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Assessment of Iodine Nutritional Status of School-Age Children in Kolkata District of West Bengal State in Post-Iodation Scenario

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ABSTRACT

To evaluate the state of iodine nutrition in post-iodation scenario, 3500 children were examined clinically for endemic goitre. Iodine and thiocyanate were measured in 240 urine samples; iodine content in 210 salt samples was measured. Total goitre prevalence was 6.1%. Median urinary iodine level was 21.80 μg/dl, and mean (±SD) urinary thiocyanate was 0.89 ± 0.49 mg/dl. Iodine content of only 11.9% salt samples was below recommended level of 15 ppm, 25.2% was between 15 and 30 ppm and 62.9% was >30 ppm. Iodine deficiency disorders are thus clinically mild public health problem of the studied population; however, they have no biochemical iodine deficiency. Studied population found exposed to thiocyanate load that might be the possible cause for persistence of endemic goitre. People of Kolkata should be advised to eat commonly consumed goitrogenic foods after boiling and decanting the water. Periodical monitoring and evaluation of iodine status should be mandatory.

KEYWORDS: iodine deficiency disorders, goitre, salt iodation, urinary iodine, urinary thiocyanate

BACKGROUND

Iodine is required for the production of thyroid hormone by thyroid. Requirement of iodine per day is 90 μg for preschool children (0–59 months), 120 μg for school children (6–12 years), 150 μg for adolescents (>12 years) and adults and 250 μg for pregnant and lactating mothers [1]. In iodine deficiency, thyroid does not produce thyroid hormone, which may lead to a spectrum of diseases named as iodine deficiency disorders (IDDs). IDDs are manifested by goitre, stillbirths, spontaneous abortion and congenital anomalies, including endemic cretinism and irreversible brain damage [2, 3].

WHO estimated nearly 2 billion people are at risk of IDDs, including one-third of school children [4]. Total goitre prevalence (TGP) was lowest in America (10.6%), whereas it was highest in Europe (52.0%). Australia is mildly iodine deficient, and in Switzerland, median urinary iodine (MUI) excretion is 14 μg/dl [5]. In Portugal, MUI is 12.9 μg/dl [6]. In Ireland, 26% population is iodine deficient [7]. TGP of Southern Ethiopia is 48.9% [8] and northwest Ethiopia is 29.1% [9]. Israel is iodine deficient (MUI 8.3 μg/dl) [10].

In India, 350 million peoples are at risk of IDDs, as they consume inadequately iodized salt [11]. Of 587 districts, 282 surveyed for IDD and 241 have been found goitre endemic [12]. TGP of Maharashtra is 11.9% [11]. Tripura, Manipur, and Uttar Pradesh are affected by IDDs [13–17]. TGP of

Nainital is 15.9% (MUI 12.5 μg/dl) [12]. In Kullu, TGP is 23.4% (MUI 17.5 μg/dl) [18]. In Udaipur, TGP is 8.4% (MUI 20 μg/dl) [19]. In Kottayam, TGP is 7.05% (MUI 17.5 μg/dl) [20].

In 1962, Government of India (GOI) launched National Goitre Control Programme (NGCP). In August 1992, NGCP was renamed as National Iodine Deficiency Disorders Control Programme [21]. In 1997, GOI banned sale of non-iodized salt. In September 2000, GOI lifted ban stating 'On point of principle, compulsions in the matter of individual choice are undesirable' that results nationwide 12% decrease in coverage of iodized salt; in May 2006, GOI reimposed the ban [5, 22].

Iodine nutritional status of school children in Kolkata during post-iodation scenario is not available. Present investigation has been undertaken to evaluate iodine status of selected population with possible involvement of dietary goitrogens (if any) in Kolkata.

MATERIALS AND METHODS

Data collection

This school-based cross-sectional study was undertaken in North, East, and West Kolkata. List of schools and their enrolment number in each area were previously collected from School Education Department. Probability proportion to size methodology was adopted for selection of schools [1]. Total 30 schools (clusters) were selected. From each school, all children in the age group of 6–12 years of both sexes were clinically examined for goitre. Sample size was calculated based on assumed goitre prevalence 5%, confidence interval 95% and permissible error of margin 20%. Sample size of 1900 was obtained [15, 16, 23]. To increase precision of study, 3500 school children were examined.

