Goiter Prevalence and Iodine Nutritional Status of School Children in a Sub-Himalayan Tarai Region of Eastern Uttar Pradesh

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Abstract

Objectives: *The present work was undertaken to evaluate the prevalence of goiter, state of iodine nutrition of the population, distribution of iodine through edible salt, bioavailability of iodine, consumption of common goitrogenic food that generally interfere with iodine nutrition in Naugarh sub-division of Siddharthnagar district in Uttar Pradesh, India.* ***Setting:*** *Five areas were selected from 5 Community Development (CD) Blocks taking one from each by purposive sampling method. In each area, Primary and Junior high schools were selected by simple random sampling to get representative target population.* ***Methods:*** *Clinical goiter survey was conducted in 1663 school-aged children from both sexes (6-12 yrs), along with the biochemical analysis of iodine (I) and thiocyanate (SCN) in 200 urine samples, iodine content in 175 edible salt samples and 20 water samples collected from the selected study areas.* ***Results:*** *The studied region is severely affected by Iodine deficiency disorders (IDD) as goiter prevalence is 30.2% (grade1: 27.1%; grade 2:3.1%).Median urinary iodine level was 96 µg/L indicating biochemical iodine deficiency. The mean urinary thiocyanate was 0.810±0.490 mg/dL and mean of I/SCN ratios in all the studied areas were above the critical level of 7. However, 22% of the individual had I/SCN ratio ≤7 indicating their susceptibility for the development of goiter. Only 12.6% of the salt samples had adequate iodine i.e., ≥15ppm while iodine content in drinking water varied between 7.5-10.7 µg/L.* ***Conclusion:*** *Iodine deficiency is the primary cause, however the consumption of cyanogenic food may have important role for the persistence of IDD in the studied region during post salt iodization phase.*

Key words: *Dietary goitrogen, Endemic goiter, India, Urinary iodine, Urinary thiocyanate.*

Introduction

Iodine deficiency disorders (IDD) refer to a complex clinical and sub clinical disorder caused for the lack of adequate dietary iodine. Iodine deficiency also affects the socioeconomic development of a community(1). In India, IDD constitute a major public health problem. Not a single state or union territory in the country is free from the problem of IDD; out of 587 districts in the country, 282 have been surveyed for IDD and 241 are found to be goiter endemic(2).

In Uttar Pradesh (UP), out of 83 districts, 34 districts have been surveyed for iodine nutriture and 29 districts have been found goiter endemic(3). IDD is not only restricted in the hilly Himalayan mountain areas in India, it has also been reported from the Sub-Himalayan flat lands (Tarai), plains, riverine areas, deltas and even costal regions(4-6). Siddharthnagar district is located in Eastern UP at a distance of about 32-48 kms from the outer range of the Himalayas. Reports on endemic goiter and current state of iodine nutrition are not available from the district and thus the present investigation has been undertaken to evaluate the prevalence of goiter, state of iodine nutrition of the population, distribution of iodine through edible salt as a part of iodization program, bioavailability of iodine

and consumption pattern of common goitrogenic food that generally interfere with iodine nutrition.

METHODS

Study on Goiter Prevalence

Selection of study area: The goiter survey was conducted in Siddharthnagar district, located on Indo-Nepal border (area: 2895 sq.km; population: 20 lakhs) between January-July 2006. It has 14 Community Development Blocks (CD Blocks) of which 10 are rural and 4 are urban. The present study was conducted in Naugarh Subdivision which consists of 5 CD Block (1 urban, 4 rural). To get proper representation, 5 areas / localities were selected from 5 CD Blocks taking one from each by purposive sampling method(7). In each study area, at least two primary schools and one junior high school were selected by simple random sampling to get adequate representative target population for goiter survey.

Selection of target population: As per recommendation of WHO/UNICEF/ICCIDD(8), the school children in the age group 6-12 yrs from both sexes were selected, because of their high vulnerability to goiter, easy accessibility and because they are representative of their age group in the community(8). As there is no adequate information on prevalence of goiter in the study area, the sample size has been calculated assuming goiter prevalence of 20% and allowable/permissible error of 10%. A sample size of 1600 was obtained. A total of 1663 school children in the age group 6-12 yrs were included from 19 schools of 5 study areas.

