained to each woman and a verbal consent of participation was obtained. Women with history of thyroid surgery or known biochemical thyroid dysfunction were excluded from study. Each woman was inducted in the study when her pregnancy was confirmed by ultrasound scan. Ultrasonography was performed inside the hospital using standard procedure on an ultrasound machine (Siemens, Germany, Model Solo line SL-1, 7.5 MHz with linear transducer). Thyroid gland size was assessed by palpation. The Institutional Review Board at University of the Punjab, Lahore approved the study.

In total 310 women were examined but only 254 women fulfilled the criteria of selection. Urine samples were collected from each selected woman. Each urine sample was

collected in an aseptic plastic container, sealed and labeled with identification code. UI concentration was determined at laboratories of Institute of Chemistry, University of the Punjab Lahore by Sandell-Kolthoff reaction (modified) as recommended by WHO/UNICEF/ICCIDD (2001).⁸ Urine was digested with ammonium persulfate and iodide, acting as catalyst in the reduction of ceric ammonium sulfate (yellow) to cerous form (colourless), was determined by rate of colour disappearance. Measurements were carried out in duplicates. Results were expressed as microgam of iodine per liter of urine ($\mu\text{g/L}$). The lower detection limit for the assay was $0.15\mu\text{g/L}$. The mean coefficient of variance (CV) for iodine concentration $0.18\mu\text{g/L}$ was 16.2%, for $0.78\mu\text{g/L}$ was 7.1% and for $1.3\mu\text{g/L}$ was 5.5%. The WHO/UNICEF/ICCIDD criteria were used to classify the pregnant women as taking sufficient, mildly deficient and moderately deficient iodine.⁸

Results are presented as means ($\pm\text{SD}$), median or otherwise specified. Data were analyzed using statistical package for social sciences (SPSS) [version 13.0 for Windows supplied by SPSS Inc. 2003, Mapinfo Corp. NY, USA]. Group means were compared by Student's t test. Chi-square test was used to compare frequencies among different groups. All statistical tests were considered statistically significant whenever $P < 0.05$.

Results

UI excretion levels showed a wide individual scatter and ranged from 34-142 $\mu\text{g/L}$ ($70.7 \pm 26.5\mu\text{g/L}$) in pregnant women. Mean and median UI was $70.7\mu\text{g/L}$ and $67.0\mu\text{g/L}$ respectively. The distribution of UI and iodine intake status of pregnant women according to WHO criteria is shown in the Table-1. Most of the pregnant woman ($n = 220$; 79.5%) had UI excretion below $100\mu\text{g/L}$. Among them 139 were mildly while 63 (24.8 percent of total sample) were moderately iodine

Table-1: Iodine intake status of pregnant women based on urinary iodine excretion.

Urinary iodine conc. ($\mu\text{g/L}$)	Correspond approx iodine intake* ($\mu\text{g/day}$)	No. of pregnant women (%)	UI mean \pm SD (range)
$>100\mu\text{g/L}$	Sufficient ($>150\mu\text{g/day}$)	52	113.5 ± 11.1 (100-142)
	Mildly Deficient (75-149 $\mu\text{g/day}$)	-20.5	68.1 ± 11.8 (50-92)
50 - 99 $\mu\text{g/L}$	Moderately Deficient (30-74 $\mu\text{g/day}$)	139	41.1 ± 03.3 (34-48)
$< 50\mu\text{g/L}$		-54.7	
		63	
		-24.8	

* WHO/UNICEF/ICCIDD (2001)

deficient ($\text{UI} < 50\mu\text{g/L}$).

Among pregnant women 87 (34.2 percent) were currently taking iodized salt and 80 (31.5 percent) women had slightly visible goiter. A comparison of UI concentration between different groups of pregnant women based on iodized salt use and presence of goiter is shown in Table-2. The

Table-2: Iodine intake status of different sub-groups of pregnant women.