Clinical examination of goitre

IDD is manifested by goitre [24]. Pupils were examined clinically for enlargement of thyroid. Grading of goitre was done as Grade 0, no palpable or visible goitre; Grade 1, goitre that was palpable but not visible when the neck was in normal position; and Grade 2, goitre that was visible when the neck was in normal position. TGP (Grade 1 + Grade 2 goitre) in school children in the age group of 6–12 years is one of the most acceptable markers of IDD. Prevalence rate of 0–4.9% was considered as none, 5–19.9% as mild, 20.0–29.9% as moderate and >30.0% as a severe public health problem [1].

Measurement of urinary iodine

Iodine content in 30 urine samples collected at random from pupils in the age group of 6–12 years gives proper idea about iodine nutritional status in a region [1]. From each locality, 80 urine samples were randomly collected from children in the age group of 6–12 years of both sexes irrespective of their thyroid status. Total 240 urine samples were measured for iodine content by arsenite method [25]. Internal quality control was adopted during analysis [12, 26]. MUI <9.9 μg/dl was considered as insufficient, 10–19.9 μg/dl as adequate, 20–29.9 μg/dl as above requirements and ≥30 μg/dl as excessive iodine intake [1].

Measurement of urinary thiocyanate

The amount of thiocyanate excreted in urine is an excellent indicator for consumption of most common goitrogenic plant foods [27]. Dietary supplies of cyanogenic glycosides and glucosinolates might be estimated from concentration of thiocyanate in urine [28]. Thiocyanate was estimated by the method of Aldridge [29] and modified by Michajlovskij and Langer [30].

Measurement of salt iodation status

In India, iodine is supplemented through edible salts because salt is inexpensive and consumed by all classes of people throughout the year in almost uniform quantity. Iodine content of at least 35 salt samples collected at random provides a valid estimate of iodation status [1]. Students were asked to carry salt samples from their household next day. From each locality, 70 salt samples were collected. Total 210 salt samples were measured for iodine by iodometric titration [31].

Data analysis and management

As per WHO/UNICEF/ICCIDD, mean, median, mode, SD, skewness (Sk), kurtosis (K) and percentile values (P_{20} and P_{80}) were computed for urinary iodine [1]. For urinary thiocyanate, mean ±SD was

computed. Salt iodine values were categorized <15, 15–30 and >30 ppm. Pearson's product moment correlation was computed between urinary iodine and iodine content of salt samples [12, 13]. All descriptive statistical analysis was done in Microsoft Office Excel 2007 [1].

RESULTS

Overall, TGP of studied population was 6.1%. In North, East and West Kolkata TGP was 5.8, 7.0 and 5.5%, respectively (Table 1).

Overall, MUI excretion of Kolkata was 21.80 μg/dl (mean ± SD = 23.65 ± 11.74 μg/dl, mode = 20.75, confidence level 95% was between 22.16 and 25.14 μg/dl). In North, East and West Kolkata, MUI was 23.10, 21.70 and 18.60 μg/dl, respectively (Table 2, Fig. 1). Frequency distribution of MUI indicated that 6.25% population was iodine deficient, 32.50% population's iodine intake was adequate, 39.17% population's iodine intake was more than adequate and 22.08% population's iodine intake was excessive (Table 3). P_{20} was 13.45 μg/dl, and P_{80} was 30.75 μg/dl. Mean was much higher than median, indicating that the distribution was heavily skewed to right (Sk = +0.934262) and histogram was platykurtic ($K = 0.773096$) (Table 2, Fig. 2).

Mean urinary thiocyanate (MUSCN) of total studied population was 0.89 ± 0.49 mg/dl. In North, East and West Kolkata, it was 0.84 ± 0.54, 0.94 ± 0.49 and 0.88 ± 0.44 mg/dl, respectively (Table 4, Fig. 3).

Overall, 11.9% of collected salt samples were <15 ppm, and 62.9% salt samples were >30 ppm (Fig. 4). In North Kolkata, 15.7% salt samples were <15 ppm, and 70% salt samples were >30 ppm. In East Kolkata, 10% salt samples were <15 ppm, and 62.9% salt samples were >30 ppm. In West Kolkata, 10% salt samples were <15 ppm, and 55.7% salt samples were >30 ppm (Table 5). A significant correlation was found in between urinary iodine excretion and iodine content of edible salt samples ($p < 0.01$).