Goiter survey method: All the students of the recommended age group who were present on the days of survey were clinically examined for the enlargement of thyroid (goiter) by trained research staff using palpation method as per recommendations(9). Goiter grading was done as per recommended criteria of WHO/UNICEF/ICCIDD [Grade 0: no goiter; Grade1: thyroid palpable but not visible; Grade 2: thyroid visible with neck in normal position](8). The age of the examined students was recorded from the school register and was rounded off to the nearest whole number.

State of Iodine nutrition

In each study area, 40 urine samples were collected because measurement of iodine content in 40 samples at random gives a clear idea regarding the consumption pattern of iodine intake of that locality(9). Samples were collected from children of both sexes irrespective of their thyroid status, maintaining proportionate representation from the entire study population. Urine samples were collected in a wide mouth screw capped plastic bottle adding a drop of toluene to inhibit bacterial growth and minimize bad odor. The samples were brought to the laboratory and kept at 4ºC till analyzed. Iodine in the urine was determined by standard methods(10) maintaining internal quality control having a known concentration range of iodine content with each batch of samples.

Distribution of Iodine through edible salts

Measurement of iodine content of at least 35 salt samples collected at random from a locality represent valid estimate about the distribution of iodine through edible salts of the locality(11). Therefore, 35 marked airtight plastic containers were distributed at random to the students of the studied schools and they were asked to bring about 20 g of 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 by iodometric titration method(12).

Bioavailabilty of Iodine

Iodine content in the foods/vegetables grown and consumed by the people of a particular geographic region is dependent on iodine content of the soil that can be assessed by measuring iodine content in drinking water(13). In each study area, 4 water samples at random were collected in clean screw capped plastic iodine free bottles from shallow tube wells (100-150 ft depth), the people use for drinking. The samples were brought to the laboratory and kept at 4ºC till analyzed. The iodine content of drinking water was determined by the method of Karmarkar, *et al.*(10).

TABLE I GOITRE PREVALENCE IN DIFFERENT STUDY AREAS OF NAUGARH SUB-DIVISION IN SIDDHARTHNAGAR DISTRICT

Study areas (CD Blocks)	Rural/ Urban areas	Total no. of children examined	Number of children with goiter*			Severity as public health problem
			Grade-1	Grade-2	Total(1+2)	
Uska	rural	341	109(32.0)	9(2.6)	118(34.6)	Severe
Jogia	rural	216	60(27.8)	9(4.2)	69(32.0)	Severe
Loton	rural	321	85(26.5)	12(3.7)	97(30.2)	Severe
Naugarh	urban	387	97(25.0)	12(3.1)	109(28.1)	Moderate
Birdpur	rural	398	100(25.1)	10(2.5)	110(27.6)	Moderate
		1663	451(27.1)	52(3.1)	503(30.2)	Severe

**Severity of public health problem:5.0-19.9% mild; 20.0-29.9% moderate;>30 % severe(parentheses indicate percentage).*

Consumption of common goitrogenic food

Thiocyanate SCN) content in urine was measured by the method of Aldridge(14) and modified by Michajlovskij and Langer(15), to study the consumption pattern of common goitrogenic foods by the population.

Statistical methods: Mean, standard deviation and median values have been used to describe the data as appropriate. Two-tail chi square test was applied to determine association between prevalence of goiter with age and sex of the school children.

RESULTS

Total goiter rate (TGR) was 30.2%, and most of the goiter was palpable or Grade1 (27.1%) (***Table* I**).

TABLE II AGE SPECIFIC GOITER PREVALENCE IN THE SCHOOL CHILDREN OF NAUGARH IN SIDDHARTHNAGAR DISTRICT

Age (yrs)	Goiter grade			
	Grade-0	Grade-1	Grade2	Total(1+2)
6 (*n*=332)	262	68	2	70(21.1)
7 (*n*=325)	240	83	2	85(26.1)
8 (*n*=308)	205	92	11	103(33.7)
9 (*n*=226)	146	67	13	80(35.4)
10 (*n*=173)	119	48	6	54(31.2)
11 (*n*=160)	102	51	7	58(36.3)
12 (*n*=149)	96	42	11	53(35.6)
All (*n*=1663)	1160	451	52	503(30.2)

P<0.001.

