Brief Report

Iodine Nutritional Status Among School Children in Selected Areas of Howrah District in West Bengal, India

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Summary

This article is a study on iodine nutritional status among 1627 school children aged 6–12 years, along with biochemical analysis of iodine of 200 urine samples, 50 drinking water samples and 175 salt samples carried out in certain randomly selected areas of the district Howrah in Gangetic West Bengal. Results indicate that the entire region is clinically goitre endemic having goitre prevalence 38%; however the median urinary iodine level was 25 μg/dl indicating no biochemical iodine deficiency, 70% salt samples had a recommended level of iodine and the region was found to be environmentally iodine sufficient. Consumption of dietary goitrogen and hard drinking water may have the possible role for the persistence of endemic goitre in the region.

Key words: endemic goitre, goitrogens, school children, urinary iodine, urinary thiocyanate.

Introduction

Iodine is an important micronutrient required for human nutrition. Iodine deficiency disorders (IDD) are major public health problems all over the world including India. The effects of IDD vary according to the person's status. The effects of IDD include goitre, hypothyroidism and loss of energy and in pregnant women, they cause miscarriages, stillbirths and mentally retarded children. Among children, some of the major effects are impaired mental and physical development, mental retardation, physical deformities and cretinism [1]. Overall, iodine deficiency affects the socio-economic development of a community [2].

The northern part of the state of West Bengal is hilly and located in the classical iodine-deficient goitre-endemic belt in India [3], while its major southern region is in the Gangetic basin where the land is plain, fertile and thickly populated. In the post-salt iodization phase, endemic goitre has been reported from many areas in India [4–8] including West Bengal [9–12]. But systematic studies on the iodine nutritional status of the population of the Gangetic regions of Howrah district are not available except our earlier preliminary work [10]. The present study was therefore undertaken to evaluate the state of iodine nutrition, distribution of iodine through iodized salt, bioavailability of iodine and consumption pattern of goitrogenic foods that generally interfere with iodine nutrition in randomly selected certain areas of Howrah district in West Bengal.

Materials and Methods

Howrah is one of the most thickly populated districts in Gangetic West Bengal. It has a population of 18 80 530 (2001 Census Report) living under 14 Community Development Blocks (CD Blocks) of which five blocks attached to the river Ganga are selected. Most of its population lives in completely rural areas whereas some others live in industrial but rural areas. People are engaged in either agricultural activities or working in factories. Their diets are mainly non-vegetarian and consist of cereals (rice), pulses, fish and vegetables of Brassica family with others.

To get the representative populations from the studied regions, five areas were selected from five CD Blocks by purposive sampling method [13]. From each selected area, one school was randomly chosen where the students (age group 6–12 years) of both sexes (as recommended by WHO/UNICEF/ICCIDD)

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[14] were available. In the areas where children of both sexes in the age group 6–12 years were not available in one school, more than one school (one primary and one secondary or one boys school and one girls school) were chosen at random.

All the students of the recommended age group who were present on the day/days of survey were clinically examined for goitre by trained research staff. Goitre grading was done according to the recommended criteria of WHO/UNICEF/ICCIDD [14] (grade 0, no goitre; grade 1, thyroid palpable but not visible and grade 2, thyroid visible with neck in normal position). The age of the students was recorded from the school register and was rounded off to the nearest whole number. In this way, 1627 students were clinically examined for the enlargement of thyroid gland.

To evaluate the state of iodine nutrition of the locality, spot casual urine samples were collected from 40 children irrespective of their thyroid status from the clinically examined enrolled student at a definite interval maintaining proportionate representative from the entire population of the studied school(s) following ICCIDD/UNICEF/WHO criteria [15] in wide-mouth, screw-capped plastic bottles adding a drop of toluene to inhibit bacterial growth and minimize bad odour. Iodine in urine was determined by the arsenite method following dry ashing in the presence of potassium carbonate [16] maintaining internal quality control having a known concentration range of each batch of test samples. In case of higher values, samples were diluted two to five times with double distilled water to get the appropriate result. Urinary thiocyanate concentration was measured from the same collected urine samples used for the analysis of iodine by the method of Aldridge [17] as modified by Michajlovskij and Langer [18].

The sources of dietary iodine are water, food and the iodized salt available in the studied areas. Iodine content in water in a region truly reflects the bioavailability of iodine [19]. To cover the entire region, 50 drinking water samples were collected from tube wells chosen at random taking 10 samples from each area. These water samples were preserved in the screw-capped plastic bottles and brought to the laboratory where it was kept at 4°C until analysis. Iodine content of such water samples was measured following the method of Karmarkar, *et al.* [16]. The presence of calcium and magnesium salt/hardness in drinking water samples was determined from the collected samples following the EDTA titration method [20]. To monitor the iodine content of salt samples available in the area, 35 marked airtight plastic containers were distributed [21] at random to the students of the studied schools and they were asked to carry samples of edible salt from their households the next day. The salt samples were kept at room temperature in the laboratory and iodine content was measured within a week following the iodometric titration method [22].