DISCUSSION

IDDs are not restricted in classical goitre endemic belt of India. Goitre is endemic in many newly introduced pockets in post-iodation scenario [11–20]. IDDs are mild to moderately prevalent in certain non-conventional plain and costal region of West Bengal (WB) [1]. Average TGP of WB is 9% [2].

Table 1. Goitre prevalence of the studied population of Kolkata district

Studied localities of Kolkata	Population studied	Goitre prevalence (%)				Severity as a public health problem
		Grade 0	Grade 1	Grade 2	TGP (1 + 2)	
North	1559	1468 (94.2)	82 (5.2)	9 (0.6)	91 (5.8)	Mild
East	1030	958 (93.0)	63 (6.1)	9 (0.9)	72 (7.0)	Mild
West	911	861 (94.5)	46 (5.1)	4 (0.4)	50 (5.5)	Mild
Total	3500	3287 (93.9)	191 (5.4)	22 (0.6)	213 (6.1)	Mild

Grade 0: no goitre, Grade 1: palpable goitre, Grade 2: visible goitre, TGP: sum of Grade 1 and Grade 2 goitre.

Table 2. Urinary iodine excretion pattern of the studied population of Kolkata district

Studied localities	Number of urine samples assayed	Mean (±SD) (μg/dl)	Median (μg/dl)	Iodine intake	Iodine nutritional status
North	80	26.43±12.67	23.10	Above requirements	More than adequate
East	80	22.57±10.20	21.70	Above requirements	More than adequate
West	80	21.94±11.85	18.60	Adequate	Optimal
Total	240	23.65±11.74	21.80	Above requirements	More than adequate

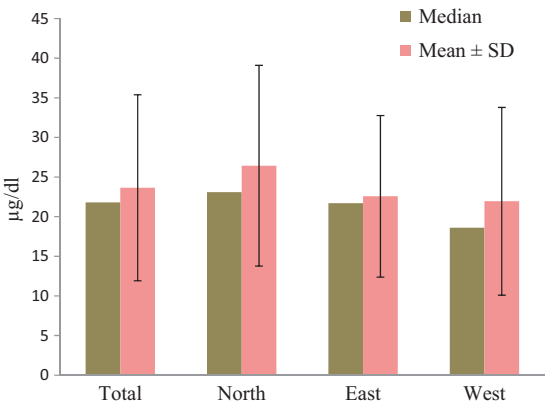

Fig. 1. Diagrammatic representation of urinary iodine excretion pattern in iodation scenario of Kolkata.

Table 3. Frequency distribution of the values of urinary iodine in Kolkata district

Urinary iodine excretion pattern	Frequency	Total	Percentage
Insufficient iodine intake			
0–4.9	6	15	6.25
5.0–9.9	9		
Adequate iodine intake			
10.0–14.9	42	78	32.5
15.0–19.9	36		
More than adequate iodine intake			
20.0–24.9 (location of median)	60	94	39.17
25.0–29.9	34		
Excessive iodine intake			
30.0–34.9	15	53	22.08
35.0–39.9	15		
40.0–44.9	3		
45.0–49.9	8		
50.0–54.9	10		
55.0–59.9	0		
60.0–64.9	2		
65.0–69.9	0		
Total	240	240	100

Of 19 districts in Gangetic WB, 8 have been surveyed for IDDs and found goitre endemic. In certain districts of WB, IDDs are still prevalent, such as Howrah (TGP 38%) [32], Sundarban Delta incorporating North 24 Parganas (33.1%) [33] and South 24 Parganas (38.2%) [34], Maldah (11.3%) [35], Birbhum (12.6%) [36], Purulia (25.9%) [37], Dakshin Dinajpur (18.6%) [38] and Darjeeling (8.7%) [39]. TGP of Kolkata is 6.1%. As per WHO/UNICEF/ICCIDD, severity of IDDs in Kolkata is clinically a mild public health problem in post-iodation scenario [1].