Age specific prevalence is depicted in ***Table* II**. Goiter prevalence among male and female school children is shown in ***Table* III**. Urinary iodine and SCN excretion pattern of the studied population is shown in ***Table* IV**. ***Table* V** depicts the iodine content of salt samples and drinking water.

Consumption pattern of common goitrogenic food: Iodine concentration in drinking water was 9.5 µg/L of water. In all 200 urine samples were analyzed and the mean urinary SCN level was 0.810± 0.490 mg/dL in the ranges of 0.578 to 1.187 mg/dL (***Table* IV**) indicating that the entire studied population is further exposed to thiocyanate load and their consumption of common goitrogenic food is relatively high.

It was found that mean of I/SCN ratios obtained from all study areas were well above the critical level of 7. However, a total of 22% studied individuals had the I/SCN ratios below or equal to the critical level (***Table* IV**).

TABLE III GOITRE PREVALENCE IN MALE & FEMALE SCHOOL CHILDREN OF NAUGARH SUB-DIVISION IN SIDDHARTHNAGAR DISTRICT

Sex	Goitre grade			
	Grade-0	Grade-1	Grade2	Total(1+2)
Male (*n*=925)	676(73.1)	227(24.5)	22(2.4)	249(26.9)
Female (*n*=738)	484(65.6)	224(30.3)	30(4.1)	254(34.4)
Combined (*n*=1663)	1160(69.8)	451(27.1)	52(3.1)	503(30.2)

P<0.001.

TABLE IV URINARY IODINE AND THIOCYANATE EXCRETION PATTERN OF THE STUDIED SCHOOL CHILDREN OF NAUGARH, SIDDHARTHNAGAR DISTRICT

Study areas (CD Blocks)	Urinary iodine excretion levels(µg/dL)					USCN level (mg/dL)	I/SCN ratios (μg/mg)	I/SCN* (μg/mg)	
	Median	%Urine samples <20	%Urine samples 20-49	%Urine samples 50-99	%Urine samples ≥100	Mean±SD	Mean±SD	≤3	≤7
Uska (*n*=40)	85	2.5	10	42.5	45	0.615±0.290	24.761±22.620	1	4
Jogia (*n*=40)	121	0	7.5	30	62.5	1.037±0.505	15.465±11.366	2	4
Loto (*n*=40)	55	10	35	35	20	0.634±0.393	13.394±13.674	2	14
Naugarh (n=40)	96	2.5	10	42.5	45	0.578±0.293	27.124±21.646	4	9
Birdpur (*n*=40)	142	7.5	5	22.5	65	1.187±0.566	21.394±35.303	–	4
Total (*n*=200)	96	4.5	13.5	34.5	47.5	0.810±0.490	20.428±22.883	9(4.5)	35(17.5)

**Number of individual having I/SCN ratio below or equal to 3 and 7; SCN, thiocyanate; I, Iodine.*

TABLE V IODINE CONTENT OF SALT SAMPLES AND DRINKING WATER FROM STUDIED AREAS OF NAUGARH SUB-DIVISION, SIDDHARTHNAGAR DISTRICT

Sl.No	Study areas (CD Blocks)	Iodine content in salts (ppm) No. of samples (%)				Iodine in drinking water (μg/L)
		0	<15 ppm	15-29 ppm	≥30 ppm	Mean±SD
1	Uska	0	33(94.3)	1(2.8)	1(2.8)	10.7±0.2
2	Jogia	0	33(94.3)	0	2(5.7)	8.0±0.9
3	Loton	1(2.8)	30(85.7)	4(11.4)	0	7.5±0.5
4	Naugarh	2(5.7)	23(65.7)	2(5.7)	8(22.8)	10.5±0.5
5	Birdpur	4(11.4)	27(77.1)	1(2.8)	3(8.6)	10.7±1.7
		7(4.0)	146(83.4)	8(4.6)	14(8.0)	9.5±1.7

No. of salt samples from each area : 35 ; Total salt samples : 175; No. of drinking water samples from each area: 4; Total water samples : 20.