The study was conducted between September 2005 and August 2006.

Results and Discussion

Prevalence of endemic goitre in school children is the most widely accepted marker to evaluate the severity of IDD in the region. According to WHO/UNICEF/ICCIDD [21] recommended criteria, a prevalence rate of 5.0–19.9% is considered mild; 20.0–29.9% as moderate and above 30% is considered as a severe public health problem. The overall goitre prevalence of 38% indicates that IDD is a severe public health problem in the studied region (Table 1). A gradual increase in goitre rate is found from the age of 6 years till the age of 9 years, followed by a short decline (Fig. 1) as observed by earlier workers [7, 10]. The reduced goitre prevalence in the higher age was probably for the compensatory adjustment of the thyroid gland.

Median urinary iodine (MUI) level indicates the state of iodine nutrition of a study area when it is based on at least 40 urine samples taken at random [15]. A MUI concentration of 10 μg/dl and when not more than 20% of the samples are below 5 μg/dl in an area indicates no iodine deficiency [23]. The MUI level in the studied region was 25 μg/dl. In analysed

TABLE 1
Goiter prevalence in different study areas of Howrah district

Sl No.	Study areas (C. D blocks)	Total number of children examined	Number of children with goiter			Severity as public health problem
			Grade 1	Grade 2	Total (1 + 2)	
1	Panchla	344	130 (37.8)	24 (7.0)	154 (44.8)	Severe
2	Ulberia-I	326	104 (31.9)	18 (5.5)	122 (37.4)	Severe
3	Ulberia-II	261	81 (31.0)	14 (5.4)	95 (36.4)	Severe
4	Shyampur-I	398	144 (36.2)	10 (2.5)	154 (38.7)	Severe
5	Jagatballavpur	298	82 (27.5)	12 (4.0)	94 (31.5)	Severe
		1627	541 (33.3)	78 (4.8)	619 (38.0)	Severe

Severity of public health problem: 5.0–19.9% mild; 20.0–29.9% moderate;>30% Severe [14]. (Parentheses indicate percentage).

200 samples, 14% samples had iodine excretion level below 10 μg/dl and 7% samples had iodine level below 5 μg/dl (Table 2). In all the studied areas, MUI level was also greater than 10 μg/dl suggesting that as per WHO/UNICEF/ICCIDD [23] criteria there was no biochemical iodine deficiency or no inadequacy in iodine intake of the population.

Iodine content measured in the edible salts fortified with iodine, were collected from households and it was found that 30.3% salt samples had iodine level below the adequate level of 15 ppm, 15.4% salt samples had iodine level above 15 ppm but below 30 ppm and 54.3% salt samples had iodine level more than 30 ppm. In Shyampur-I and Jagatballavpur blocks, more than 50% salt samples had iodine level below 15 ppm (Table 2). It has been recommended that 90% of the households should get iodized salt at the recommended level of 15 ppm [23]. So our study reveals that studied population consume inadequate iodine through edible salt though MUI value of more than 10 μg/dl reflects that the population might be having some other sources of dietary iodine.

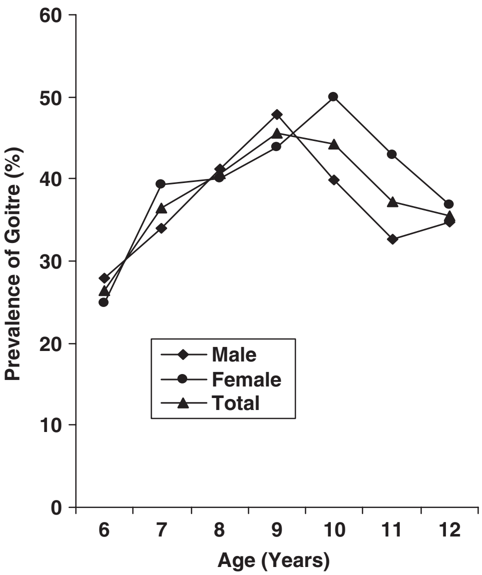

FIG. 1. Age specific goitre prevalence in the studied region in Howrah district, West Bengal.

To study the bioavailability of iodine, the iodine content of drinking water was estimated. Zeltser, *et al.* (1992) [24] have categorized the iodine deficient zones as the severe deficient zone having iodine less than 4 μg/l of water; moderate deficient zone with iodine level 4–10 μg/l of water and the relative deficient zone having iodine level 20 μg/l of water. The median iodine value, 60 μg/dl in the ranges from 41.5 to 92.5 μg/l indicates that the region is environmentally iodine sufficient or the soil is rich in iodine.