Factor women	Sub-group (No. of women)	UI ($\mu\text{g/L}$)	No. of with $\text{UI} < 50\mu\text{g/L}$ (%)
Presence of Goiter	Goitrous (80)	66.7 ± 23	19 (23.7)
	Non-goitrous (174)	72.5 ± 27.8	44 (25.3)
Iodine Salt Intake	User (87)	75.4 ± 27.3	18 (20.7)
	Non-User (167)	68.8 ± 26	45 (26.9)

difference in mean UI excretion was not significantly different in pregnant women taking or not-taking iodized salt. However, percentage of women with moderate iodine deficiency was reduced, though did not reach a significant level ($P=0.06$), among iodized salt user as compared to non-user. Similarly the difference in mean UI concentration between goitrous and non-goitrous pregnant women was not significant ($P=0.08$). Among women with moderate iodine deficiency only 18 (28.5 percent) had goiter while 9 (17.3 percent) women with sufficient iodine intake also had goiter. This difference was not significant ($P=0.36$).

Discussion

Our results indicated that most of the pregnant women included in this study had iodine intake well below the recommended limit.⁸ With median UI excretion $67.0\mu\text{g/L}$, the cohort of the pregnant women under study was moderately iodine deficient.¹¹ A study in Lahore has already reported mild to moderate iodine deficiency, as estimated by cord blood TSH, in pregnant women.¹² Our results are in accordance to those reported by National Nutrition Survey 2001-2002 of Government of Pakistan. According to that survey mean UI excretion in pregnant women was $69.2 \pm 78.9\mu\text{g/L}$ and 76 percent women had $\text{UI} < 100\mu\text{g/L}$.⁹ However, there is a

noteworthy difference: no women in this cohort had severe iodine deficiency ($\text{UI} < 20\mu\text{g/L}$) while about 36.5 percent mothers were reported to have severe iodine deficiency in above mentioned survey. The reason may be in the difference in study population. Our study was based on urban women of only one city but Nutrition Survey recruited pregnant women

from selected urban and rural areas of Pakistan.

Iodine intake status is ethnic specific and is most likely because of differences in dietary behaviours among different groups.¹³ The dietary content of iodine in typical Pakistani diet is reported to be 60 µg/day.¹⁴ Akhter et al (2004) has reported that excluding the iodine intake from sea food and water, this daily intake is further reduced to 40 µg/day. This figure is 3.8 times lower than recommended for adult subject (150 µg/d). The low iodine content of the Pakistani diet might be due to the dietary habits of the Pakistani people, to diet composition (mainly a lack of sea food), to the natural environment of the country (lack of iodine in the Pakistani soil) and to iodine losses during cooking.¹⁵

The history of iodine availability prior to and during pregnancy is determinant of UI excretion.¹⁶ It is proposed that depending upon dietary iodine intake each woman has her own specific UI-threshold termed "iodostat" during pregnancy.¹⁷ The increased renal clearance of iodine above the UI-threshold results in increased iodine loss. This UI-threshold is masked at higher iodine intakes but in iodine deficiency lead to negative iodine balance and thyroid depletion. This loss is compensated by increased thyroid volume to allow for more iodine storage.¹⁸ The existence of this compensatory mechanism to offset increased iodine loss is an adaptation to ensure the retention of extra iodine during gestation. Thus increased thyroid volume (goiter) during pregnancy indirectly indicates iodine deficiency. About 31 percent of pregnant women of this cohort had slightly visible goiter. However, we observed goiter in only 28 percent pregnant women who were moderately iodine deficient. Moreover, no significant difference in UI excretion between goitrous and non-goitrous pregnant women was observed. Thus, existence of slightly visible goiter in pregnant women of this cohort does not indicate low iodine intake and was independent of UI excretion. The increase in thyroid volume may also be due to protein malnutrition as reported in Indian pregnant women by Sakinah et al.¹⁹

Present study revealed that only 34 percent pregnant women were taking iodized salt. An investigation from a neighboring city, Faisalabad, reported that among pregnant women only 6 percent were taking iodized salt.²⁰ These figures are not impressive and warrant the recognition of obstacles to on going iodine supplementation program of Government of Pakistan. This study indicated that iodized salt intake improved the iodine intake status of pregnant women. Iodine sufficiency can be achieved only through mandatory iodine supplementation of pregnant women. Maternal iodine sufficiency is necessary as recent investigations had shown that foetal brain formation is the main target of iodine deficiency that is affected by maternal thyroid status during gestation.^{6,7}

A shortcoming of this study was that of small sample size restricted to one hospital only that did not made it representative of Lahore City. Further, large studies are needed to elucidate iodine and thyroid hormone deficiency in pregnant women and its complications like pregnancy-induced hypertension, placenta abruption, foetal distress, postpartum haemorrhage, postpartum depression and major complications at delivery. These aspects of reproductive health requires immediate attention.

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