DISCUSSION

In the studied region, the total goiter prevalence was 30.2% (Grade1 - 27.1%; Grade 2 - 3.1%) indicating that IDD is a severe public health problem. There is a significant association between the age of school children and prevalence of goiter. In addition the prevalence among girls was more than boys. The observed result is almost consistent with earlier observations(16,17).

Urinary iodine level is used as a valuable indicator for the assessment of IDD in a region because 90% of body's iodine is excreted through urine(9). A median urinary iodine concentration of 100 μg/L *i.e.,* 50% of the samples should be above 100 μg/L. When not more than 20% of the samples are below 50 μg/L in an area, it indicates that there is no iodine deficiency in the population(11). In the present study the median urinary iodine concentration was 96 μg/L *i.e.* below 100 μg/L. Therefore, the studied region is biochemically iodine deficient at present or the people are not getting adequate dietary iodine.

It has been recommended by WHO/UNICEF/ ICCIDD that 90% of the household should get iodized salt at the level of 15 ppm(18). The present study showed that 83.4% of the households are

WHAT IS ALREADY KNOWN?

- Iodine deficiency is the major cause of endemic goiter.

WHAT THIS STUDY ADDS?

- Iodine deficiency is the primary cause for the persistence of iodine deficiency disorders during post salt iodization period in the studied region; however consumption of cyanogenic foods as an additional factor can not be ruled out.

consuming salt with inadequate iodine (<15 ppm), 4% households consuming salt without iodine and only 12.6% are consuming salt with recommended level of iodine. In the crystalline salt samples, 99.0% of the samples had iodine level below 15 ppm and in the powdered salt samples, 72.6% had iodine below that recommended value. All these results suggest that there is need to strengthen the system of monitoring the quality of salt to ensure the availability of 15 ppm of iodine at the consumption point or household level.

Iodine content in the drinking water indicates whether the region is environmentally iodine deficient or the soil is poor in iodine. Iodine deficient zones are categorized as 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(19). According to these criteria, the studied region should be considered as moderate deficient zone as evidenced by iodine content in drinking water.

Excretion of SCN indicates the consumption pattern of cyanogenic plant foods (such as cabbage, cauliflower, radish, mustard, maze, turnip, sweet potato *etc.*) of the population in an area. In post iodization phase, thiocyanate appears to have an important role in goiter formation especially among poor children in India(20). It has been mentioned that mean value obtained from non-endemic population was 0.504±0.197 mg/dL. In the present study the mean urinary thiocyanate value was 0.810±0.49 mg/dL that indicates the consumption of cyanogenic plant foods of the population in the studied region is relatively high.

Available literature suggests that development of goiter does not necessarily depends upon the consumption of large quantities of food containing SCN precursors but critically related to the balance between the dietary supplies of iodine and thiocyanate(21). The ratio is higher than 7 under normal condition and endemic goiter develops when it reaches a critical threshold of 3(22). A total of 22% of the studied population had I/SCN ratio below or equal to 7 indicating their susceptibility for the development of goiter. Therefore involvement of thiocyanate or thiocyanate precursors such as goitrin and isothiocyanates present in the food consumed by the people of the studied region may have additional role along with iodine deficiency for the persistence of endemic goiter during post salt iodization phase.

The present study indicates that biochemical iodine deficiency due to intake of less iodine is the main cause for the persistence of IDD in Naugarh sub-division; besides iodine deficiency, the anti thyroidal / goitrogenic substances present in foods may have the additional role for IDD during post salt iodization phase.