Dietary survey in the study areas showed that the vegetables of cyanogenic origin (SCN precursors) which are commonly available and used are cabbage, cauliflower, radish, mustard, turnip, sweet potato, maize, drum stick, cucumber, etc. The consumption of cyanogenic plant foods was also evident from the urinary excretion of thiocyanate (SCN). The urine samples that were analysed for urinary iodine were further tested for quantification of thiocyanate. The mean urinary thiocyanate level was 0.955 ± 0.52 mg/dl indicating that the entire studied population was exposed to thiocyanate load (Table 2). In India, large numbers of cyanogenic plants (SCN precursors) are used as common vegetables and IDD thus persists in many such regions in spite of recommended iodine intake [5, 8]. Cyanogenic plant foods used as common vegetables have potent anti-thyroid activity and supplementation of extra iodine usually fails to counteract their effect [25]. Thus involvement of thiocyanate or thiocyanate precursors present in foods consumed by the people of the region may have an important role for the persistence of goitre in

TABLE 2

Urinary iodine and thiocyanate excretion of studied population, iodine content in salt, iodine and hardness content in drinking water in different study areas of Howrah district

Sl. No	Study areas (C. D blocks)	Median Urinary iodine levels (μg/l)	Urinary thiocyanate (USCN) level mg/dl Mean ± SD	Percentage of salt samples containing iodine >15 ppm	Iodine content in drinking water (μg/l)	Calcium hardness (ppm)	Magnesium hardness (ppm)
1	Panchla	342	0.885 ± 0.35	88.6	41.5 ± 2.8	91.6 ± 8.5	526.4 ± 24.5
2	Ulberia-I	227	1.130 ± 0.62	80.0	92.5 ± 4.3	30.0 ± 12.4	242.0 ± 19.8
3	Ulberia-II	222	0.747 ± 0.45	85.7	51.5 ± 2.5	66.1 ± 6.8	371.8 ± 22.6
4	Shyampur-I	287	1.060 ± 0.54	48.6	47.0 ± 3.6	64.5 ± 11.2	161.5 ± 11.5
5	Jagatballavpur	215	0.930 ± 0.51	40.0	56.0 ± 2.4	76.1 ± 7.8	161.9 ± 21.1
		250	0.955 ± 0.52	69.7	60.7 ± 33.7	68.7 ± 38.4	305.9 ± 224.6

No. of urine samples from each area—40; Total urine samples—200.
No. of salt samples from each area—35; Total salt samples—175.
No. of drinking water samples from each area—10; Total water samples—50.

the studied region. In addition, high mineral content, particularly magnesium and calcium salt or hardness of water have been implicated as goitrogenic factor in water [26–28]. In the studied area, according to the specification of Indian standard drinking water [29], calcium level is within the permissible limit i.e. 200 ppm but presence of magnesium salt is higher in all the areas than the permissible limit i,e. 100 ppm. So, presence of magnesium salt in drinking water may pose a serious threat of thyroid disorders in the studied region.

Present study therefore indicates that goitrogenic and anti-thyroid substances that come through food and water from the environment and their action not antagonized by available iodine may have a possible role for the persistence of endemic goitre during post-iodization period in the Gangetic region of West Bengal.