The indicator of IDDs eradication is a median value for urinary iodine concentration of 10 µg/dl, iodine content of 50% of urine samples should be >10 µg/dl and not <5 µg/dl of 20% population [1]. MUI of the studied region is well above the recommended level, and thus, in general, deficient iodine intake is not the criteria for the persistence of mild goitre endemicity (Table 1). MUI of the studied region found more than adequate, and they are susceptible to iodine-induced hyperthyroidism (IIH) in long run. Increasing dose of iodine in microgram range may cause hyper or hypothyroidism in those with past or present thyroid abnormalities [5]. In children, excess dietary iodine has been associated with goitre and thyroid dysfunction [40]. In present study, 6.25% population found iodine deficient, 32.50% population's iodine intake was adequate, 39.17% population's iodine intake was more than adequate and they are susceptible to IIH and 22.08% population's iodine intake is excessive and they are susceptible to risk of adverse health consequences like IIH and autoimmune thyroid diseases [1, 5]. MUI is reported >20 µg/dl in several states and districts of India [32, 41–43]. In Delhi and Jammu and Kashmir, MUI was >30 µg/dl [44, 45]. In present study, TGP is 6.1%, and 6.25% population is iodine deficient. It may be concluded that persisting goitre prevalence may be because of iodine deficiency or insufficient iodine intake.

Dietary goitrogens are substances that can interfere directly on iodine metabolism in thyroid. Goitrogens are classified as thiocyanate, isothiocyanate, cyanogenic glycosides, glucosinolates, thiourea, thionamides, flavonoids, etc. [46–50]. MUSCN is an excellent marker to study consumption of dietary goitrogens. Thiocyanate is exogenous in origin and a potent goitrogenic substance as anion with same molecular size as iodine. It inhibits thyroidal

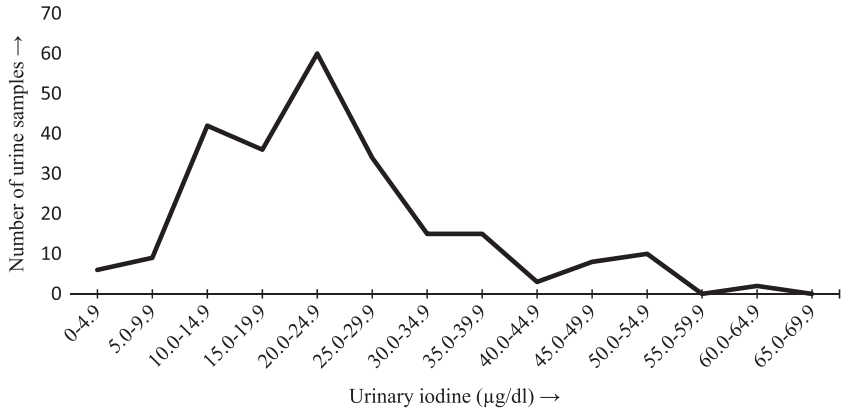

Fig. 2. Histogram to show distributions of urinary iodine values after iodation in Kolkata.

Table 4. Urinary thiocyanate excretion pattern of the studied population of Kolkata district

Studied localities	Number of urine samples	Mean (±SD) (mg/dl)
North	80	0.84 ± 0.54
East	80	0.94 ± 0.49
West	80	0.88 ± 0.44
Total	240	0.89 ± 0.49

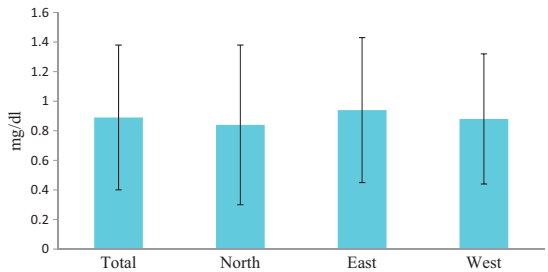

Fig. 3. Diagrammatic representation of urinary thiocyanate excretion pattern of Kolkata.