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REFERENCES

1. 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. p. 195-208.
2. Kapil U. Progress made in elimination of IDD and possible impact of lifting ban on sale of non iodized salt. J Acad Hosp Admin 2000; 12: 33-41.
3. Kapil U, Singh P. Status of iodine content of salt and urinary iodine excretion levels in India. Pak J Nutr 2003; 2: 361-373.
4. Clugston GA, Dulberg EM, Pandav CS, Tilden RL. Iodine deficiency disorders in South East Asia. *In* : Hetzel BS, Dunn JT, Stanbury JB, eds. The Prevention and Control of Iodine Deficiency Disorders. Amsterdam; Elsevier; 1987. p. 273-308.
5. Chandra AK, Tripathy S, Ghosh D, Debnath A, Mukhopadhyay S. Iodine nutritional status and prevalence of goiter in Sundarban delta of South 24-Parganas, West Bengal. Indian J Med Res 2005; 122: 419-424.
6. Chandra AK, Tripathy S, Ghosh D, Debnath A, Mukhopadhyay S. Goiter prevalence and the state of iodine nutrition in Sundarban delta of North 24-Parganas in West Bengal. Asia Pac J Clin Nutr 2006; 15: 1-4.
7. Cochran WG. Sampling Techniques, 3rd edn. Calcutta: Wiley Eastern Limited ;1977.
8. WHO/ UNICEF/ ICCIDD. Indicators for assessing iodine deficiency disorders and their control through salt iodization. Geneva: WHO/NUT/94.6; 1994.
9. 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. p. 13-20.
10. Karmarkar MG, Pandav CS, Krishnamachari KAVR. Principle and Procedure for Iodine Estimation, A Laboratory Manual. New Delhi: ICMR; 1986. p.10-12.
11. Indicators of tracking progress in IDD elimination. IDD Newsletter 1994; 10: 37-41.
12. Titration methods for salt iodine analysis. *In*: Sullivan KM, Houston E, Gorestein J, Cervinskas J, eds. Monitoring Universal Salt Iodization programme. Atlanta, Georgia, USA: UNICEF/ ICCIDD/PAMM/WHO 1995; p11.
13. Hetzel BS. The Story of Iodine Deficiency: An International Challenge in Nutrition. Delhi: Oxford University Press; 1989.
14. Aldridge WN. The estimation of micro quantities of cyanide and thiocyanate. Analyst 1945; 70: 474-475.
15. Michajlovskij N, Langer P. Studien uber Benzie hungen Zwischen Rhodanbildung and kropfbildender Eigenschaft Von Nahrungsmitteln. *In:* Gehalt einiger Nahrungs Mittel an praformierten Rhodanid. Hoppe Seyless. Z Physiol Chem 1958; 312: 26-30.
16. 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. p.7-31.
17. Chandra AK, Ray I. Influence of age, sex and caste on goiter prevalence of the people in Tripura, North East India. J Hum Ecol 2001; 12: 313-317.
18. ICCIDD/UNICEF/WHO. Assessment of iodine deficiency disorders and monitoring their elimination. A Guide for Programme Managers. 2nd edn. WHO/NHD/01.1; 2001.
19. Zeltser ME, Aldarkhanov BA, Berezhnaya IM, Spernasky GG, Bazarbekova RB, Nurbekova AA, *et al.* Iodine deficiency and its clinical manifestation in Kazakhastan. IDD Newsletter 1992 ; 8: 5-6.
20. Marwaha RK, Tandon N, Gupta N, Karak AK, Verma, Kochupillai N. Residual goitre in the post iodization phase: iodine status, thiocyanate exposure and autoimmunity. Clin Endocrinol 2003; 59: 672-681.
21. Delange F, Vigneri R, Trimarchi F, Filetti S, Pezzino V, Squatrito S, *et al*. Etiological factors of endemic goiter in North Eastern Sicily. J Endocrinol Invest 1978; 2: 137-142.
22. Delange F, Bourdoux P, Colinet E, Courtois P, Hennart R, Lagasse M, *et al*. Nutritional factors involved in the goitrogenic action of Cassava. *In*: Delange F and Ahluwalia R, eds. Cassava Toxicity and Thyroid Research and Public Health Issues. Ottawa, Canada: International Development Research Center; 1982. p.17-26.