References

1. Hetzel BS. An overview of the prevention and control of iodine deficiency disorders. In: Hetzel BS, Dunn JT, Stanbury JB (eds). The Prevention and Control of Iodine Deficiency Disorders, Amsterdam: Elsevier, 1987; pp. 7–31.
2. Levin HM. Economic dimensions of iodine deficiency disorders. In: Hetzel BS, Dunn JT, Stanbury JB (eds). The Prevention and Control of Iodine Deficiency Disorders, Amsterdam: Elsevier, 1987; pp. 195–208.
3. Banerjee U. Social forest in Sundarban: a tool for rural development. In: Bose AN, *et al.* (eds). Coast Zone Management of West Bengal, Calcutta: Sea Explorer's Institute, 1989; p. C12.
4. Kapil U. Current status of iodine deficiency disorders control programme. Indian Pediatr 1998;35:831–6.
5. Marwaha RK, Tandon N, Gupta N, *et al.* Residual goitre in the post-iodization phase: iodine status, thiocyanate exposure and autoimmunity. Clin Endocrinol 2003;59:672–81.
6. Pandav CS, Malik A, Anand K, *et al.* Prevalence of Iodine Deficiency Disorders Among School Children of the National Capital Territory of Delhi. ICCIDD, New Delhi: Regional Office of South Asia and Pacific, 1996.
7. Chandra AK, Ray I. Dietary supplies of iodine and thiocyanate in the etiology of endemic goitre in Tripura. Indian J Pediatr 2001;68:399–404.
8. Chandra AK, Ray I. Evaluation of the effectiveness of salt iodization status in Tripura, northeast India. Indian J Med Res 2002;115:22–7.
9. Chandra AK, Tripathy S, Mukhopadhyay S, Lahari D. Studies on endemic goitre and associated iodine deficiency disorders (IDD) in a rural area of the Gangetic West Bengal. Indian J Nutr Diet 2003;40:53–8.
10. Chandra AK, Tripathy S, Lahari D, Mukhopadhyay S. Iodine nutritional status of the school children in a rural area of Howrah District in the Gangetic West Bengal. Indian J Physiol Pharmacol 2000;48:219–24.
11. Chandra AK, Tripathy S, Ghosh D, *et al.* Iodine nutritional status and prevalence of goitre in Sundarban delta of South 24-Parganas,. West Bengal. Indian J Med Res 2005;122:419–24.
12. Chandra AK, Tripathy S, Ghosh D, *et al.* Goitre prevalence and the state of iodine nutrition in Sundarban delta of North 24-Parganas in West Bengal. Asia Pac J Clin Nutr 2006;15:357–61.
13. Cochran WG. Sampling Technique, 3rd edn. Calcutta: Wiley Eastern Limited, 1977.
14. WHO/UNICEF/ICCIDD. Indicators for Assessing Iodine Deficiency Disorders and Their Control Through Salt Iodization. WHO/NUT/94.6, 1994.
15. Dunn JT, Van der Haar F. Detection of iodine deficiency. In: A Practical Guide to the Correction of Iodine Deficiency. Technical Manual No. 3. The Netherlands: ICCIDD/UNICEF/WHO Publication, 1990;13–20.
16. Karmarkar MG, Pandav CS, Krishnamachari KAVR. Principle and procedure for iodine estimation. A Laboratory Manual, New Delhi: Indian Council of Medical Research, 1986.
17. Aldridge WN. The estimation of micro quantities of cyanide and thiocyanate. Analyst, 1945;70:474–5.
18. Michajlovskij N, Langer P. Studien uber Benziehungen Zwischen Rhodanbildung und Kropfbildender Eigenschaft Von Nahrungsmitteln. In: Gehalt einiger Nahrungs Mittel an praformierten Rhodanid. Z Physiol Chem 1958;312:26–30.
19. Hetzel BS. The story of iodine deficiency: an international challenge in nutrition. Delhi: Oxford University Press, 1989.
20. Rand MC, Greenberg AE, Taras MJ. edn. Hardness. In: Standard Methods for the Examination of Water and Waste Water, 14th edn. Washington DC: Published by American Public Health Association, American Waste Works Association, Water Pollution Control Federation, 1975; pp. 200–20.
21. Indicators for Trackling Progress in IDD Elimination. IDD Newsletter, USA, 1994;10:37–41.
22. UNICEF/ICCIDD/PAMM/WHO. Titration methods for salt iodine analysis. In: Sullivan KM, Houston E, Gorestein J, *et al.* (eds). Monitoring Universal salt iodization programme, The Netherlands: Medknow Publications, 1995; p. 11.
23. ICCIDD/UNICEF/WHO. Assessment of Iodine Deficiency Disorders and Monitoring Their Elimination, A Guide for Programme Managers, 2nd edn., The Netherlands, WHO/NHD/01.1, 2001.
24. Zeltser ME, Aldarkhanov BA, Berezhnaya IM *et al.* Iodine deficiency and its clinical manifestation in Kazakhastan. IDD Newsletter, USA, 1992;8:5–6.
25. Chandra AK, Mukhopadhyay S, Lahari D, Tripathy S. Goitrogenic content of Indian cyanogenic plant foods & their *in vitro* anti-thyroidal activity. Indian J Med Res 2004;119:180–5.
26. Koutras DA. Trace elements, genetic and other factors. In: Stanbury JB, Hetzel BS (eds). Endemic Goiter and Endemic Cretinism, New York: Wiley and Sons, 1980; 225–68.
27. Langer P. History of goitre. In: Endemic Goiter, World Health Organization (ed), WHO, Geneva: Monograph Series 44; 1960, 9–25.
28. Das SC, Isichei UP, Otokwula AA, *et al.* Neonatal chemical hypothyroidism in Nigeria. Ann Trop Paediat 2004;24:237–40.
29. Indian Standard Drinking Water – Specification. First Revision, UDC 628.1.003, Bureau of Indian Standards, New Delhi, IS 10500: 1991 (Amendment 1993).