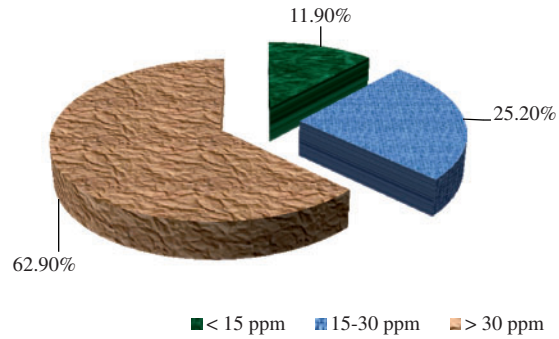

Fig. 4. Diagrammatic representation of overall salt iodation status of Kolkata district.

accumulation of iodide. At higher dose, it competes with iodine during protein binding [46–50]. It serves as a substrate for thyroid peroxidase (TPO) to inhibit iodination of tyrosyl residues of thyroglobulin molecule [51]. Within normal thyroid, iodine serves a dual role, as a substrate for iodination of thyroglobulin and as a ligand for coupling reaction [52]. Thiocyanate binds with same regulatory site as iodine but has a slightly different affinity [51]. Boiled extracts of goitrogenic plant foods showed maximum inhibition of TPO activity followed by cooked and raw extracts [53]. Regular consumption of goitrogenic plant foods affects thyroid physiology and may develop endemic goitre [54]. A large number of goitrogenic plant foods are commonly consumed by Indian population, and IDDs persists in many such newly introduced pockets, despite recommended iodine intake [13–18, 32–34]. Earlier workers mentioned that mean value of urinary thiocyanate excretion obtained from non-goitrous population is 0.71 ± 0.34 mg/dl [55]. We have considered that value as a control. MUSCN excretion of studied region is significantly higher than control ($p < 0.01$). Therefore, the studied population of Kolkata metropolitan city in the age group of 6–12 years is exposed to excess thiocyanate load (Table 4).

Table 5. Salt iodation status of the studied population of Kolkata district

Studied localities	Number of salt samples tasted	Iodine content of edible salt samples of Kolkata		
		< 15 ppm	15–30 ppm	> 30 ppm
North	70	11 (15.7%)	10 (14.3%)	49 (70.0%)
East	70	7 (10.0%)	19 (27.1%)	44 (62.9%)
West	70	7 (10.0%)	24 (34.3%)	39 (55.7%)
Total	210	25 (11.9%)	53 (25.2%)	132 (62.9%)

According to Prevention of Food Adulteration act, iodine content of the salt samples shall not be <15 ppm at household level [56]. As per National Family Health Survey-III, 51% households were using adequately iodized salt whose iodine content was ≥ 15 ppm. Another 25% were using salt that had iodine content below recommended level of 15 ppm, and 24% of households were found to be using non-iodized salt [57]. In Kolkata, 11.9% of salt samples have iodine <15 ppm, 25.2% of salt samples have iodine from 15 to 30 ppm and 62.9% of families are consuming iodized salt >30 ppm of iodine (Table 5). The goal of salt iodation programme is that >90% of household should use iodized salt [1]. In Kolkata, 11.9% families' dietary need for iodine is not properly fulfilled by salt iodation programme, but their iodine status is optimum as evidenced by MUI. Kolkata is not located in iodine-deficient zone, so their extra need for iodine is managed by drinking water and other consumable foods. Iodine status of overall studied population is more than adequate, as studied population excretes more iodine because of intake of edible salts whose iodine content is more than recommended level of 30 ppm. Distribution of values of MUI around median indicates that there was no iodine deficiency, and salt iodation therefore had the required impact. Central tendency (median) of distribution lies between ranges 20 and 24.9 μg/dl, which indicates that iodine intake of studied population is more than adequate, and they are susceptible to iodine-induced thyroid diseases. After iodine prophylaxis, an increased iodine intake is associated with increased thyroiditis [58].

Findings of present investigation indicate that studied population of Kolkata is clinically mild iodine deficient. They are susceptible to adverse health consequences like IIH and autoimmune thyroid diseases in long run. Their mild goitre endemicity may be because of iodine deficiency and relatively high consumption of goitrogenic plant foods.

People of Kolkata are ignorant about iodine nutrition, and they should be encouraged to avoid goitrogenic plant foods for eating or they should eat those foods after boiling and decanting water. The Central Health Directorate and salt manufacturing companies should be aware to maintain iodine concentration 30 ppm in edible salt samples at manufacturers' level. Periodical monitoring and evaluation of iodine status is necessary.

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